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VOLUME THE THIRTY-FIFTH

SESSION 1941-42

PART I

NOVEMBER — APRIL



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1942

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[October 14, 1941]

Interactions of Fat and Carbohydrate Metabolism— New Aspects and Therapies

PRESIDENT'S ADDRESS*

By R. D. LAWRENCE, M.D., F.R.C.P.

ABSTRACT.—No one has so far produced anything approaching a clear picture of either fat or carbohydrate metabolism and the interactions of the two are still more involved and elusive although they clearly exist. Plants and animals build up reserves of fat from carbohydrate, but the reverse process (fat into carbohydrate), proved in plant seeds, is still unproven in animals, although theoretically possible.

In normal human metabolism fat-carbohydrate interactions are almost hidden. The disturbances shown in the metabolism of a diabetic seem to give us the clearest indications of these interactions. Either carbohydrate or fat can be used as the main source of body fuel, but their metabolic course is very different, both as regards chemistry and function. It is only when carbohydrate is not available, either in starvation or severe diabetes, that fat provides the fuel of the body; this contrast is also manifest in the blood and internal organs, especially the liver. Under the commonest normal conditions of diet carbohydrate is predominantly and preferentially used for metabolism. The liver is rich in glycogen, poor in fat; the blood fat is minimal and ketone bodies, although perhaps present in small amount in the blood at most times, are absent on common tests. As soon as carbohydrate is insufficiently available for the needs of metabolism, depot fat flows to the liver and is there catabolized to ketone bodies which recent proof has shown to be *burned peripherally* in the muscles independent of carbohydrate metabolism. This is a *normal process*, harmful only in diabetes, and especially harmful when it occurs suddenly, e.g. when insulin is cut off from a fat diabetic dog or human patient. A diabetic supports with ease a *prolonged* severe ketosis but suffers from one of *sudden* onset, although of milder severity. Insulin in the diabetic and sugar in the starved switches metabolism from fat to carbohydrate usage very quickly and ketonuria usually disappears in three to six hours.

"Diabetic obesity" is very common and is often seen in the earliest stages and again after insulin treatment. It seems probable that hyperglycemia causes this obesity and this has been clearly established by observations on an unusual case of lipemia, diabetes and lipodystrophy.

Nov.—THERAP. 1

Lipæmia may occur in two opposite phases of metabolism, one *anabolic*—when fat is on its way to storage, the other *catabolic*—when it is flowing from stores to the liver. The latter is the usual condition obvious in disease.

Work has also been done which suggests that other lipotropic factors—choline, lipocaic, &c., exert an influence on carbohydrate-fat balance, more specifically the glycogen-fat balance in the liver.

In America attention has been drawn to the *frequent* and *persistent* occurrence of fatty enlargement of the liver in diabetic children. The author has seen many diabetic children (usually in a state of chronic ketosis) with enlarged livers, but such enlargement has rapidly disappeared with better management of the diabetes. Only two out of some 500 diabetic children have clearly shown the unmistakable syndrome of "hepatomegalic dwarfism". In these two cases choline and lipocaic were given over prolonged periods without any effect: the liver, however, of one of these cases has since become normal by the addition of zinc protamine insulin.

RÉSUMÉ.—Jusqu'à présent aucune description claire n'a été présentée du métabolisme des hydrates de carbone ou des graisses, et leurs réactions mutuelles sont encore plus compliquées et difficiles à saisir, quoiqu'elles existent certainement. Les plantes et les animaux transforment les hydrates de carbone en réserves de graisse, mais le procédé inverse (transformation de graisse en hydrate de carbone), qui a été prouvé dans les graines des plantes, reste encore à être démontré chez les animaux, quoiqu'il soit théoriquement possible.

Dans le métabolisme humain normal, les réactions entre la graisse et l'hydrate de carbone sont presque complètement masquées. Les troubles métaboliques du diabétique semblent donner les indications les plus claires de ce que sont ces réactions. Le combustible principal de l'organisme peut être l'hydrate de carbone ou la graisse, mais les deux procédés métaboliques sont très différents, autant chimiquement que par leurs fonctions. La graisse forme le combustible de l'organisme seulement quand l'hydrate de carbone vient à manquer, comme dans l' inanition ou dans le diabète grave; cette différence se montre aussi dans le sang et dans les organes internes, le foie en particulier. Sous les conditions normales les plus ordinaires, l'hydrate de carbone est utilisé d'une manière prédominante et de préférence dans le métabolisme. Le foie est riche en hydrate de carbone et pauvre en graisse, la graisse sanguine est au minimum, et les corps cétoniques, quoique le sang en contienne peut-être presque toujours de petites quantités, ne sont pas décelés par les épreuves ordinaires. Dès que l'hydrate de carbone devient insuffisant pour les besoins du métabolisme, la graisse est transportée des dépôts dans le foie, où elle est transformée en corps cétoniques qui sont brûlés à la périphérie, dans les muscles, indépendamment du métabolisme des hydrates de carbone, ainsi qu'il a été prouvé récemment. Ce procédé est *normal*, et nuisible seulement chez le diabétique, et surtout s'il se déclenche brusquement, par exemple chez un chien ou un malade diabétique gras privé d'insuline. Le diabétique supporte bien une cétose grave *prolongée*, tandis qu'il souffre d'une cétose moins grave à *début brusque*. L'insuline dans le diabétique et le sucre dans l' inanition changent très rapidement le métabolisme de l'utilisation de la graisse à l'utilisation de l'hydrate de carbone, et la cétonurie disparaît le plus souvent en 3 à 6 heures.

L' "obésité diabétique" est très fréquente, et se voit souvent dans les premiers stades de la maladie, et de nouveau après le traitement insulinaire. Elle est probablement due à l'hyperglycémie, et ceci a été clairement démontré par l'observation d'un cas rare de lipémie, diabète et lipodystrophie.

La lipémie peut exister dans deux phases opposées du métabolisme, l'une *anabolique*, quand la graisse est en route vers ses dépôts, et l'autre *catabolique*, quand elle passe de ces dépôts vers le foie. Cette seconde phase est celle qui se manifeste le plus souvent en pathologie.

Des recherches ont été faites qui indiquent que d'autres facteurs lipotropiques—la choline, le lipocaic, etc.—ont une influence sur l'équilibre graisse-hydrate de carbone, surtout dans le foie.

En Amérique l'attention a été attirée sur la *fréquence* et la *persistance* de l'hépatomégalie graisseuse chez les enfants diabétiques. L'auteur a observé de nombreux enfants

diabétiques (généralement dans un état de cétose chronique) avec de gros foies, mais cet agrandissement a disparu rapidement avec un traitement plus efficace du diabète. Deux seuls, parmi environ 500 enfants diabétiques, ont montré le syndrome manifeste du "nanisme hépatomégalique". Chez ces deux l'administration prolongée de choline et de lipocaïc n'a eu aucun effet. Toutefois, dans un de ces cas le foie est redevenu normal depuis l'emploi de l'insuline-protamine-zinc.

RESUMEN.—Aún no se ha producido nada como cuadro claro del metabolismo graso o hidrocarbonado, y los intercambios de los dos encuéntranse más confusos todavía y difíciles de comprender, si bien claro está que existen. Las plantas y los animales fabrican reservas grasientas con el hidrocarbonato, pero el proceso contrario (de grasa al hidrato de carbono), probado para los granos de plantas, no se ha probado aún para los animales, aunque sea en teoría posible.

En el metabolismo normal del hombre los recambios de grasa e hidrocarbonato se esconden casi enteramente. El signo más claro de estos recambios, parecen darnos las perturbaciones de metabolismo en el diabético. Se puede utilizar como fuente principal de energía o el hidrocarbonato o el graso, pero son distintísimos sus cursos metabólicos, con respecto a la química así como a la función. Sólo es cuando falta el hidrocarbonato, en la inanición o en la diabetes grave, que la grasa provee de combustible al cuerpo; el mismo contraste se manifiesta en la sangre y los órganos internos, sobre todo en el hígado. En las condiciones más normales de régimen se sirve principalmente y de preferencia del hidrocarbonato para el metabolismo. El hígado está rico en glucógeno, pobre en grasa; la grasa de la sangre está mínima y los cuerpos cetónicos, aunque pequeñas cantidades sean generalmente presentes en la sangre, no se comprueban a las reacciones acostumbradas. Apenas revélase deficiente el hidrocarbonato para las necesidades del metabolismo, la grasa de depósito fluye al hígado y allá se cataboliza a cuerpos cetónicos, los cuales, como lo han demostrado las pruebas recientes, son quemados *periféricamente* en los músculos aparte del metabolismo hidrocarbonado. Eso es un *proceso normal*, dañoso solamente en la diabetes y sumamente cuando instálase bruscamente, por ej., cuando se suprime la insulina a un perro gordo diabético o a un enfermo humano. Un diabético soportará fácilmente una acetosis grave *prolongada*, pero sufrirá de un ataque *brusco* más leve. La insulina en el diabético y el azúcar en la inanición desvían muy pronto el metabolismo de la combustión de grasa a la de hidrocarbonato, y la acetonuria desaparece por lo general en 3 a 6 horas.

La "obesidad diabética" es muy común; se ve a menudo en las etapas iniciales, y otra vez tras el tratamiento insulínico. La hiperglucemia parece probable que es causa de esta obesidad, y el hecho se ha probado por observaciones sobre un caso insólito de lipemia, diabetes y lipodistrofia.

La lipemia puede encontrarse en dos fases opuestas del metabolismo, la una, *anabólica*—cuando la grasa está en ruta de almacenarse, la otra, *catabólica*—cuando pasa de las reservas al hígado. Esto es la condición usual que muestra la enfermedad.

También se ha realizado investigaciones indicando que otros factores lipotrópicos—colina, lipocaïc, etc., influyen en el balance de hidrocarbonato y grasa, más específicamente el balance de glucógeno y grasa en el hígado.

En América se ha llamado atención a la ocurrencia *frecuente y persistente* de hipertrofia grasosa del hígado en los niños diabéticos. El autor ha visto sinnúmero de niños diabéticos (generalmente en estado de cetosis crónica) con el hígado hipertrofiado, pero dicha hipertrofia desapareció prontamente en mejor tratar la diabetes. Sólo dos sobre como 500 niños diabéticos mostraron claramente el síndrome unequivoco de "enanismo hepatomegálico." En estos dos casos se administraban colina y lipocaïc durante períodos prolongados sin efecto alguno: con todo, se ha normalizado después el hígado de uno caso gracias a la adición de protamina-cinc-insulina.

RESUMO.—Até hoje ninguém tem conseguido qualquer resultado que mostre claramente o metabolismo da matéria gorda ou do hidrato de carbono e as ações recíprocas de ambos estão ainda muito complicadas e mal definidas, apesar de evidentemente existentes. As plantas e os animais armazenam reservas de matéria gorda proveniente dos hidratos de

carbone, mas o processo inverso (materia gorda em hidrato de carbone) que está provado existir nas sementes das plantas, não está ainda descoberto nos animaes, apesar de ser possível teoricamente.

No metabolismo humano normal as ações reciprocas da materia gorda e do hidrato de carbone estão quasi por descobrir. As alterações que apparecem no metabolismo de um diabetico parecem dar-nos indicações claras sobre estas ações reciprocas. O hidrato de carbone ou a materia gorda podem ser usados como a maior fonte de combustível do corpo, mas o seu processo metabolico é muito diferente, tanto quanto á quimica, como á função. É sómente quando o hidrato de carbone não existe, quer por inanição, quer por diabetes muito adelantada, que a materia gorda abastece o combustível do corpo; este contraste tambem se manifesta no sangue e nos órgãos internos, especialmente no figado. Nas condições normaes mais usuaes de dieta, o hidrato de carbone é preponderantemente e de preferencia usado para o metabolismo. O figado é rico em glicogeno e pobre em materia gorda; a materia gorda do sangue é insignificante e os corpos quetona ainda que talvez, presentes no sangue, em pequena quantidade, na maior parte das veses, estão ausentes em experiencias vulgares. Logo que o hidrato de carbone não existe em quantidade suficiente para as necessidades do metabolismo, a materia gorda armazenada corre para o figado e lá é catabolisada em corpos quetona, que provas recentes nos mostram serem *queimados perifericamente* nos musclos, independentemente do metabolismo do hidrato de carbone. Este é o *processo normal*, prejudicial sómente na diabetes, e muito prejudicial, quando se dá repentinamente, isto é, quando a insulina é sustada num cão diabetico gordo, ou num doente humano. O diabetico suporta facilmente uma quetos severa e *prolongada*, mas sofre de uma, com ataque *repentino*, ainda que seja de menor intensidade. A insulina no diabetico e o assucar no faminto muda o metabolismo do uso da materia gorda para o do hidrato de carbone muito depressa e a quetonuria desaparece geralmente de 3 a 6 horas.

A "obesidade diabetica" é muito comum e encontra-se no periodo inicial da doença e tambem depois do tratamento com a insulina. Parece provavel que a hiperglicemia seja a causa desta obesidade, e isto tem sido claramente provado com as observações feitas em casos pouco frequentes de lipemia, diabetes e lipodistrofia.

A lipemia pode apparecer em duas fases opostas do metabolismo, uma *anabolica*—quando a materia gorda está a caminho do deposito, e outra *catabolica*—quando está a correr do deposito para o figado. Esta ultima é a condição usual obvia na doença.

Tambem se tem feito trabalhos que sugerem a idea de que outros factores lipotropicos—cholina, lipocaico, etc., exercem uma influencia no equilibrio entre os hidratos de carbone e a materia gorda, mas especialmente mais, no equilibrio glicogeno gordoso no figado.

Na America tem-se observado uma occurencia digna de atenção, e que é a de apparecerem casos *frequentes e persistentes* de dilatação gordosa do figado em creanças diabeticas. O autor tem visto muitas creanças diabeticas (em geral no estado de quetosis cronica) com figados dilatados, mas esta dilatação desaparecia com um tratamento melhor da diabetes.

Duas sómente, de 500 creanças diabeticas, mostraram claramente o sindroma evidente de "paragem de crescimento hepatomegalica". Nestes dois casos cholina e lipocaico foram administrados durante muito tempo, mas sem efeito: o figado, porem, de um destes casos ficou normal pela adição de insulina protamina de zinco.

The subject of diabetes lends itself peculiarly well to controlled observations on human metabolism and many such observations may legitimately be classified as experiments. Indeed the fundamental test for the new advances for which pharmacologists and therapeutists work must be human—does a new remedy, be it a drug or a diet, succeed in human disease? Most of the controlled experiments I have carried out on more than 30 substances have been distressingly negative. These have included beans from Bali, wisps of grass from Africa, dried roots from Chili, evil smelling and tasting herbs from all over the world, from which, in spite of their miraculous local reputation as diabetic cures, one expects nothing. But someone must test these and give the lie to error. Worse, because immoral, has been the flow of proprietary oral preparations, some from reputable drug firms and even advertised in our best medical journals—all negative—and one could not even

name them and publish the results for fear of a libel action from vested interests. But worst of all, as involving much serious work, are new chemical and endocrine remedies published with apparent honesty by reputable scientists and often supported by convincing protocols—none of which could be confirmed. The error in most of the last cases has arisen from the clinicians concerned being unable to appreciate what a controlled experiment in diabetes means and involves. At any rate these disappointing experiences have led me to the following sobering if somewhat cynical reflections on clinical research.

We may be quite sure that in medical research, especially in the rapidly progressing but almost unknown fields of metabolism and biochemistry, any guesses we make (call them hypotheses if you like) will mainly be wrong. The best we can strive to do is to establish a new fact—as often as not merely a new relationship between two already known phenomena. Any total explanation we give and any large hypothesis we build up round this treasured new “fact” will almost certainly be proved later, by ourselves or others, to be wrong or at most half-right. Such imaginative hypotheses are fascinating and inevitable to the inquiring scientific mind and indeed are integral stepping-stones in the process of research. But I find I must beware of them. It is all too easy to become biased in favour of one's own hypothesis, to plan a series of experiments to give confirmatory results, to shut one's eyes and harden the mind to contradictory results and other theories and so to perpetuate error.

Another great difficulty of which I am acutely aware, for instance in the wide subject I am dealing with, is the impossibility of reading and digesting all the old and new work impinging on the problem—and one thing one must do oneself is one's own reading. Such width of reading and knowledge seems now only possible to whole-time workers on a limited field—and what a revelation it is to suck their brains.

Working on fields that are largely unknown, error is inevitable, but I have learned to recognize and tried to guard myself a little against two tempting crimes in clinical research: (1) the premature publication of improperly controlled and half-proven facts which, especially if emanating from a serious source, may take a generation to disprove; (2) the building up of *wide* generalizations and hypotheses, even on true evidence, which are apt not only to cloud the future critical clarity and work of the author himself, bias being almost inevitable, but to lead others astray for a long time.

The Interactions of Fat and Carbohydrate Metabolism must sound a very ambitious subject and no comprehensive picture will be expected. No one has so far produced anything approaching a clear picture of either fat or carbohydrate metabolism and the interactions of the two are still more involved and elusive, although they clearly exist. It is certain for example that plants and animals build up reserves of fat from carbohydrate, but the reverse process (fat into carbohydrate), although proved in plant seeds and theoretically equally possible in animals, is still unproven in the latter after years of experiment and controversy. In normal human metabolism fat-carbohydrate interactions are almost hidden and laborious studies of the respiratory quotient can give us apparently only vague, wide indications, but no glimpse of underlying mechanisms. Diabetic metabolism seems to give us the clearest indications we have of these interactions, and it is the disturbances shown in this disease—no doubt akin to similar conditions seen in normals during starvation—with which I shall mostly deal. The last few years have brought important new experimental facts which change and indeed simplify our ideas.

It is clear that either carbohydrate or fat can be used as the main source of body fuel but that their metabolic course is very different both as regards chemistry and function. Carbohydrate is poorly stored and the maximum content of body glycogen, if used alone, provides calories for less than one's day's activity whereas the fat stores of the very obese can theoretically provide fuel for about six months. Carbohydrate is quickly used and seems to be the preferential fuel of muscular activity. It is only when carbohydrate is not available, either in starvation or severe diabetes, that fat provides the fuel of the body. How it can do so has been clarified recently.

This contrast is also manifest in the blood and internal organs, especially the liver which is an essential middleman for the metabolism of both carbohydrate and fat. The

carbohydrate-fed liver shows a high percentage of glycogen, a small amount of fat. As soon as carbohydrate is unavailable, in the early stages of starvation or in severe diabetes, glycogen virtually disappears and fat accumulates. *Pari passu*, the blood fat increases from the flow of stored tissue fat to the liver for catabolism, and may be so gross as to be visible in the serum as well as by chemical analysis. If this persists long the liver palpably enlarges from its extreme content of fat and later still the spleen may enlarge from lipid deposits (foam cells) in its reticulo-endothelial system. Also at any time when a switch-over from predominantly carbohydrate to fat metabolism occurs, *ketone* bodies accumulate in the blood and urine, products of fat metabolism manufactured, like sugar, entirely in the liver.

These ketone bodies have occupied for long, and still do, a prominent place in the diabetic picture, not only in theoretic but also in therapeutic significance. They were recognized to be degradation products of fatty acids, produced supposedly by β -oxidation, but their ultimate fate and function remained unknown. Some supposed that they were oxidized completely to CO_2 and water, but only by the intervening oxidation of carbohydrate and indeed a chemical coupling with carbohydrate ("fat burns in the flame of carbohydrate"). On this assumption various ketogenic/antiketogenic ratios were worked out and applied to diabetic diets—my original Line Ration Scheme was based on this principle. Others thought that ketone bodies were a stage in the conversion of fat to sugar, which seemed an essential premise when carbohydrate was thought to be the only fuel of muscular activity. None of these, or indeed other theories, were either satisfactory or fully proven and need concern us no longer, as it has now been proved that the *ketones are burned peripherally in the muscles independent of carbohydrate oxidation*. The appreciation of this fact has not yet widely reached the domains of clinical medicine but I consider it of such importance as to mention the experimental evidence briefly.

KETONES BURNED IN MUSCLES

As early as 1928 Snapper and Gruenbaum found a considerable disappearance of ketones in perfused striated muscle and Chaikoff and Soskin (1928) noted that injected aceto-acetate was used by the muscle of both normal and diabetic eviscerated dogs. This was confirmed by Friedmann (1936), Mirsky and Broh-Kahn (1937). More recently Blixenkrone-Møller (1938, *a* and *b*) showed by perfusion experiments that ketones are utilized by the muscles of normal and diabetic cats. From the greatly increased utilization by actively contracting muscle, they concluded that much of the total energy requirements can be furnished by ketone oxidation. Toennessen and Brinkman (1938) obtained the same results in rabbits.

In the last two years Stadie, Lukens and others (1940, 1941) have published a series of experiments, mainly on diabetic cats, which seem to give full confirmation of the above view. They summarize and explain the new belief (i) that the normal and diabetic animal and man burn ketone bodies peripherally independent of carbohydrate oxidation and (ii) that ketones are formed in the liver, not by β -oxidation but by "multiple alternate oxidation" of the fatty acid chain whereby 1 molecule fatty acid yields 4 of ketone bodies.

This one new fact enables a much simpler picture to be drawn of the interchanges of fat and carbohydrate metabolism which I would outline (temporarily at least) as follows:

Under the commonest normal conditions of diet, carbohydrate is predominantly and preferentially used for metabolism. The liver is rich in glycogen, poor in fat; the blood fat is minimal and ketone bodies, although perhaps present in small amounts in the blood at most times, are absent on common tests. As soon as carbohydrate is cut off or is insufficiently available for the needs of metabolism, depot fat flows to the liver and is there catabolized to ketone bodies which then provide adequate fuel for the muscles. Of course the body-weight falls by the use of stored fat. This is a *normal process* and is really harmful only in diabetes, where the intensity of the process may so poison the patient as to cause death in coma. It is not yet clear whether this is mainly due to a specific toxic action of the ketones themselves, or to other accompanying factors of acidosis and dehydration. Ketosis is especially harmful when it occurs suddenly, as

when insulin is cut off from a fat diabetic dog or human patient. The body has adequate defences against acidosis, ammonia formation by the kidneys, &c., and I have been struck by the ease with which a diabetic supports a prolonged severe ketosis but suffers from one of sudden onset, although of milder severity. Normal man and animals too develop a much more intense ketosis when first changed to fat metabolism than later. It appears, as with so many functions that are commonly in abeyance, that the body takes some time to get the function into full and efficient use.

This utilization of ketones by the muscles explains many puzzling clinical observations. Petren (1924) showed that on a diet of 200 g. fat and only 20-30 g. carbohydrate and protein an initially precomatose state and heavy ketosis might disappear and that a reasonable activity and increase in carbohydrate tolerance could be achieved, presumably by the muscles taking over an efficient use of ketone bodies and the pancreas recovering with less carbohydrate load. Others were less successful with such high fat diets, probably, I think, because they allowed more protein, which seems to me to be more ketogenic than fat itself. I have noticed too that some normal people develop ketosis more readily than others. Von Gierke's glycogen disease is the best example of this, but a few normal individuals begin to show ketonuria when breakfast has been delayed by only two hours.

Insulin in the diabetic and sugar in the starved switches metabolism from fat to carbohydrate usage very quickly and ketonuria usually disappears in three to six hours.

LIPÆMIA AND DIABETIC OBESITY PRODUCED BY HYPERGLYCÆMIA

In most normal individuals an intake of carbohydrate in excess of metabolic requirements results in its conversion to stored fat and ultimately to overt obesity. The chemical changes and the mechanisms involved are unknown and never obvious. In diabetes, too, obesity is very common and is seen (1) in the earliest, almost pre-diabetic stages, when perhaps only a glucose tolerance test reveals by hyperglycæmia the commencing defect in carbohydrate metabolism and (2) after insulin treatment when obesity often develops on quite low calorie diets. Before discussing these conditions, I wish to describe some observations on an unusual case of lipæmia and diabetes in which a clear relation between glycæmia and lipæmia has been established.

This is a woman, aged 30, whose diabetes is accompanied by a persistent lipæmia and hypercholesterolemia and a lipodystrophic condition with widespread disappearance of subcutaneous fat. Other striking abnormalities are progressive huge enlargement of the liver and spleen and an extremely high basal metabolic rate, + 150 having been found on several tests. In spite of prolonged study, I have no idea of the real nature of her disease and think it undescribed in medical literature. An attempt to abolish the lipæmia by many variations in diet, high carbohydrate, no fat, &c. failed, but full control of the diabetes (over 2,000 units of insulin per day was required) made the lipæmia normal and a rise in blood sugar reproduced a gross lipæmia. In this case hyperglycæmia causes hyperlipæmia and this has become clear for the reason, I think, that one fundamental defect in this case is the inability to store fat, at least subcutaneously, and therefore the fat which would otherwise be hidden in body stores, circulates as an overt lipæmia.

I have thought vaguely for years that there must be a blood-sugar threshold above which glycæmia is converted into fat and the above case supports this idea. I imagine that when ingested carbohydrate has filled the liver to its physiological capacity for glycogen and sugar, further carbohydrate will cause sufficient hyperglycæmia to be converted into stored fat—this is possibly the type of speculation better unpublished. A recent paper by Drury (1940) is interesting in this respect. His experiments on depancreatized animals strongly suggest that an important action of insulin is not only to store absorbed sugar, but to turn much of it into fat when large amounts of sugar are absorbed.

Pre-diabetic obesity may now be considered in the above light. A high proportion (77%, Joslin) of diabetics are overweight before symptoms of the disease become manifest and the usual loss of weight sets in. This applies, I find, not only to the elderly who may have been too fat for years, but to many, if not the majority of cases in the third and fourth decades of life. They commonly give a history of becoming 10 to 20 lb. heavier than their constant weight for a year or two before thirst and polyuria develop in spite of making no change in their activity or eating habits. The usual, rather vague, explanation has been that this obesity overstrains the pancreas and produces diabetes. I think

the opposite more, likely, that a mild symptomless hyperglycaemia comes first and causes the obesity. Later when carbohydrate tolerance falls and the loss of sugar rises, typical symptoms including loss of weight develop.

After insulin treatment too obesity is common and many cases become overweight on diets containing no excess of calories and cling to this gain in weight in spite of rigorously low diets. If enough insulin is given to utilize enough food for active life and to prevent ketosis, but insufficient to prevent a raised blood sugar for several hours of the day (a state of affairs which I cannot prevent in severe cases) obesity develops in many from these periods of hyperglycaemia.

This obesity does not occur or is reduced if too little insulin is given so that heavy glycosuria and perhaps ketonuria are present with a resultant mobilization of fat from body stores. I have slimmed some vain young diabetic women in this way by reducing their insulin heavily (one lost 8 lb. in the first week) but this manœuvre is not without danger unless close watch is kept on ketosis.

It is clear that lipaemia may occur in two opposite phases of metabolism, one *anabolic*, when fat is on its way to storage, the other *catabolic*, when it is flowing from stores to the liver. The latter is the usual condition obvious in disease and the former, apart from a few hours after a large fatty meal, is hardly ever visible.

LIPOTROPIC FACTORS, CHLORINE AND LIPOCAIC

So far I have dealt with some interactions which depend on the presence of available carbohydrate and insulin. Now I wish to draw attention to work which suggests that other lipotropic factors exert an influence on carbohydrate-fat balance, more specifically the glycogen-fat balance in the liver.

It was noted by 1924 that many depancreatized dogs maintained on certain mixed diets and insulin ultimately failed to thrive and that before death their sugar excretion and insulin requirements diminished. Their livers were found to be large, loaded with fat and impoverished in glycogen and the condition was accompanied by hypolipemia. It was soon discovered that the addition of raw pancreas to the diet prevented this type of fatty liver and kept the dogs healthy. Later it was found that lecithin could replace raw pancreas and ultimately Best and others came to the conclusion (amply confirmed by Channon in this country) that choline was the active principle in this phospholipid and have published a review of the present position of choline (Best and Ridout, 1939). Best himself has made no claims for the therapeutic use of choline or betaine, as it is so abundantly present in most diets, but this has not prevented its being tried empirically in clinical conditions of fatty hepatomegaly.

Since Best's work, it has been shown that a fatty liver is only one manifestation of a wider pathological process induced by choline deficiency. Griffith and Wade (1939) found large and haemorrhagic kidneys in rats on choline-free diets and Engel and Salmon (1941) confirm this and add the details of haemorrhages in the adrenals, lungs and heart as well as atrophy of the thymus in young rats. Choline seems likely to be promoted from a lipotropic factor to a major vitamin.

Dragstedt (1936, 1940, 1941) on the other hand, while allowing that choline has the above action, claims to have isolated a separate lipotropic factor from the pancreas which he has called *LIPOCAIC* (fat burning). Although other workers have thrown doubt on its existence (I think an open mind must be kept at this time) it has already been applied in clinical medicine and claimed to cure some cases of hepatomegaly and primary xanthomatosis. If lipocaic may be expected to have a therapeutic action, it is by analogy with animal work, most likely to be seen on hepatomegaly in diabetics, a subject which I shall now consider.

Fatty enlargement of the liver in diabetics has been noted since the last century and recent attention has been drawn to its frequent and persistent occurrence in diabetic children. Marble *et al.* (1938) from Joslin's Clinic publish its occurrence in 60 of their children and note the associated dwarfism, poor condition and poor diabetic control.

White *et al.* (1938) found that treatment with raw pancreas and choline had little effect, but that protamine insulin, by improving the diabetic control, reduced or removed the hepatomegaly. Grayzel and Radwin (1938) treated three such cases with lipocaine and claimed that the liver became normal during administration but enlarged again on omitting the substance. Rosenberg (1938) also reports a success, but I consider his case too surgically complicated to afford much evidence. Snell and Comfort's case (1937), where lipocaine reduced the large liver of a diabetic whose pancreas was destroyed by multiple stones with resulting steatorrhea, is impressive and more akin to the pancreatectomized dog than any other human case.

My personal experience of this syndrome, which we may call *hepatomegalic dwarfism* is very slight. I have seen many diabetic children with enlarged livers, usually in a state of chronic ketosis, but such enlargement has rapidly disappeared with better management of the diabetes. Only two boys out of some 500 children have clearly shown the unmistakable syndrome, and I have no idea why these two occurred as they were no worse controlled than hundreds of other cases. Such children develop large livers and protuberant bellies, stop growing and developing, have chubby moon-like faces and podgy infantile hands, but are otherwise healthy and vigorous. In some American cases attacks of jaundice and abdominal pain and even dilated abdominal veins are reported, but I have not seen this. Neither of my cases has responded at all to prolonged periods of choline and lipocaine administration, but one has recently been made normal by the addition of protamine insulin.

Bensley and Woerner (1938) make the interesting suggestion that the function of the *alpha cells of the pancreas* is connected with fat metabolism. They claim that an active principle of the cells is obtained in a special aqueous extract and that its effect on guinea-pigs is to increase liver fat, decrease glycogen and to load the *a* cells with granules. In their discussion they suggest that their active principle is correlated if not identical to lipocaine, but the basis of the argument is not clear.

OTHER ENDOCRINES AND VITAMINS

There is much evidence, both clinical and experimental—but little exact knowledge—to show that other endocrines, and even vitamins, exert an influence on both fat and carbohydrate metabolisms. The pituitary gland is clearly a most important influence as it contains active principles which are ketogenic and diabetogenic. Cushing's pituitary syndrome is a striking example in which an obesity of a pituitary type is accompanied by an upset in carbohydrate metabolism. Thyroid disease too manifests simultaneous though less striking changes in both carbohydrate and fat and the adrenal shows an influence on both. The ubiquitous vitamins also come into the picture and vitamin B₁ is accepted as having a fat-sparing action. It is concerned with the synthesis of fat from carbohydrate, probably through its action in pyruvic acid metabolism and it increases both liver and depot fat. But I have no intention of leading you further, and losing myself, in misty vistas.

In conclusion, it is obvious that I have been unable to present any uniform logical picture of the relations of fat and carbohydrate metabolism and have merely dealt with a few isolates and no harmonious whole. My excuse for so doing is my personal interest in the subject. Probably it will soon be proved, and I hope the time will not be long, that to-day's remarks are well-meaning but erroneous guesses on a difficult subject.

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President—G. DE BEC TURTLE, M.D.

[October 10, 1941]

MEETING HELD AT THE WESTMINSTER HOSPITAL, S.W.1

Idiopathic Steatorrhœa with Severe Anæmia and Pyrexia.—Sir ADOLPHE ABRAHAMIS, O.B.E., M.D., F.R.C.P.

Female, aged 19.

In February 1940, i.e. eighteen months ago, tetany in the hands first developed, often occurring several times a day, and she experienced generalized paræsthesiæ and twitchings of the eyes. The stools became bulky and pale and in May 1940 she had diarrhœa lasting a week. She was losing weight in spite of a good appetite. There was no history of diarrhœa or abdominal trouble in childhood. Catamenia regular. Family history irrelevant.

Examination on admission.—June 1940. Small girl with no bony deformities except wide subcostal angle due to large abdomen. Liver and spleen not felt. Skin normal. Underdeveloped breasts. No clubbing of fingers. Lenses clear. Trousseau's sign and Chvostek's sign positive. Knee-jerks sluggish. Apyrexial.

Investigations (June to July 1940).—Serum calcium 5.7 mg.% rising to 8.7 mg.% six weeks later and remaining at that level. Fæcal fats, total 82.4% with only 8.7% neutral fats. Later total fat figures were 39.6% and 61.8%. Blood-count at first normal then reduced i.e. R.B.C. 3,570,000; Hb. 84%; C.I. 1.0; W.B.C. 3,900 (polys. 72%, eosinos. 3%, lymphos. 20%, monos. 3%). Anisocytosis. Average diameter of red cells 7.9 μ . Blood urea 16 mg.%. Test meal: Hypochlorhydria. Sugar tolerance normal. Barium enema: Megacolon.

The signs of hypocalcæmia ceased after admission and only recurred once or twice. She was treated with a low fat diet, marmite, Bland's pill, calcium lactate, and later vitamins A and D. She was discharged in good condition and remained well for four months, but during evacuation she had no treatment and returned in May 1941 with anæmia and sore tongue.

July 3, 1941: Readmitted as an emergency. Temperature 105°, pulse 130, gross anæmia. R.B.C. 2,140,000; Hb. 22%; C.I. 0.5; W.B.C. 4,200 (polys. 60%; lymphos. 27%; monos. 11%; neutrophil myelocytes 2%). 504 normoblasts per c.mm.

There was no obvious cause for the pyrexia though she admitted a slight sore throat for one week. With blood transfusion she improved quickly but relapsed and the following blood-count was made: R.B.C. 860,000; Hb. 20%; C.I. 1.2; W.B.C. 1,700, reticulocytes less than 1%. Two further periods of pyrexia occurred during the next five weeks and so ill was she that transfusion was given on each occasion and with benefit. *B. coli*

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President—G. DE BEC TURTLE, M.D.

[October 10, 1941]

MEETING HELD AT THE WESTMINSTER HOSPITAL, S.W.1

Idiopathic Steatorrhœa with Severe Anæmia and Pyrexia.—Sir ADOLPHE ABRAHAMIS, O.B.E., M.D., F.R.C.P.

Female, aged 19.

In February 1940, i.e. eighteen months ago, tetany in the hands first developed, often occurring several times a day, and she experienced generalized paræsthesiæ and twitchings of the eyes. The stools became bulky and pale and in May 1940 she had diarrhœa lasting a week. She was losing weight in spite of a good appetite. There was no history of diarrhœa or abdominal trouble in childhood. Catamenia regular. Family history irrelevant.

Examination on admission.—June 1940. Small girl with no bony deformities except wide subcostal angle due to large abdomen. Liver and spleen not felt. Skin normal. Underdeveloped breasts. No clubbing of fingers. Lenses clear. Trousseau's sign and Chvostek's sign positive. Knee-jerks sluggish. Apyrexial.

Investigations (June to July 1940).—Serum calcium 5.7 mg.% rising to 8.7 mg.% six weeks later and remaining at that level. Fæcal fats, total 82.4% with only 8.7% neutral fats. Later total fat figures were 39.6% and 61.8%. Blood-count at first normal then reduced i.e. R.B.C. 3,570,000; Hb. 84%; C.I. 1.0; W.B.C. 3,900 (polys. 72%, eosinos. 3%, lymphos. 20%, monos. 3%). Anisocytosis. Average diameter of red cells 7.9 μ . Blood urea 16 mg.%. Test meal: Hypochlorhydria. Sugar tolerance normal. Barium enema: Megacolon.

The signs of hypocalcæmia ceased after admission and only recurred once or twice. She was treated with a low fat diet, marmite, Blaud's pill, calcium lactate, and later vitamins A and D. She was discharged in good condition and remained well for four months, but during evacuation she had no treatment and returned in May 1941 with anæmia and sore tongue.

July 3, 1941: Readmitted as an emergency. Temperature 105°, pulse 130, gross anæmia. R.B.C. 2,140,000; Hb. 22%; C.I. 0.5; W.B.C. 4,200 (polys. 60%; lymphos. 27%; monos. 11%; neutrophil myelocytes 2%). 504 normoblasts per c.mm.

There was no obvious cause for the pyrexia though she admitted a slight sore throat for one week. With blood transfusion she improved quickly but relapsed and the following blood-count was made: R.B.C. 860,000; Hb. 20%; C.I. 1.2; W.B.C. 1,700, reticulocytes less than 1%. Two further periods of pyrexia occurred during the next five weeks and so ill was she that transfusion was given on each occasion and with benefit. *B. coli*

Nov.—CLIN. I

bacilluria did not seem an adequate explanation for the pyrexia. The hæmoglobin rose gradually with the help of further transfusions and anahæmin and she eventually made a complete recovery. She is now in very good health with a blood-count of R.B.C. 3,770,000; Hb. 85%; C.I. 1.1; W.B.C. 5,900. Normal blood calcium 10.4 mg./%. Fæcal fats 37.3%.

This case is of interest because of the severity of the anemia with temporary inactivity of the bone-marrow and high pyrexia.

Chronic Miliary Tuberculosis.—SIR ADOLPH ABRAHAM, O.B.E., M.D., F.R.C.P.

Female, aged 19.

History.—The patient comes from a non-tuberculous family. Her parents and three sisters and a brother are well. Apart from jaundice at the age of 13 she was healthy until three years ago, since when she has attended Moorfields under Mr. Juler for relapsing iridocyclitis. In June 1941 she had transient pain in the right lower chest and a skiagram revealed miliary tuberculosis. She is free from cough, has continued to gain weight and was at work until admission.

Examination on admission (28.7.41).—General condition good. Weight 7 st. 5 lb. Apyrexial. Pulse 80. Blood-pressure 90/55. No definite signs in chest. Spleen palpable, not tender. No enlarged glands in neck, axillæ or groins. Signs of old iritis.

She was under observation for five weeks, felt perfectly well throughout, and maintained her weight. There was no pyrexia and the pulse averaged 80.

Investigations.—Skiagrams of chest: (30.6.41), (28.7.41), (21.8.41), no appreciable difference.

Sedimentation rate: (29.7.41) 20 mm. in one hour; (22.8.41) 14 mm. in one hour.

Blood-count: R.B.C. 4,750,000; Hb. 88%; C.I. 0.9; W.B.C. 9,300 (polys. 68%, lymphos. 30%, monos. 1%, basos. 1%).

Blood Wassermann reaction and Kahn test negative.

Stomach contents showed no T.B. on direct examination and culture.

Mantoux test positive to 1:100, negative to 1:1,000. Two "patch" tests negative.

Fracture of the Skull in an Infant Associated with Hæmatoma Formation and Followed by Development of an Encephalocele.—G. H. MACNAB, F.R.C.S.

History.—On 18.10.40 an infant aged four months was blown out of its mother's arms by blast from a bomb, and admitted to hospital unconscious, suffering from severe shock.

On examination a hæmatoma about 2 in. in diameter was found over the right parietal bone. Child severely shocked; pulse-rate 170.

20.10.40: Child no longer unconscious, but very drowsy. Hæmatoma had spread forward to the orbit, back to the occiput and up to the mid-line of the vault.

24.10.40: Hæmatoma had spread over mid-line involving the whole skull, so that the head looked almost twice its normal size. Child vomiting, but not in relation to the taking of food. Right facial weakness present.

The child's general condition gradually improved; the hæmatoma subsided after remaining over the right parietal bone for a long period. The facial weakness cleared up.

1.10.41: A perfectly healthy child. No evidence of disturbance of the central nervous system. There is a deficiency in the right parietal bone 1½ in. in diameter and a hydroencephalocele is present.

X-ray examination.—21.9.40: Fracture of right parietal bone. 12.12.40: Appearance suggests absence of portion of parietal bone. 15.9.41: Large deficiency in right parietal bone.

Exophthalmic Ophthalmoplegia (Unilateral) with Papilloedema.—S. P. MEADOWS, M.D., F.R.C.P.

R. S., male, aged 37.

History.—About nine months' history of increasing exophthalmos of right eye, with occasional blurring of vision and diplopia, but without pain. He attributes the onset

to drinking cheap wines in large amounts. The prominence of the eye is stated to have subsided for a few days early this year. He has felt slightly nervous, but has no palpitations and has lost little weight.

Examination.—Well nourished. Pulse-rate 80-100. Thyroid gland not appreciably enlarged. No tremor of hands or sweating (i.e. little evidence of general signs of thyrotoxicosis).

Eyes: Gross exophthalmos right eye (12 mm.), with marked chemosis. Marked retraction right upper lid. No exophthalmos or lid retraction left side, but slight lid lag on left (fig.). Visual acuity: R. $\frac{6}{12}$ partly; L. $\frac{6}{6}$ (uncorrected). Visual fields full. Right optic fundus shows moderate degree of papilloedema and engorgement of retinal veins. Ocular



To show exophthalmos, lid retraction and chemosis of right eye.

movements: Right eye elevation and abduction markedly impaired; adduction and depression slightly impaired. Left eye, full movements except very slight weakness of abduction. No orbital bruit. No other signs.

Investigations.—Basal metabolic rate normal. Galactose index 96. X-ray: orbit normal; chest: no evidence of retrosternal goitre. Biopsy of subcutaneous nodule from arm: lipoma. Electrocardiogram: normal. Sinus arrhythmia. Blood: Kahn test negative.

Dr. F. PARKES WEBER, in regard to the question of possible thyroidectomy, suggested that the basal metabolism should be estimated before and after a course of iodine (Lugol's solution), to ascertain the extent of the drop (if any) in the basal metabolism rate and whether the drop was sufficient to indicate the presence of thyrotoxicosis as a causal factor.

Arsenical Pigmentation and Hyperkeratosis. Disseminated Sclerosis with Epilepsy.—
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Arsenical Pigmentation and Hyperkeratosis. Disseminated Sclerosis with Epilepsy.—
S. P. MEADOWS, M.D., F.R.C.P.

of blurred
s previously.

of 35 years.
ongue biting

on at least one

Examination.—Nodules of hyperkeratosis present on annular surfaces of both hands.

Brown "rain-drop" pigmentation of trunk, but limbs and face little affected.

Central nervous system.—Moderate pallor of both optic discs. Relative central scotoma right visual field. Visual acuity: Right $\frac{6}{4}$. Left less than $\frac{6}{6}$.

Wasting and weakness of left deltoid muscle and, to less extent, of left spinati (since injury at age of 7 years).

Comment.—This case is probably one of disseminated sclerosis (epilepsy and acute unilateral retrobulbar neuritis). It appears that the man has been taking to minims of liquor arsenicalis daily for the past five years. This is presumably the cause of the arsenical pigmentation and the hyperkeratosis.

Gross Hyperpiesia well tolerated for eleven years.—C. J. GAVEY, M.D., M.R.C.P.

E. C., female, aged 65. Married. Two normal pregnancies and one miscarriage between. No renal disease. Joint pains when aged 18, bed fourteen days only. Menopause thirteen years ago.

Eleven years ago she came to the eye department with failing vision and headaches of one month's duration. Mr. Griffiths found moderate retinal arteriosclerosis with hemorrhages in both retinae and exudate in the right. Vision: R. $\frac{6}{6}$, L. $\frac{6}{6}$, improved to R. $\frac{6}{12}$, L. $\frac{6}{6}$. He referred her to Dr. Arnold Stott who found hypertension 300/160, an enlarged heart, and a thin cloud of albumin in a pale urine of S.G. 1010. Her general condition was good. Glasses were prescribed and her headaches ceased.

Since she has been observed regularly and blood-pressure has averaged 270/160, never below 250/140. Albumin occasionally present. No dyspnoea, pain in the chest, palpitation, oedema or frequency of micturition. Her only symptom is insomnia due to domestic worry.

Examination.—Spare build. Looks younger than age. Pulse regular. Radial arteries slightly thickened. Blood-pressure right arm 295/175, left arm 290/170. No pulsus alternans. Leg vessels pulsate normally. Apex beat forcible, in 5th space, $4\frac{1}{2}$ in. from mid-line. Soft apical systolic murmur. Aortic 2nd sound accented. No oedema. No evidence of lung congestion. Liver edge just felt. Optic fundi: Some arteries abnormally thin. Nipping of veins. Few pin-head exudates. Right disc edges indistinct compared with left. Right inferior temporal artery obliterated and appears as white cord. Vision: L. $\frac{6}{12}$ R. $\frac{6}{12}$ with glasses.

Recent investigations.—Urine: Albumin present in considerable amount. S.G. 1010. Few leucocytes and red cells but no casts. Blood urea 32 mg.%. Urea clearance 70% of normal. Wassermann reaction and Kahn test negative in blood. Skiagram of chest: Lung fields normal. Left ventricle moderately enlarged and displaced to left by scoliosis. Aorta uncoiled. Electrocardiogram: Normal rhythm. Left ventricular preponderance. T negative in leads 1 to 4. P-R prolonged in lead 4.

This case is exceptional in that she originally attended hospital with arteriosclerotic retinitis, has tolerated an unusually high systolic and diastolic pressure over at least eleven years without any evidence of heart failure, and remains relatively free from symptoms.

Sir ADOLPHE ABRAHAM said that examples of gross hyperpiesia in women without apparent disability, if not common, were at least familiar. He himself had observed one for over twelve years. But had anybody ever reported an instance of a male with a blood-pressure of such a degree persisting for any considerable length of time? What was the explanation of this anomaly? It was well recognized that the most gloomy prognosis was appropriate to the identification of a blood-pressure of say 200 mm. Hg in a middle-aged man, granting the absence at the time of any renal deterioration, yet an entirely different attitude was justified in the case of a woman of the same age. Was it to be supposed that there was a different sexual response to the same process or that an entirely different pathology was the explanation?

Ophthalmoplegia as Sole Manifestation of Myasthenia Gravis over Twenty-five Years.—

C. J. GAVEY, M.D., M.R.C.P.

G. D., male, aged 55.

History.—Twenty-five years ago, about a year after recovery from severe dysentery in Malta, the left upper eyelid began to droop and diplopia appeared. He noticed

sluggishness of the eye movements. Condition stationary for several years, then a left upper lid prop was fitted. Eighteen months ago the right eye became similarly affected. Though varying in severity, worse when fatigued, he has never been free from these symptoms since the onset. Dysphagia, difficulty in mastication or articulation, and weakness of limbs, have been absent throughout.

Examination.—Severe bilateral ptosis, left more than right. Gross weakness of orbicularis oculi on both sides. All movements of both eyes restricted as shown in table. Pupils equal and active. Articulation normal. Can whistle. No weakness of limbs. Tendon-jerks brisk and equal. Plantar reflexes flexor. No sensory loss. Heart, lungs and abdomen normal. Blood-pressure 170/105.

EFFECT OF PROSTIGMIN 2.5 MG. (ROUGH ESTIMATES IN DEGREES).

| | Right | | Left | |
|------------|--------|-------|--------|------------|
| | Before | After | Before | After |
| Elevation | 0 | 5 | 0 | 0 |
| Depression | 12 | Full | 12 | Full—tires |
| Abduction | 10 | 15 | 20 | 20 |
| Adduction | 10 | 45 | 5 | 15 |

Orbicularis oculi very much improved.

Case for Diagnosis : ? Hilar Tuberculosis, ? Sarcoid.—F. DUDLEY HART, M.D., M.R.C.P.

N. F., male, unmarried. Aged 21.

Admitted to hospital June 1940 with what was thought to be fading erythema nodosum. His only symptom was aching of the legs on standing. The fading, raised, blue-red lumps were confined to the front of the shins and were completely gone in two weeks. He had no other symptoms and no loss of weight. Physical signs were completely absent at this time but for the fading skin condition.

Investigation.—A radiograph of the chest (fig. 1) showed enlarged hilar glands with calcification in the left hilum. Blood-count normal. Sedimentation rate 40 mm.% in one hour, taking some five weeks to return to a normal figure. Percutaneous tuberculin test negative, Mantoux 1:10,000, and 1:1,000 negative, only just positive to 1:100 (1 mg. O.T.).

The original diagnosis of tuberculosis had been abandoned in favour of lymphadenoma and he received X-ray treatment as for this condition with no apparent change in the X-ray appearances. He was discharged in November 1940, returned to work in a week, and since then he has remained completely free from all symptoms.

A routine radiograph 13.8.41 (fig. 2) showed hilar shadows little changed from the original picture but in addition a generalized mottling and bronchial congestion. At the time the patient was entirely symptom-free. Since then serial radiographs have shown a decrease in the mottling which was most marked in the right middle zone.

Blood-count (20.9.41): R.B.C. 5,110,000; Hb. 104%; C.I. 1.0; W.B.C. 6,200 (polys. 80%, eosinos. 1%, lymphos. 11%, monos. 8%). Sedimentation rate: 3 mm. at end of one hour.

Percutaneous tuberculin test negative. Mantoux negative 1:100 (1 mg. = 0.1).

No physical signs.

Past history.—Allergic rhinorrhoea in the months of June and July for the last four or five years, responding rapidly to benzedrine therapy.

In view of the mild nature of the illness with complete absence of physical signs and symptoms it is thought that sarcoidosis might be an alternative diagnosis.

Comment.—The skin eruption on the shins was fading and was not typical when this patient was first seen, but as the diagnosis of erythema nodosum had been made an X-ray of the chest was taken. The glandular enlargement demonstrated did not alter appreciably on prolonged deep X-ray therapy, but the later skiagrams appeared to show a

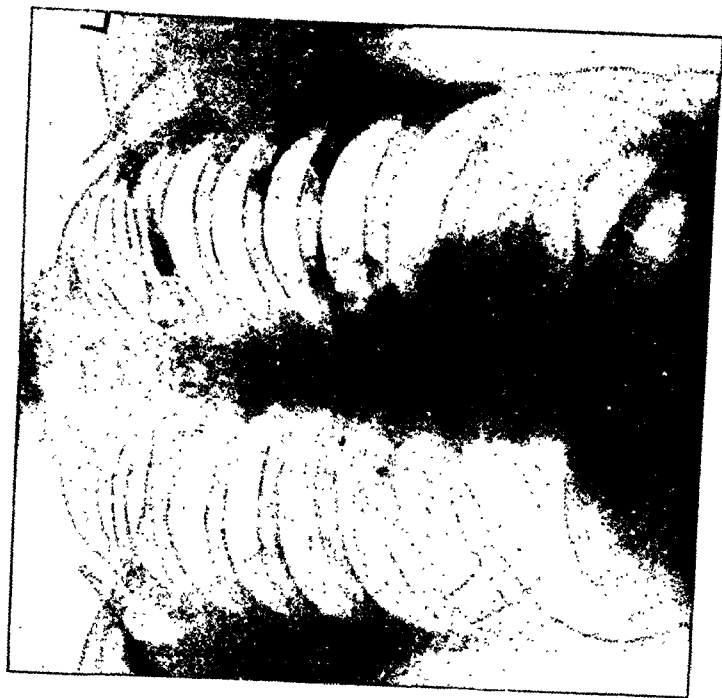


FIG. 2.—August 1941. Appearance of scattered lung lesion, most marked in the mid-zones, right more than left.

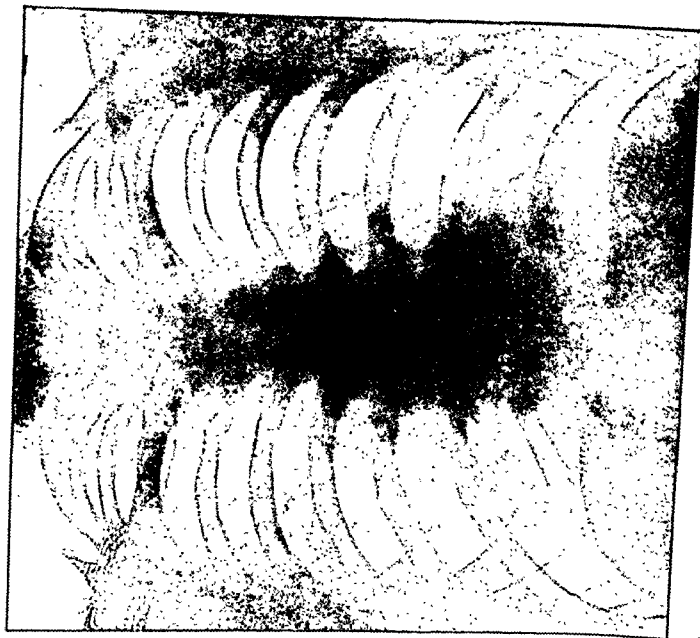


FIG. 1.—August 1940. Showing hilar glandular enlargement.

spontaneous improvement occurring at about the time of the development of the parenchymal lesions.

The case from many points of view corresponds more to a picture of sarcoidosis than tuberculosis or lymphadenoma, in that: (1) He has been symptom-free throughout. (2) At the time of the development of parenchymal lesions he was well and gaining weight and doing a full day's work; and (3) The distribution and spontaneous improvement in the chest lesions as demonstrated by serial skiagrams; (4) The failure to react more than faintly to tuberculin intracutaneously and negative percutaneous reactions to bovine and human tuberculin and to purified protein derivative.

Although many such reported cases of sarcoidosis finally develop widespread tuberculosis and appear to have been throughout due to the tubercle bacillus it is possible that this case may be different and turn out to be "non-tuberculous" sarcoidosis.

An old primary lesion is present at the left hilum. There are no signs of tuberculosis or sarcoidosis elsewhere in the body.

Dr. F. PARKES WEBER thought the case was an example of tuberculosis becoming manifest after an attack of erythema nodosum. He had never heard of any connexion between erythema nodosum and sarcoidosis. The allergic disposition of the patient, evidenced by the previous history of rhinorrhœa, suggested that the hilar shadowing in the radiogram might be greatly in excess of the actual tuberculous lesion.

Quiescent Acromegaly with Insulin-Resistant Diabetes.—F. DUDLEY HART, M.D., M.R.C.P., and RAYMOND GREENE, D.M.

Miss M. L., aged 32.

Menarche at the age of 15. Menses normal in amount and duration but sometimes irregular in time from one to six monthly intervals. In 1932 when 25 years old there started a period of four years' amenorrhœa. Treatment in Germany by her own doctor consisted of injections of progynon. In 1932 her practitioner in Germany noticed that her face and hands were becoming larger, the skin coarser and more dry, and at this time there was a sharp increase in weight. From 1932 to 1936 she was treated for the amenorrhœa as before, mostly by injections of progynon. The symptoms and physical signs of acromegaly continued and increased.

In 1936 glycosuria was discovered for the first time and was controlled at the start by a restricted carbohydrate diet. She noticed considerable weakness and had many boils. Some months later in July 1937 she came to England for two months and during this time felt weak and thirsty and complained of frequency of micturition. On returning to Germany she was admitted to hospital and was found to have considerable glycosuria and a raised blood-sugar. She was stabilized on 20 units soluble insulin, thrice daily, and this treatment rendered her urine practically free from sugar. She was referred to Dr. Guttmann of the Neurological Institute at Breslau. He found her to be a typical case of acromegaly with concentric diminution of both visual fields, especially the right. An X-ray showed enlargement of the sella turcica. An operation was performed by the frontal route. The pituitary was found to be replaced by a cyst filled with bright yellow fluid. This was opened. There was considerable œdema of the frontal lobes and a decompression was performed. She recovered rapidly from the operation and an immediate, but incomplete, relief from her signs and symptoms of acromegaly was seen. Since this time the acromegaly seems to have remained quiescent.

She first attended this hospital under Dr. Raymond Greene in the endocrine clinic and subsequently has been an in-patient on two occasions under Dr. Lloyd and Sir Stanley Woodward. Her diabetes is resistant to insulin and a blood-sugar curve with insulin given intravenously differs little from that in which isotonic saline was given instead of insulin. She occasionally becomes markedly ketosed and on the verge of diabetic coma. She has never been hypoglycæmic.

Her case is of interest in that acromegaly definitely preceded the onset of the diabetes. Radiographs of the skull remain unchanged.

Comment.—Many cases of acromegaly develop glycosuria and hyperglycæmia, some the symptoms of diabetes mellitus. In the last eight cases of acromegaly treated as

in-patients at this hospital three have had diabetic blood-sugar curves and two the symptoms of it—polyuria, polydipsia, &c.

In the literature on the subject some authors have found such cases in no way a different therapeutic problem from ordinary cases of diabetes, others have found them insulin-resistant. This case is unusual in that though apparently insulin-resistant she occasionally becomes markedly ketosed and suffers from mild diabetic coma. She has never been hypoglycemic.

Professor Himsworth has kindly performed certain investigations on this case. The centrifuged deposit of the patient's urine injected into rabbits has given no true evidence of producing insulin resistance in the experimental animals, although one blood-sugar curve was suggestive of a very small amount of anti-insulin in the urine, though not enough to be conclusive.

What is the best way of treating such a case? The acromegaly seems not only to be stationary but actually regressing while the diabetes is—if anything—getting worse. She has expressed her desire to have no deep X-ray therapy even if it is thought advisable. Is it possible that she is passing from a pituitary insulin-resistant diabetes to a more insulin sensitive "pancreatic" type as secondary changes take place in her islets of Langerhans?

Periarteritis Nodosa.—A. M. STEWART, M.D.

S. W., female, aged 5 years.

History.—Admitted January 31, 1941, with one week's history of sore throat and anorexia, with vague abdominal pain. Swelling of neck noticed on morning of admission.

Past history.—Nothing relevant.

Condition on admission.—Temperature 101.2°. Pulse 144. Respirations 52. Well-nourished child with general toxæmia, dry cough and slight stridor. Both sides of neck swollen and tender but no glands felt. Erythematous patch on wrist and chin. Throat slightly reddened. Small shotty subcutaneous nodules under abdominal wall. No other physical signs.

Progress.—Unremitting pyrexia for eight weeks (102°-99°), during which time child became increasingly ill. Developed intense pain in all the limbs, screaming when touched. At times large areas of œdema appeared in various sites and subsided, and erythematous patches came and went in the skin. The nodules in the abdomen disappeared but fresh crops appeared on various occasions. Dry cough persisted throughout. By the end of the eighth week there was extreme cachexia and alopecia; the skin was harsh and dry and contractures had developed in the limbs. Next week the condition began to improve; no fresh swellings appeared and the pains gradually ceased. By the thirteenth week the child was afebrile, had gained in weight and become alert and interested.

Improvement persisted until the eighteenth week when the child developed an impetiginous skin eruption (hemolytic streptococci). Two days later high temperature with painful swellings in both knees and scattered nodules in the skin in all areas. This time the nodules were accompanied by much greater skin reaction, the areas being red, brawny and swollen. They began with extreme tenderness, reached their maximum size in two to three days and then slowly subsided, but new areas appeared at the time. In the twenty-third week all the fingers were intensely swollen and painful. After these swellings had subsided gangrene developed in the tips of three fingers.

The after course, as previously, was characterized by fresh crops of subcutaneous nodules, continuous pyrexia and steadily increasing emaciation and lassitude. No further cough after the eighth week.

The child was taken home on September 18, eight months after admission. Throughout her illness there were no physical signs in the heart or lungs, no vomiting or abdominal pain other than that caused by obvious nodules or plaques in the skin.

Investigations.—Repeated urine tests: no microscopic blood; only an occasional trace of albumin. No organisms. Blood-counts: High polymorphonuclear leucocytosis (34,000—44,000 per c.mm.) except during the ninth to the fifteenth week. Steadily increasing

anæmia which improved rapidly during the remission and then relapsed. Biopsy: Subcutaneous tissue showing subacute inflammation with cellular exudate concentrated round the small arterioles and venules. Fibrinous necrosis of these vessels. Thrombosis not a feature. No organisms demonstrated. Condition: Periarteritis nodosa.

Mantoux and Widal's tests negative. X-ray examination negative. Electrocardiogram (eight months after onset) normal.

Polycythæmia Rubra Vera.—DOUGLAS ANDREW, M.D., D.M.R.

Mrs. L., aged 52.

1934: Complained of slight weakness in the legs and arms and noticed she had a high colour which was sometimes blue. At this time she also noticed she was intolerant to extremes of heat and cold. No digestive symptoms, though she had some pain in the chest on exercise relieved by standing still.

1935: At the beginning of the year she noticed some enlargement of the abdomen and had swelling of the ankles after standing. In April she had "influenza" and during this illness had sudden pain in the left side of the lower part of the chest lasting for some hours. She called in her own doctor, who discovered her splenic enlargement. She saw Mr. Cade in August, and in September was treated at Westminster Hospital with radium plaque.

R.B.C. 8,200,000; Hb. 120%; W.B.C. 19,200.

W.R. negative. Albumin present in urine. Indirect van den Bergh 1.25 units.

1937: Further treatment with radium plaque at Mount Vernon in July with good result. Further application of radium plaque at Mount Vernon in December with less striking result. The spleen was enlarging again and she had a return of symptoms of tiredness, weakness and excitability.

On examination.—Thin, marked malar flush, some cyanosis. No engorged veins in neck, no œdema of feet. Slight clubbing of fingers (this was noticed in 1935). Heart and lungs: N.A.D. B.P. 130/90 (same as in 1935). Abdomen grossly enlarged, edge of enlarged spleen visible. Spleen enlarged as far as umbilicus, stretching round to left anterior superior spine. Several notches palpable. Liver 4 in. below costal margin, quite smooth and regular. Liver and spleen tender, small para-umbilical hernia. No ascites. Central nervous system: N.A.D. Eyes: Some lens and vitreous opacities, fundi normal. Legs: Much pigmentation lower part of both legs, and cyanosis of feet with clubbing of toes (slight). She has been treated on this occasion with superficial X-ray and later phenylhydrazine hydrochloride gr. 1 daily for four days.

7.9.41: R.B.C. 7,090,000; Hb. 112%; W.B.C. 12,400.

8.10.41: R.B.C. 6,800,000; Hb. 110%; W.B.C. 5,000. Platelets 200,000. B.M.R. -1%.

Blood calcium 9.4 mg.%. Van den Bergh 1.25 mg.%. Urine: Albumin present. No casts. Sternal puncture September 18, number of cells normal, slight shift to the right in both erythrogenic and myelogenic cells. No evidence of myeloid transformation.

The interest in this case lies in the fact that all the treatment given has been directed to the spleen, contrary to the accepted practice of irradiating the long bones. In every case there has been a good response, judged both by diminution of the blood-count and also in the size of the spleen. Following treatment the patient always feels much better in herself. It would appear from the graphs of several blood-counts, however, that the condition is becoming less sensitive to radiation, as the fall in the count is slower and less marked.

Following the initial treatments the fall in the red count continued for long but varying periods after the cessation of treatment.

On admission on this occasion the liver was noticed to be enlarged, for the first time, and this enlargement has increased coincident with, and possibly due to, the administration of phenylhydrazine.

Ophthalmoplegia Co-existent with Active Thyrotoxicosis.—F. F. RUNDLE, F.R.C.S.

H. T., male, aged 35. Electrician. Married.

History.—Symptoms of thyrotoxicosis for one year, loss of weight nearly 1 st., palpitations and breathlessness on exertion, excessive sweating, irritability and attacks of diarrhoea. Eyes became prominent one year ago. Diplopia noticed only in the last two months.

Examination.—Moderate thyrotoxicosis (basal metabolic rate + 47%). Slight lid retraction and considerable proptosis. Limitation of upwards movement in both eyes, particularly the left.

Sir ADOLPHE ABRAHAM asked if he was right in supposing that ophthalmoplegia of some degree was more frequent in Graves' disease than we had supposed. It was only of comparatively recent date that our attention had been directed to the phenomenon and there seemed no necessity to suppose a separate syndrome as Dr. Russell Brain had suggested. He asked if the cause was a simple mechanical one or if some pathological alteration occurred in the ocular muscles.

Section of Urology

President—G. E. NELIGAN, M.C., F.R.C.S.

[October 23, 1941]

Case of Ureteric Transplantation into the Large Bowel and Total Cystectomy for Multiple Papillomata of the Bladder.—CLIFFORD MORSON, O.B.E., F.R.C.S.

November 1936: P. V. D., aged 54, admitted to hospital on account of hæmorrhage due to multiple papillomata of the base and internal meatus of the bladder.

Operation.—Suprapubic cystotomy and removal of growths with the endothermy current.

May 1937: Recurrence of growths with severe bleeding. The diathermy current was applied on many occasions, but it failed to control the hæmorrhage.

The patient became so exsanguinated that by August it was deemed advisable to perform complete cystectomy following transplantation of the ureters into the large bowel.

The latter operation was carried out at the end of August, both ureters being inserted at the same time into the pelvic colon by the method advocated by Henry Wade, except that no drain was left in the lumen of either duct. One week later the patient had complete control of rectal micturition and his general condition rapidly improved. Convalescence was uneventful.

In October 1937 complete cystectomy was performed and again the convalescence called for no comment.

Patient remained in good health until February 1940, when he developed pyelitis on the right side. This, however, subsided in a few days.

Four years have now elapsed since the ureters were transplanted and the bladder extirpated, and the patient is in excellent health. The rectum empties its contents every four to six hours but when the urge for evacuation comes the patient is unable to discriminate between fluid, flatus, and fæces.

Case of Impotence of Two Years' Duration in a Man of 54, Treated by Lowsley's Ribbon Gut Plication Operation.¹—TERENCE MILLIN, F.R.C.S.

T. S., aged 54.

August 1937 attended All Saints' Hospital complaining of complete impotence for two years. Urological and neurological investigations negative.

August 19, 1937: Lowsley plication operation under spinal anaesthesia.

Patient discharged from hospital three weeks later.

¹ *Proc. Roy. Soc. Med.*, 1935, 29, 817 (Sect. Urol., 27).

Painful erections experienced from day following operation. Successful intercourse ten days after leaving hospital. Regular intercourse during past two years.

After-effects of Plastic Operations in Two Cases of Hydronephrosis.—H. P. WINSBURY-WHITE, F.R.C.S.

(1) Male, aged 29. Von Lichtenberg plastic operation for left hydronephrosis with calices somewhat expanded was carried out ten years before, resulting in disappearance of symptoms for eight years; but during this time urine was hazy with pus and calices still dilated as shown by intravenous urography. Subsequently constant headaches for two years and urine as stated. Left nephrectomy advised and carried out.

Pre-operatively blood-pressure was 185/118.

One month after operation: No headaches; blood-pressure 140/80; urine free from pus and organisms.

Comment.—In spite of cure of symptoms result of plastic operation must be considered unsatisfactory as ultimately revealed by headaches and elevated blood-pressure.

The disappearance of these signs after nephrectomy showed that an important degree of toxæmia had been present. It is not easy to get a good result when the calices are already expanded before operation.

(2) Single, female, aged 31. Early right hydronephrosis: simple excision of redundant pelvis four years before.

Result: Symptoms gone; urine crystal clear, but a few coliform bacilli present. Intravenous urogram showed good function and no dilatation of pelvis or calices.

Comment.—It is not difficult to get a good result from a simple plastic operation on an early case of hydronephrosis.

Hydronephrosis Causing Œdema of the Legs.—ALEXANDER E. ROCHE, F.R.C.S.

In 1938 I was asked by Dr. Konstam to see a patient at the West London Hospital, diagnosed by him as having hydronephrosis.

Male, aged 40. There had been no loin pain, but, for four weeks, mid-abdominal pain one to one and a half hours after meals, nausea, two and a half weeks' pain in the right lower chest, and one week's pain, swelling and discoloration of the left leg, the pain commencing like sciatica in the left buttock, and thence spreading down the leg, and necessitating injections. There had also been bluish discoloration of the left flank and iliac fossa. On admittance the whole left lower limb had been swollen, and the skin bluish-red and warmer than on the right. However, by the time I saw him, nine days later, the left leg, pitting deeply, was swollen only up to the knee, but the right leg and thigh, the latter of almost elephant's size, were tense, and the skin, pitting deeply, was shiny and bluish up to the pelvis. The right buttock was bulging with œdema, and the right loin was congested and œdematous. There were engorged epigastric veins.

Filling the right loin, and extending down into the right iliac fossa and across to the left epigastrium, was a tense, fluctuating, football-sized mass. The urine was normal; Wassermann and hydatid tests negative; leucocytes 15,500, with slight daily pyrexia, often to 100° F.

Plain X-ray films showed the ascending colon shadow apparently coinciding with that of the descending colon, the right side of the diaphragm high, and the right psoas shadow obscured by a large vague blur. Left uroselctan outlines were normal; the right were absent.

Having never previously seen a hydronephrosis causing œdema of the legs from pressure on abdominal veins (nor had Mr. Sydney MacDonald) I at first thought the patient might have left small saphenous thrombosis (I could feel a median left popliteal cord) and right femoral thrombosis. However, this would hardly account for the œdema and congestion of the right loin.

Having once seen a right renal growth cause œdema of legs and scrotum by growing into the inferior vena cava, I suggested right ascending pyelography, but could not find

the right ureteric orifice after prolonged search, though the left was easily seen, in spite of the presence of varicose veins, which ran down to the regions of both ureteric orifices.

Against growth were the absence of hæmaturia and the maintenance of weight, and I hoped for, and on the whole believed in, hydronephrosis as a diagnosis, though still fearing growth.

On 16.3.38, via a long right paramedian transperitoneal incision, a large hydronephrosis was removed, weighing 8 lb. 4 oz., and a good view of the duodenum and inferior vena cava obtained. The ascending colon was practically in the mid-line of the body. The loin was stabbed, and a tube left in, but it drained practically nothing. The pulse-rate, 96 on the day after operation, rose within forty-eight hours to 144, taking two or three days to settle. However, the patient remained a good colour, and apart from a few days' hiccoughs, made a good recovery.

The day after operation the right thigh felt more lax, and a week after, it was normal, though the legs were still œdematous. They appeared normal on the patient's discharge from hospital twenty-three days after operation, and when last seen, ten days ago (13.10.41) the patient was well and symptomless.

There was no stricture in the four inches of ureter removed and a No. 7 French bougie passed easily. The undilated ureter was found looped over a leash of vessels, above which it gradually widened to nearly the diameter of a little finger, as it ran along the cyst to open into it about one inch internal to the vessel-crossing. The kidney was a thin, translucent, but tough shell, with two or three fibrous-looking septa.

Up to five years before operation patient says that he used to run 100 yards in just under eleven seconds, and cycle for twelve hours.

Hunner's Ulcer.—GEOFFREY E. PARKER, F.R.C.S.

The patient gave a characteristic history of severe stabbing pain on micturition over a period of four years.

On cystoscopy, the bladder showed only a mild degree of chronic cystitis, except on the left side of the fundus where there was an ulcer about the size of a shilling, the surface of which was bright and bled freely, and was sharply demarcated from the surrounding bladder wall.

Cystoscopy had only been possible under full ether anaesthesia, as the conscious patient could not tolerate more than 2 oz. distension, and even then with great pain. Treatment by repeated over-distension, as recommended by Kretschmer, seemed for this reason to be out of the question, and the ulcer excised without difficulty by suprapubic partial cystectomy.

The differential diagnoses of malignant and tuberculous ulceration were discussed, and the specimen of the ulcer, together with the microscopic sections were demonstrated.

COMMENT: Fenwick published a report in 1896 on a series of cases of bladder ulcer. Many of these cases had multiple ulcers, and these were all situated on the bladder base and not on the vault. They do not therefore correspond to the classical description first given by Hunner in 1915. Fenwick treated his cases by instillations of a strong solution of silver nitrate, and reported good results from the "electroplating effect".

Other authors discuss treatment by fulguration of the ulcer surface, but Folsom points out that there is a danger of producing a perforation through this devitalized portion of bladder wall, which would no doubt be disastrous as in the area affected the bladder is covered by peritoneum.

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Calcified Hypernephroma of Right Kidney of Eight Years' Known Duration.—H. M. GRANT, F.R.C.S.Ed.

Case history.—The patient was first seen in 1933, complaining of vague abdominal pain. A hard movable tumour was palpable in the right iliac fossa and X-ray showed a partly calcified sphere lying just above the right sacro-iliac joint (fig. 1). No operation was advised at this time.

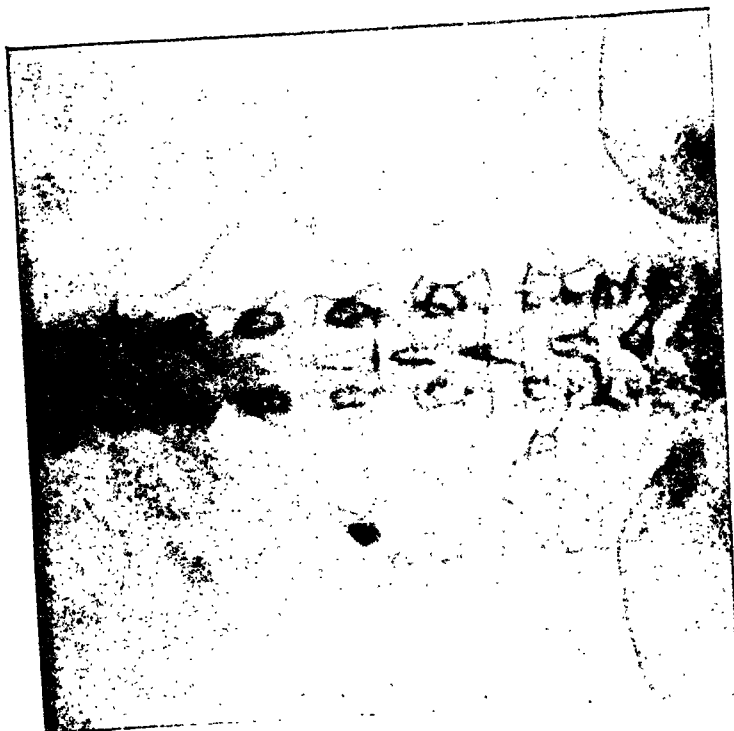


FIG. 2.—1941. X-ray excretion urography showing calcified sphere more definitely.



FIG. 1.—1933. X-ray barium meal, shows calcified sphere lying at a level just above the right sacro-iliac joint. (a and b indicate calcified sphere.)

She was seen from time to time, and in May 1941 another X-ray was taken, which showed considerable increased calcification.

A laparotomy was performed and the swelling was found to be retroperitoneal and apparently attached to the right kidney. The abdomen was closed after the removal of the appendix. An excretory pyelogram then showed that the calcified area was in close relationship to the lower pole of the right kidney. There was some elongation of the inferior calix (fig. 2).

A second operation was performed, and the mass was found to be attached to the lower pole of the kidney, almost pedunculated. The tumour was removed and the pathological report was as follows:

"A solid acinar and tubular type of hypernephroma with surrounding atrophy of renal tissue, cyst formation and blood extravasation. There are some patches of calcification in the dense hyaline fibrous matrix of the tumour tissue." (Fig. 3.)

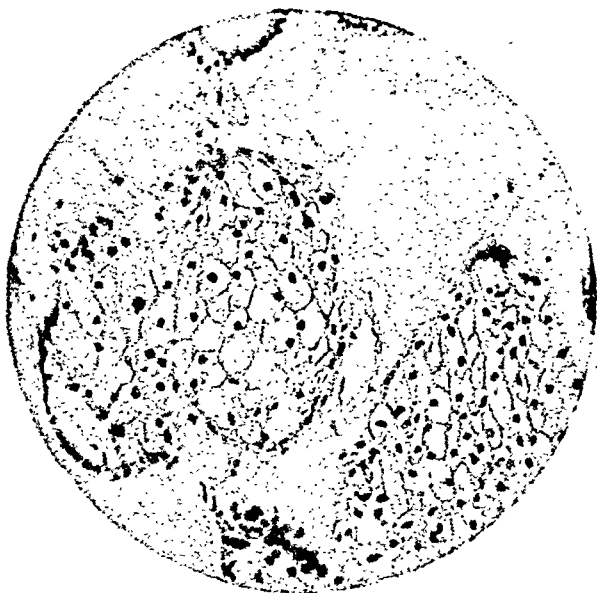


FIG. 3. $\times 250$.

COMMENT. *History.*—Points of importance in the history are: The long duration—the patient is known to have had the tumour for at least eight years; the absence of urinary symptoms; the difficulty in diagnosis owing to the very low position of the tumour.

Differential diagnosis.—Other conditions to be considered are: Calcified aneurysm of the renal artery or one of its larger branches; calcified solitary serous cyst; calcified cyst of spleen—this might be confused with a lesion of the left kidney; calcification in tuberculosis or rarely in chronic pyelonephritis; calcified hydatid cyst; it is possible that calcified mesenteric glands or pedunculated fibroids might also have to be distinguished; calcareous changes may also take place in simple adenomata or papillary cystadenomata (Mackenzie).

Prognosis.—The prognosis of malignant tumours of the renal parenchyma is not good. According to Mackenzie (1936) the five-year cures in various series ranges from 5-20%. Judd and Hand found 74 cases out of 283 i.e. 26% alive at the end of five years.

Calcification in tumours is generally considered to be a characteristic of those which are slow-growing and of low malignancy.

The significance of calcification in the prognosis of tumours of the renal parenchyma

has been discussed in two papers, one by Cahill and Melnikow (1938), and the other by Braasch and Griffin (1936). The former had 12 cases with calcification in a series of 82 tumours. Calcification occurred in all types—hypernephroma, papillary and even alveolar carcinoma. They found these cases had a very poor prognosis. Nine were dead within the first year, and after two years the remaining three were still alive but two had metastases and only one was without any evidence of metastases. Most of these tumours were large ones.

Braasch and Griffin noted seven cases in one series. At the end of three years six were alive, and at the end of five years four were alive i.e. 57%. This is very much higher than even the most favourable figure, as found by Judd and Hand, for all tumours of the renal parenchyma.

Hand and Broders (1932), in a statistical investigation of 193 tumours of the renal parenchyma, analyse the signs, symptoms, and operative and pathological findings and their relationship to prognosis. Calcification is not mentioned in this paper.

They place great emphasis on Broders' classification by cellular differentiation as a guide to prognosis. They found also, amongst other factors such as fixation, &c., that the size of the tumour was of great significance. The larger the tumour the less the expectation of post-operative life. The average duration of post-operative life of patients who had had large tumours removed, was half that of those who had had small tumours removed.

The technical difficulties in mobilizing large tumours and the danger of disturbances of the diaphragm, peritoneum and sympathetic nerves, all affect the immediate mortality, and the extra manipulation required in handling a large tumour, possibly dispersing malignant emboli, may affect the distant mortality.

Other factors such as fixation and perinephric involvement are also shown to be of grave significance, but I mention the prognostic importance of size especially, because Cahill and Melnikow note specifically that their tumours were large ones. It is probable that it is the size rather than the calcification which was significant.

At the last meeting of this section, Roche (1941) showed a case where a calcified hypernephroma was known to have been present for five years before operation. This case, which I have shown, was known to have been present for eight years. These long histories and the histological picture show that they were not, at least, rapidly growing tumours.

Calcification in itself, apart from any other features present in the growth, is likely, therefore, to be of favourable significance.

[This case was shown by kind permission of Mr. Z. Cope.]

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Mr. Alexander E. Roche also showed a specimen of papilloma of renal pelvis and of ureter.

Mr. Morton Whitby reported a case of recovery from non-gonococcal peri-urethral abscess with extravasation of urine and cellulitis of scrotum.

Section of Dermatology

President—MAJOR W. J. O'DONOVAN, R.A.M.C.

[April 17, 1941]

Chemicals in Fabrics as Potential Skin Irritants

By H. E. Cox, D.Sc., Ph.D., F.I.C.

[Manuscript unavoidably delayed, due to enemy action.]

THE study of the action of chemicals in fabrics as potential skin irritants is of interest to both the chemist and the dermatologist. It is by their combined efforts in research and practical clinical experience that our knowledge of such reactions will become wider and more accurate. The *modus operandi* of even a primary irritant is imperfectly understood and when the effects of sensitivity or idiosyncrasy are considered our knowledge is almost nil; facts almost more strange than fiction are duly observed and recorded, but the underlying reactions are unknown. Perhaps this is partly due to the incomplete mental equipment of most of us; so few have the many-sided competence necessary for such study. The chemist is usually without clinical experience and knows little pathology, and the physician or dermatologist generally has but a limited knowledge of chemical industrial materials and processes. So except for those rare individuals who have exceptional qualifications, real understanding must come from co-operative efforts and the free exchange of information. With such thoughts in mind I propose to discuss briefly some of the possible causes of dermatitis mainly arising from fabrics.

It has been well said that the modern miss is the patron saint of chemistry; she is adorned from head to foot by the art of the chemist, and it must be agreed that if she avoids excesses she does look very attractive. I want to present her in another less happy character, that of the experimental animal for which not even a vivisection license is required (but to whom large damages may be payable). The manufacturer aids and abets her in applying to herself all kinds of new compounds the nature and properties of which from our present point of view little or nothing is known. Small wonder that dermatitis cases arise and that their incidence is credibly reported to be increasing. Her hair having been bleached with peroxide and ammonia and shampooed with a soap substitute is dyed with a compound diamine. Her eyelashes may be the product of the grease pencil, her lips and cheeks may derive their beauty not from oxyhemoglobin but from eosin and ponceau; her teeth—natural or artificial—are brushed with a waterproofed synthetic fibre, her fingers are adorned with nitro-cellulose dissolved in complex solvents and plasticized with still more complex chemicals. Perhaps she has applied chloramine T or an oxyquinoline derivative to her axillæ or even used mercapto-glycollic acid as a depilatory. But I want to pass from these personal details to consider more particularly what she is wearing. Perhaps she has a corset made of latex rubber with fillers and antioxidants in it, a bleached woollen vest impregnated with synthetic finishes, other intimate garments made of cotton with synthetic resins as anti-crease, stockings of de-lustred viscose, a frock of rayon containing perhaps regenerated cellulose and cellulose ethers impregnated with

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The technical difficulties in mobilizing large tumours and the danger of disturbances of the diaphragm, peritoneum and sympathetic nerves, all affect the immediate mortality, and the extra manipulation required in handling a large tumour, possibly dispersing malignant emboli, may affect the distant mortality.

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peroxide, there is formed first Bandrowski's base and next an insoluble azine in and on the fibres. There is no evidence of the presence of the di-imides which were at one time suspected. the free p-phenylenediamine can be extracted with water, though it is very difficult to remove the last traces on a commercial scale. The Bandrowski's base can be extracted by pyridine and certain other organic solvents, and the evidence seems to be that Bandrowski's base is in fact harmless. So the examination of a suspect fur generally resolves itself primarily into a search for traces of unoxidized p-phenylenediamine and allied compounds. Frequently the colour of the dye is modified by a poly-phenol such as resorcinol, pyrogallol or quinol; in such cases an oxazine results instead of a simple azine [2], and again search must be made for phenolic compounds. There are of course other matters to be thought of: there may be an excess of acid or of salt; there may be traces of soluble chromate or chromic acid left from the oxidizing agent. Poisonous metals such as lead or antimony may also be present. The chemical aspect may be complicated by the presence of vegetable tannins, by logwood, fustic, gallotannic acid and other such products. If the fur has been chrome tanned different considerations arise: there is unlikely to be free diamine or amino-phenol but there may be soluble chromium compounds and textile dyes and their reduction products, the nature of which must be investigated.

WOOLLENS

Wool presents on the whole a more difficult problem than fur usually does: it may be treated in so many ways and with so many substances. It will be within the knowledge of many that both leading judgments ruling dermatitis cases in the Courts arose from woollen garments. In *Grant v. the Australian Knitting Mills* the plaintiff's skin was normal but the garment contained an excessive proportion of sulphite, whereas in *Griffiths v. Peter Conway* the plaintiff's skin was abnormal but the garment was normal. White wool, such as a blanket or underwear is quite commonly bleached with sulphite, and garments containing small quantities of sulphite must be worn by millions of people. The sulphite will not be completely removed by even two or three launderings. All such wool—indeed most woollens too—are acid [3]. How much sulphite and what degree of acidity is to be regarded as potentially dangerous? Undyed wool quite commonly shows 2 or even 2.5% of acid reckoned as sulphuric acid. The wool protein is itself acidic and there is evidence [4] that the disulphide ($R.CH_2.S.S.CH_2.R$) linkages in the keratin are continuously oxidized in light with the production of more acid in the form of sulphite and sulphate. In dyed wool some of the acid linkages have combined with the dye and 1% or less acid is found. The amount of sulphite found in normal cases too is somewhat variable; quantities such as 0.2 or 0.3% are common enough in bleached undyed wool. In dyed wools it is unusual to find any appreciable quantity [5].

Then there are the dyestuffs. It was at one time common to blame the dye in cases of dermatitis from a dyed cloth; often it may have been true, but nowadays most of the known deleterious dyes have gone out of use. Very fast insoluble dyes are now available and standardized tests for fastness and the absence of bleeding have been prescribed by the Society of Dyers and Colorists. Generally speaking it may be taken that the more fast and insoluble a dye the less likely is it to be an irritant [6]. So we must find what kind of dye has been used and whether it is fast to water and to perspiration. The identification of the dyes in a fabric is apt to be very difficult and mixtures are common, so it is not often possible to say exactly what has been used, but it is possible to discover the class of dye present. Here may I emphasize that such textile dyes as are usually applied to wool do not contain p-phenylenediamine: this substance is an intermediate and not a dye and is not applied to cloth (though it may have been used in the original manufacture of the dye). The oft-repeated suggestion that a piece of cloth contains p-phenylenediamine or had been dyed with it arises from a misconception of dyeing practice.

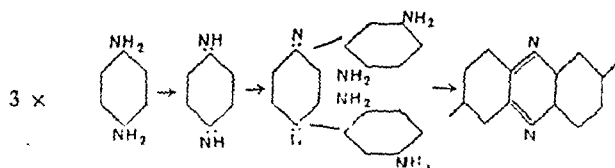
Many dyes are fixed to wool with the assistance of a mordant such as chromium salts. Others are after-chromed to improve lustre and fastness. Chromic acid and dichromate are potential irritants, so it is important to ascertain that there is no soluble chrome compound left in the material. Other less common mordants include copper, tin, nickel, zinc, and cobalt.

quaternary ammonium compounds to render them water-resisting; her shoes are of new plastics resembling snake skin and her fur cape is of leopard skin in which the familiar spots have been produced by mating a polyphenol with a diamine. Possibly she has spectacles of synthetic glass with sides of plastic resin, a handbag of suede leather compounded of cotton and rubber and an orchid made from doped and dyed cellulose in her button-hole, perfumed with otto of the laboratory, not attar of rose. She is in fact one grand experiment; small wonder that nature sometimes revolts. A rash appears; she goes to the doctor. I suppose the principal function of the doctor or the dermatologist is to effect a cure, but the matter certainly does not end there; he will be concerned to find out the cause of the outbreak if only to prevent a recurrence, and not infrequently there may arise questions of liability and of compensation. There is an increasing awareness of this problem and in truth it is a very difficult one, chiefly because of the variations in personal idiosyncrasy, sensitivity or allergy. In general there will be little difficulty in arriving at a shrewd suspicion in cases where the dermatitis is due to clothing and a patch test may afford strong confirmation or otherwise, but the matter cannot properly be left there. It is probable that the suspect garment is one of a kind worn by thousands of people; it may contain a known primary irritant; it may contain some new type of compound to which many or few people are susceptible; it may contain only substances believed as result of long experience to be quite innocuous but to which the individual is idiosyncratic. It is important to ascertain the facts in the interest of all concerned as well as in the interest of science. Only by accumulating known, well ascertained, facts shall we be able really to find what substances are harmless or harmful and how they react with the human subject. In a Report on dermatitis from wearing apparel by Dr. Schwartz of the U.S. Public Health Service, the Service has devised a questionnaire in which the importance is stressed of discovering the actual chemical in the fabric which is causing the irritation and on the methods employed for such determination. In this way certain potentially dangerous substances introduced in the States have been eliminated from use.

Recent legal judgments in this country too have tended to emphasize this aspect. The Court now requires to know whether the plaintiff's skin was normal or abnormal, or conversely—perhaps it amounts to the same thing—was the garment normal or abnormal; did it contain some trace of substances that ought not to have been there; was it reasonably fit to wear by a person not having an idiosyncrasy. So while the dermatologist is occupied with the patient's skin, the garment comes back to the chemist who must try to discover any abnormality or imperfection in the material such as would be likely to cause the troubles experienced. This is often a very difficult problem requiring much knowledge and skill; let me just indicate the possibilities or probabilities.

FURS

Furs present what is now perhaps the simplest case. The dyeing of fur by oxidation of a diamine such as paraphenylenediamine was discovered about fifty years ago, but only within the past twenty years has fur dermatitis become a recognized condition and its cause fairly well ascertained. The reason for the method used in dyeing fur is that while most dyestuffs must be applied by boiling, fur cannot be boiled without damage (unless it first be chrome tanned). So the colour must be produced by reactions on the fibres; these reactions are not usually quite complete nor is the pigment completely absorbed in the fibres, so there is apt to remain on the fibres (1) unoxidized base and (2) intermediate oxidation products of the base in addition to the real, believed harmless, pigment formed in the fibres.



I have described these reactions in detail in another place [1] and it will suffice here to point out that when a simple diamine such as p-phenylenediamine is oxidized by

peroxide, there is formed first Bandrowski's base and next an insoluble azine in and on the fibres. There is no evidence of the presence of the di-imides which were at one time suspected. the free p-phenylenediamine can be extracted with water, though it is very difficult to remove the last traces on a commercial scale. The Bandrowski's base can be extracted by pyridine and certain other organic solvents, and the evidence seems to be that Bandrowski's base is in fact harmless. So the examination of a suspect fur generally resolves itself primarily into a search for traces of unoxidized p-phenylenediamine and allied compounds. Frequently the colour of the dye is modified by a poly-phenol such as resorcinol, pyrogallol or quinol; in such cases an oxazine results instead of a simple azine [2], and again search must be made for phenolic compounds. There are of course other matters to be thought of: there may be an excess of acid or of salt; there may be traces of soluble chromate or chromic acid left from the oxidizing agent. Poisonous metals such as lead or antimony may also be present. The chemical aspect may be complicated by the presence of vegetable tannins, by logwood, fustic, gallotannic acid and other such products. If the fur has been chrome tanned different considerations arise: there is unlikely to be free diamine or amino-phenol but there may be soluble chromium compounds and textile dyes and their reduction products, the nature of which must be investigated.

WOOLLENS

Wool presents on the whole a more difficult problem than fur usually does; it may be treated in so many ways and with so many substances. It will be within the knowledge of many that both leading judgments ruling dermatitis cases in the Courts arose from woollen garments. In *Grant v. the Australian Knitting Mills* the plaintiff's skin was normal but the garment contained an excessive proportion of sulphite, whereas in *Griffiths v. Peter Conway* the plaintiff's skin was abnormal but the garment was normal. White wool, such as a blanket or underwear is quite commonly bleached with sulphite, and garments containing small quantities of sulphite must be worn by millions of people. The sulphite will not be completely removed by even two or three launderings. All such wool—indeed most woollens too—are acid [3]. How much sulphite and what degree of acidity is to be regarded as potentially dangerous? Undyed wool quite commonly shows 2 or even 2.5% of acid reckoned as sulphuric acid. The wool protein is itself acidic and there is evidence [4] that the disulphide ($R.CH_2.S-S.CH_2.R$) linkages in the keratin are continuously oxidized in light with the production of more acid in the form of sulphite and sulphate. In dyed wool some of the acid linkages have combined with the dye and 1% or less acid is found. The amount of sulphite found in normal cases too is somewhat variable; quantities such as 0.2 or 0.3% are common enough in bleached undyed wool. In dyed wools it is unusual to find any appreciable quantity [5].

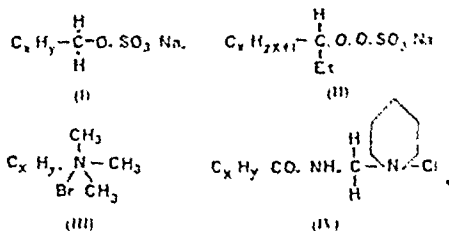
Then there are the dyestuffs. It was at one time common to blame the dye in cases of dermatitis from a dyed cloth; often it may have been true, but nowadays most of the known deleterious dyes have gone out of use. Very fast insoluble dyes are now available and standardized tests for fastness and the absence of bleeding have been prescribed by the Society of Dyers and Colorists. Generally speaking it may be taken that the more fast and insoluble a dye the less likely is it to be an irritant [6]. So we must find what kind of dye has been used and whether it is fast to water and to perspiration. The identification of the dyes in a fabric is apt to be very difficult and mixtures are common, so it is not often possible to say exactly what has been used, but it is possible to discover the class of dye present. Here may I emphasize that such textile dyes as are usually applied to wool do not contain p-phenylenediamine: this substance is an intermediate and not a dye and is not applied to cloth (though it may have been used in the original manufacture of the dye). The oft-repeated suggestion that a piece of cloth contains p-phenylenediamine or had been dyed with it arises from a misconception of dyeing practice.

Many dyes are fixed to wool with the assistance of a mordant such as chromium salts. Others are after-chromed to improve lustre and fastness. Chromic acid and dichromate are potential irritants, so it is important to ascertain that there is no soluble chrome compound left in the material. Other less common mordants include copper, tin, nickel, zinc, and cobalt.

TEXTILE AUXILIARIES

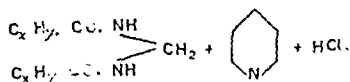
During recent years great progress has been made in the development of a large range of textile auxiliary agents. In this term are included wetting and levelling agents, dressing and de-lustering compounds, anti-creasing preparations, waterproofing substances, and various other chemical compounds, which improve the texture, feel, appearance, or properties of the fabric. There are reasons for thinking that some of the cases of dermatitis which have been attributed to dyes were in fact due to such a textile agent; as an example may be quoted a recent outbreak in the U.S.A. [7] which was proved to be due to a synthetic resin used as a dressing for textiles.

Most of these compounds depend for the effect upon a property, first discovered by Langmuir, possessed by molecules which consist of a long chain of hydrocarbons attached to a terminal polar group. (A polar group is an ionizable group of which one atom gives up an electron.) The polar group has an affinity for water, whereas the long chain has not, so the molecule when in contact with water stands up on end with the head, or polar group in the water and its tail stretching up. If the proportions are correct a monomolecular layer is so formed. If in such compounds the hydrocarbon group is short (say 12 C. atoms) the result is a wetting agent. In order to dye goods a level shade it is essential that they should be evenly wetted: a small proportion of a sulphonated fatty alcohol, e.g. sulphonated lorol or similar compound, enables the material to be wetted uniformly and quickly and so is a great aid to dyeing. When the carbon chain is somewhat longer, say 16 or 18 C. atoms, a valuable detergent property is developed and a washing compound free from alkali or fat is produced. Such compounds are the basis of soapless shampoos and are much used in the preparation of textile fabrics. The long chain compound may be a sulphonated fatty alcohol (i), or an ester (ii), or may be a quaternary ammonium compound (iii) or pyridinium compound (iv).



The physico-chemical properties of these compounds are truly remarkable and their uses diverse, so it is not altogether surprising that they should have rather marked physiological activities and be capable at times of causing severe skin irritation. I have had occasion to examine some of these substances; two properties stand out, one that they can pass through the skin and the other that they exert a marked haemolytic effect on red blood corpuscles. Such properties may be of significance from the dermatitis point of view. I do not know of evidence which would class them as primary irritants, but there have been sufficient cases of dermatitis following their repeated use to indicate that where susceptibility exists they are capable of being irritants. So it is clear that in our search for the chemical causes of the irritation we must find if any excess of such substances remains in the fabric.

Yet another class of long chain compound with a terminal polar group is used to make the fibres water resistant or water repellent. Such a compound may be a pyridinium compound of the type (iv), which upon being heated in the treatment of the fabric gives rise to a fatty amide and liberates pyridine and acid;



all these substances should be removed. Then there are synthetic resins used to give the fabric anti-creasing properties; they may be condensation products of urea and formaldehyde or of glycerol and phthalic acid. Formaldehyde if not entirely removed may irritate or may combine with ammonia to form hexamethylene-tetramine to which a few people appear to be sensitive [7]. These condensations in general tend to produce insoluble compounds of high molecular weight which are without any marked physiological property. There may, however, be residues of their constituents as impurities, and there is evidence that to some of them some people are susceptible. Cases arising from spectacle frames belong to this category.

Another synthetic resin used on textiles is ester gum, which is reported by L. Schwartz and others [8] to have caused quite a number of cases of dermatitis last year in the U.S.A. Ester gum is formed by heating glycerol with colophony resin, the principal constituent of which is abietic acid. Colophony is known to cause irritation in various other industries; it contains abietic acid and unsaturated acid anhydrides related to the terpenes, pinene and retene, which are possible causes of its irritant effects.

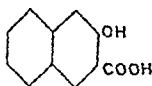
So an examination of woollens and other fabrics must include consideration of and tests for all these diverse and rather difficult compounds which may be implicated in any particular case, and it may be necessary to conduct patch tests on the separated suspect substance if complete proof is to be obtained.

COTTON AND SILK.

These fabrics are unlikely to contain acid or bleach, but they may be weighted with a variety of inorganic compounds such as tin salts or titanium oxide, not that these substances are harmful; and it will be necessary to consider the dyes which have been used and any traces of impurities arising therefrom, also to find out if any active chemical auxiliaries are present in an uncombined state. Anti-crease compounds such as urea-formaldehyde polymers and the glyptals are possibilities and water-resistant substances of the amido-pyridinium class may have been used; if so there should be no free acid or free pyridine or formalin. In the practical examination of these materials it is desirable to differentiate clearly the different kinds of fibres and so far as the dyes are concerned to examine different colours separately. The reason for this is of course that different classes of dye will be used for vegetable fibres and animal fibres. The use of suitable quaternary ammonium compounds greatly increases the fastness of the direct cotton dyes.

RAYON.

In my experience various kinds of rayon are suspected in more cases of dermatitis than the natural animal or vegetable fibres. Union fabrics are more likely to be concerned than single component fabrics and black is more likely than most of the colours. There are, however, considerations which modify the force of such general observations; for example, rayon is immensely popular, union fabrics are more difficult to dye uniformly than others and black is by far the most used colour. It is also the colour which requires a maximum quantity of dye and the one most frequently applied in re-dyeing an already other coloured garment. In examining a rayon garment it is particularly needful to consider what the fibres are made of. The type of dye applicable to acetate-silk may be quite different from that used, say, on viscose. Cellulose acetate was for some years difficult to dye, and new types of colour have been evolved since 1920 to meet the difficulty. Notable among these are the ionamines (1922) and insoluble azoic colours prepared by coupling aminoazo compounds with β -hydroxynaphthoic acid or one of its arylamides.



Traces of this β -hydroxynaphthoic acid are difficult to remove completely, particularly in the case of union fabrics, and in my experience a few cases of dermatitis have been traceable to such cause. In testing one has to avoid producing it by hydrolysis during

supersensitive and idiosyncratic individual. The law has recently been distinctly more helpful to industry in this direction, and that I think makes it still more desirable that we should together explore more thoroughly the relationship between chemical constitution and irritant propensities, together with the cognate subject of induced sensitization.

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President—H. C. SEMON, M.D.

[October 16, 1941]

DISCUSSION ON A SELECTION OF THERAPEUTIC PROBLEMS

ACRODERMATITIS (DORE)

Dr. S. E. Dore, who was asked by the President to speak on a condition to which his name was appended, said: I have never made any special claim to this condition. Some years ago at a meeting of the British Association of Dermatology and Syphilis¹ I described a short series of cases characterized by vesicular and pustular lesions on the thenar and hypothenar eminences of the hands and on the soles of the feet, the eruption being very chronic, relapsing, refractory to treatment, and sterile on culture. At the same time Dr. Barber wrote a much more comprehensive paper describing the histology of the disease which he subsequently included in his group of "pustular psoriasis" and showed cases in which the two conditions occurred together, a combination which I had never seen in my own cases.

Dr. Goldsmith typified the condition I had described as an eczema of the pompholyx type entirely different from psoriasis and coupled my name with it. Naturally I was somewhat surprised and gratified when Dr. Goldsmith called it by my name, but I am quite willing to abide by Dr. Barber's verdict regarding the nature and classification of the disease. It is a characteristic eruption in view of the peculiar "lacunar" pustules in the epidermis accompanied by eczematization, the special localization, the chronic history, refractoriness to treatment, and tendency to relapse. Dr. Barber pointed out that in some cases focal sepsis was an important factor in its causation, but in my cases I have not found this to be a prominent feature of the disease.

Dr. Prosser Thomas: So far as treatment goes, ordinary measures clear up the attacks temporarily. Crude coal tar in Lassar's paste is probably the most useful local application. Some of the dyes, such as gentian violet, are helpful, also silver nitrate, and I think Castellani's carbolfuchsin paint has been advocated. Whether or not the condition is related to pustular bacteride, it is important to find a focus of infection. It might be allowable to use one of the sulphonamide group of drugs in this condition as it may be directly or indirectly of microbic origin. I have not had an opportunity of doing so. I should suggest sulphathiazole. I have had at least temporary success with thorium X. The eruption is supposed to be resistant to X-rays and they have been known to make it

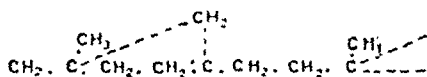
¹ *Brit. J. Dermat. & Syph.*, 1928, 40, 12.

the extraction and when it is found consideration must be given to the quantity. A few parts per million seem to be inevitable, but larger quantities may be found which are irritant to susceptible individuals. It is not a primary irritant, but one to which some people are sensitive.

Like cotton and silk, rayon is very frequently treated with a filler or some textile auxiliary compound. The material and its manufactures are so versatile that quite æsthetically beautiful new fabrics are constantly being produced, new colours, new textures, new properties giving nothing but delight to the vast majority of purchasers but unfortunately producing irritations in a few idiosyncratic individuals.

RUBBER GARMENTS.

In my experience quite a number of cases of irritation have arisen from the wearing of latex rubber often in immediate contact with the skin. Most people can do this with impunity and the garments last for quite a reasonable time. Breakdown ultimately occurs, the rubber loses its elasticity and becomes sticky. The curious feature is that this change is much accelerated by sweat and the sweat of some people is much more potent than that of others. Chemically the breakdown is really a polymerization of isoprene



resulting in the formation of a resin [9]. In terms of analysis this is marked by a sharp rise in the solubility in acetone. In a degraded rubber it may reach 20 or 25%, instead of some 3 or 4%. The active agent in the degradation is an oxidase present in the sweat of some people, and it is on this account that artificial sweat mixtures are not entirely comparable with the natural product. To counteract this natural tendency to oxidation many substances, known as antioxidants are or may be added. Usually these are complex hydroxylated phenols, amines or sometimes quinones. What are their physiological properties? Little is known, though in the case of quinone there is some evidence that it is harmless when fed to rats for several generations. Another aspect is that these oxidases and antioxidants are very potent substances; they are active when present in quite minute percentages and exert a most marked effect. Oxidation and reduction in the tissues is a fundamental biological activity and my suggestion is that any substance which inhibits it is *prima facie* suspect. As an example may be cited the familiar p-phenylenediamine, which has been shown by Keilin and others [10, 11] to exert a considerable influence on tissue respiration; if this is so is it not probable that di-β-naphthyl-p-phenylenediamine will be similarly active.

So one might continue reviewing the different garments—hats, shoes, brassieres, corsets, &c.—our modern miss may wear. In the manufacture of all of them potent chemicals have been used, and traces may remain which may be themselves irritant or to which our damsel may be supersensitive. But the point I really want to make is the need for more careful co-operative study between the dermatologist and the chemist, of the substances used on garments or otherwise in contact with the skin. One property these substances have in common, be they auxiliaries, dyes, antioxidants or what, is that their value depends upon special reactivity. They are substances of which a fractional percentage exerts a great effect; they can wholly alter the physical properties of the fibres or the penetrating effect of dyes. It has been shown that some of them are intensely active when in contact with blood or serum, so it is not surprising that sometimes unwanted effects develop on the human subject. Unwittingly Miss Blank may thus become our experimental animal. This ought not to be; the physiological properties of substances applied to our outsides as well as to our insides ought to be very carefully explored and in general the results if favourable should be made known to all concerned; if unfavourable then, of course, the substance is unfit for use. The great difficulty is arranging for a sufficient number of experiments to complete the necessary research: it is not difficult to discover the properties and reactions of a new product, nor is it difficult to detect and eliminate the primary irritants, but it is very difficult to foresee and provide against the

formed, in which new pustules continue to appear. In this stage there may be a close resemblance to pustular psoriasis, but even so the patches are usually less sharply defined and more eczematoid in appearance, and the scales are less dry and silvery than in this condition. The two eruptions, however, have this in common, that the contents of the pustules dry up to form brownish scabs, which are gradually exfoliated in the parakeratotic horny layer. If there is a difference clinically, I would say that these tend to be larger and of a deeper colour in pustular psoriasis.

The first case which led me to believe that focal infection might be responsible for the condition under discussion was that of a young girl who had been under Dr. Dore's care, and he had recognized it as being an example of his acrodermatitis of mild type. At that time I was quite ignorant of the cause, and did not connect the eruption either with psoriasis or focal sepsis. Looking back I think it was a case of pustular bacteride, and not of pustular psoriasis. When she came to me she had had the eruption for eighteen months and I tried various treatments without success over a further period of two and a half years. The pustules were sterile on culture. She was exceptionally healthy except that she was subject to recurrent attacks of tonsillitis, and she herself observed that at these times the pustulation of the hands and feet became much aggravated. I therefore suggested that the tonsils should be enucleated, and this was done. The effect was dramatic. There was an acute exacerbation of the pustulation twenty-four hours after the operation, but in about a fortnight the eruption had almost disappeared. There was a slight relapse six months later after a sore throat, but since then she has remained well. I have seen several other cases in which an equally rapid cure has resulted after enucleation of the tonsils or treatment of other major foci of infection, but in some improvement is gradual and subsequent treatment with one of the sulphonamides or vaccinotherapy is indicated.

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Dr. A. C. Roxburgh: I regard "acrodermatitis perstans" as comprising at least two conditions, possibly three, viz. pustular psoriasis, pustular bacteride and possibly a peculiar form of eczema. At least I have seen it in a few patients who had typical patchy eczema elsewhere.

For the pustular bacteride no treatment short of removing the septic focus is any good. For the other types White's Tar Paste is as good as anything.

I think pustular psoriasis tends to be more localized and the pustular bacteride more diffuse, but it is difficult to draw a hard and fast line in every case.

Dr. Twiston Davies: The majority of cases I see do not appear to fall into the groups just described and I can hardly remember a case in which either the dental surgeon or the aurist could be persuaded to discover septic foci, and certainly none in which removal of alleged focal sepsis did any good. I agree that the lesion may be closely simulated by both eczema and psoriasis, but I am sure that this is an entirely separate condition which characteristically affects the ball of the thumb and instep of the sole symmetrically and in comparatively circumscribed patches. No speaker has yet mentioned arsenic, which, given in doses gradually increased to 8 or 9 drops of Fowler's solution three times a day, and combined with the cignolin paste originally suggested by Ingram, will always greatly improve these cases. I have never seen a case that got completely well.

worse. I applied thorium X in alcoholic solution, 1,500 electrostatic units to the c.c., at ten day or fortnightly intervals and the condition cleared up but I was unable to follow up the patient for any length of time.

Dr. H. W. Barber: Some years ago, in conjunction with Dr. Whitteridge, I read a paper (unpublished) before the British Association of Dermatology on "Cases of Pustular Bacteride", and I expressed my views, which I have since seen no reason to change, on this condition and on the subject of a previous paper—Pustular Psoriasis of the Extremities [1]. I referred to the history of these conditions which may be summarized as follows:

(1) Dr. Dore [2] in 1927 read an excellent paper entitled "Notes on Cases of a Chronic Mild Localized Type of Acrodermatitis Perstans".

(2) Dr. Roxburgh [3] showed two characteristic cases at the October meeting of the Dermatological Section in the same year.

(3) At the Association Meeting in 1930 [4] I read a paper in which I differentiated between "Acrodermatitis Continua (Hallopeau) vel Dermatitis Repens (Crocker)", which had been the subject of a previous communication by Eyre and myself [5] in 1927, and the eruption under discussion, and I maintained that the latter was really a pustular form of psoriasis affecting the palms and soles, as a rule symmetrically.

(4) At the International Congress of Dermatology held at Budapest in 1935, G. Clinton Andrews made a communication upon "a group of cases of recalcitrant pustular eruptions of the palms and soles in which focal infections appear to play an aetiological role". With Birkman and Kelley [6] he had published in the previous year, a paper on the same subject, and with Machacek [7] in December 1935 one entitled "Pustular Bacterides of the Hands and Feet" which was an elaboration of his communication at Budapest. It was clear from his description and from the photographs submitted that the condition corresponded to Dr. Dore's acrodermatitis of mild type, but some of Clinton Andrews' cases were almost certainly pustular psoriasis of the extremities.

(5) I then recognized that I had been wrong in thinking that this symmetrical pustular eruption of the palms and soles is *always* a pustular form of psoriasis, but I maintained that in many cases it is so.

I summarized my views as follows:

(a) There are two groups of cases, which have this in common, that the eruption tends to occur symmetrically on the palms, particularly on the thenar and hypothenar eminences, and on the central parts of the sole, and that it is characterized by the formation of pustules, the contents of which reveal no micro-organisms in a state of active growth and are sterile on culture except for accidental contaminations.

(b) In both groups the cause of the eruption is a focal infection. I would go so far as to say that the presence of the eruption is certain evidence that the patient has somewhere an important focus of infection. By far the commonest site is the throat; this was also the experience of Andrews and his collaborators. It is probable, judging from my own experience, that in the great majority, if not in all cases, the responsible organism is a streptococcus: in other words that the eruption is a pustular or, as will be noted later, sometimes a vesiculo-pustular-streptococcide, and that the leucocytic invasion of the skin, which produces the pustules, is a response to streptococcal toxin, or possibly to an actual bacteremia, coming from a distant focus.

(c) The essential difference between the two groups of cases is this, that in one the patients are psoriatics, in the other they are not.

(d) I pointed out that in the psoriatic cases the pustular element is, so to speak, engrafted on patches of psoriasis, and these may be actually confluent with patches of ordinary psoriasis on the sides of the palms and soles. In some cases the patient may have had ordinary psoriasis for years, in others the pustular patches are the first to appear and subsequently a widespread non-pustular psoriasis may develop. In pustular bacteride, on the other hand, the eruption is eczematoid in type, as Goldsmith and Freudenthal pointed out in one characteristic case, and Andrews compares it to the vesicular epidermophytide. As the eruption becomes chronic, red scaly patches are

Occlusion with strapping, providing some degree of rest and protection to the affected area of skin has been advocated. I have not had experience of this method, but Franklin [4] has had favourable results.

Some cases of granuloma annulare show slight atrophy when they heal; this is not surprising when there is such extensive destruction of the collagen seen in some sections. Many of the methods of treatment may add to this atrophy, e.g. blistering doses of ultra-violet light and carbon dioxide snow.

X-ray treatment in half skin doses, is often effective but the disease may recur, and in my opinion it is not justifiable to repeat such doses, particularly in children. Thus in one case quoted by Michael, 2½ pastille doses were given in a single dose without benefit.

Gold therapy has been suggested, but the use of this dangerous agent is not indicated. The same comment could be made on the use of sulphonamide, and the results quoted by Coombes and Canizares [5] have been unfavourable.

In an attempt at specific therapy, those who favour the tuberculous ætiology of granuloma annulare, have injected Old Tuberculin locally with favourable results. The risks of such therapy are not great. The success, as in other methods, may well be the production of a local inflammatory hyperæmia. In one case, reported by Michael [6], a subcutaneous injection of tuberculin (0.05 c.c. of 1:1000 dilution of Old Tuberculin) given for a relapse of granuloma annulare, resulted in a marked local lymphangitis and there was a moderate systemic reaction. The granuloma annulare disappeared although no focal reaction appeared.

The subcutaneous injection of tuberculin should be criticized on the grounds that it might produce a general reaction and activate a tuberculous focus, present in a number of people. Such a focus may or may not be of significance in the ætiology of the granuloma annulare.

Believing in the possible streptococcal ætiology, Barber and I have treated our cases by the injection of hæmolytic streptococcal vaccine into the lesions of granuloma annulare. This method has the merit of safety, and I do not remember any evidence of more atrophy than that which might be expected in the natural resolution of the condition. The results are good; in six cases followed up, a cure was obtained in periods of two weeks to four months. The time was prolonged in cases showing numerous lesions or in patients who attend irregularly, for only a small number of lesions were treated at any one time. The patients who were sensitive to the vaccine and therefore showed a marked local reaction gave the most dramatic results.

Lastly, there is the question of the prevention of relapses of granuloma annulare. Bearing in mind the possibility of a tuberculous ætiology, one would recommend a regime designed to increase the patient's immunity, but I have not found any study of comparative series of cases of granuloma annulare to indicate that this general therapy is, in fact, successful in preventing recurrences. Nor does there seem to be any published series of cases comparing the different methods of treatment mentioned above, as to their relative success in treatment and the liability to recurrence.

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Dr. John Franklin: Whenever I see a new case of granuloma annulare I put a piece of elastoplast around the part for a week or fortnight; this makes the lesion disappear, but it always returns within a few weeks. I have never succeeded in curing a case permanently by strapping.

Dr. F. S. Airey: Some members may recall a case of this disease which I showed before this Section in April 1940¹. The child had a greatly enlarged cervical gland, which

¹ *Proc. Roy. Soc. Med.*, 33, 582 (Sect. Dermat., 6).

Dr. J. E. M. Wigley : I have also seen cases in which the tonsils were condemned and removed, without any benefit to the patient, even after a considerable time. With short treatment arsenic has not produced any appreciable improvement.

Dr. F. Hellier : A short time ago I saw a man with an injury in his palm which had developed into a lesion resembling acrodermatitis. There was definitely a history of trauma and I considered the condition to be dermatitis repens. My particular problem was to decide whether this was an industrial disease or not. How does one distinguish between the condition which follows trauma and is usually on the fingers and this other condition which is more common in the palms and is definitely spontaneous in origin?

The President : The case might be cultured. A positive result should decide the question between acrodermatitis and industrial dermatitis.

Dr. H. W. Barber : I think the case to which Dr. Hellier referred must have been one of true acrodermatitis continua of Hallopeau, which corresponds to the dermatitis repens of Crocker. This condition was studied by Professor Eyre and myself. There is nearly always a history of injury, and from its site a pustular eczematous eruption spreads, and may become generalized. The investigations of Professor Eyre, Dr. Embleton, and myself suggest that a virulent strain of *Staphylococcus aureus* is responsible, which grows in media usually inimical to staphylococci.

GRANULOMA ANNULARE

Dr. L. Forman : Discussion of the therapy of any disease should be guided by its known or projected aetiology, which, in this disease, is still unsettled although most attention has been directed towards its relation to tuberculosis. Tubercle bacilli have been found in sections on rare occasions only (Dittrich [1]). Most of the arguments are based on associated visceral and skin tuberculosis. Thus three cases of Halliwell and Ingram [2] had lesions of lupus vulgaris, and occasional references are made to associated papulo-necrotic tuberculide and sarcoid. Two cases out of seventeen of mine had significant findings. One girl of 20 years who had had granuloma annulare for four years developed a pleurisy, not proved to be tuberculous. Another girl had X-ray evidence of enlargement of the hilar glands; again not proved to be tuberculous. Tuberculin reactions recorded show a high proportion of negative results but the discussion of their significance would be out of place here.

X-ray examination of the chest of a series of cases by Epstein [3] did not give any significant evidence in favour of tuberculosis.

In my series of 17 cases, five showed clinical evidence of infection of the nasopharynx (four had septic tonsils, and the fifth a chronic antrum). Four cases gave a strong positive reaction to hæmolytic streptococcal vaccine injected intracutaneously, so that a coccal aetiology might be argued.

It is accepted that granuloma annulare does often spontaneously remit and that relapses are frequent, usually at the edges of the original lesions. The patients are in good health with no general toxæmia—there is no loss of weight or energy—and the development of any frank tuberculous disease must be uncommon. I have come across one case in a limited search of the literature, of a papulo-necrotic tuberculide appearing a year after the appearance and treatment of granuloma annulare.

We are dealing, therefore, with a benign condition; if it is associated with visceral disease (tuberculous or otherwise) these lesions remain cryptic and show no subsequent extension or systemic involvement.

A tuberculous focus should always be looked for, the sedimentation rate estimated, and the appropriate general treatment instituted. Further, the possibility of a septic focus should be remembered. One case, a girl of 8 years, with numerous nodules on the limbs, improved when the nodules were injected with streptococcal vaccine, but did not clear up until enlarged and chronically inflamed tonsils were removed.

Should then the lesions be treated at all, in this benign and often self-limited condition? The answer might be "Yes", if only for cosmetic reasons. The array of successful methods of treatment in use, indicate their non-specificity.

Dr. F. Hellier : There is a great histological similarity between granuloma annulare lesions and the rheumatic nodules. This is interesting because granuloma annulare occurs on the extensor surfaces and has other points in common with rheumatic nodules. The French believe that rheumatoid arthritis is caused by tuberculosis, and on that basis Forestier started to treat arthritis with gold. Although we do not admit the theory, he had successful results, and it might be suggested that granuloma annulare would respond to gold treatment.

Dr. A. M. H. Gray : Dr. Hellier may have seen granuloma annulare which had subcutaneous nodules and which are indistinguishable clinically from rheumatic nodules. I have shown a case of the kind here myself. I concur in Dr. Barber's view as to the relation between erythema multiforme and the persistent type of granuloma annulare.

Dr. J. E. M. Wigley : I should like to quote Dr. Sequeira, who had an enormous clinical experience, and whose teaching was that although occasionally some theory was put forward about granuloma annulare being associated with tuberculosis, in his experience the condition almost invariably resolved spontaneously, although it might take a long time to do so. In that connexion it is unjustifiable to risk incurring telangiectasis, for condition which in the large majority of cases gets well without any treatment.

The President : I agree that it is unjustifiable to risk giving X-rays or any other form of cicatrizing treatment in a condition which eventually clears up spontaneously.

SO-CALLED MONILIA INFECTIONS OF THE NAIL FOLD

Dr. Godfrey Bamber : Everyone here is well acquainted with this condition, although only so far back as 1927 Dr. MacCormac wrote that he believed this type of monilia infection to be of singular rarity. Monilia can be cultured from smears taken from beneath the affected nail folds and from the solitary bead of pus that occasionally appears in the majority of cases; but occasionally no fungus is found, only bacteria, most often staphylococci, but sometimes other organisms such as *B. coli* and *B. proteus*.

Whether this monilia infection is the primary lesion is debatable; first because the dermatologist seldom has the opportunity of seeing and making cultures from the lesion soon after its onset. The swelling of the nail fold and its separation from the nail plate have generally been present for weeks and even months before the patient seeks expert advice. Secondly monilia is not uncommonly present on apparently normal skins, and so may be a secondary invader. On the other hand the affection is almost entirely confined to women—I can recall only three men, two chefs and a potman—who handle foodstuffs that harbour and nourish monilia. Another interesting point to which Dr. Twiston Davies first drew my attention is the relatively high incidence of cold cyanotic hands in this type of patient.

Where the affection is already present, one of the early signs of extension to hitherto unaffected nail folds is the separation of the fold from the nail plate. Trauma from the misuse of orange sticks and cuticle removers has been suggested as a cause of this separation, but many hospital patients do not use these cosmetic aids. A slight separation of the nail folds is common in occupational dermatitis when the eczematous reaction extends to the distal phalanges, but the sulcus is not so deep, and the bolsterlike swellings and the acutely painful exacerbations characterizing the so-called monilia infections seldom occur.

If the infection be monilial, it is curious that it never seems to lead to moist erosion of the upper layers of the epidermis on and in the nail fold, a feature of the same infection in intertriginous areas; on the contrary the grooves often look dry and hard, and the firm bolsterlike swellings give the impression that the inflammatory reaction is chiefly in the subcutaneous tissues. The degree of reaction may perhaps be due to the virulence of the infection or to a supersensitive state of the tissues. With the latter alternative one might expect to find agglutination and complement-fixation reactions in the blood, but investigations on these points have not given conclusive results. Intradermal reactions with iodimycin are likewise unreliable diagnostic aids.

was a fluctuant mass at the time of the demonstration. It preceded the eruption of typical granuloma annulare, which was scattered over the face, limbs and trunk. Some of the lesions cleared up spontaneously, to be followed by others in various sites. The child also had what I preferred to call lichen scrofulosorum, on the trunk and arms. The surgeons were reluctant to touch the gland mass and I was anxious to see if the lesions would respond to conservative treatment. However, Dr. Gray suggested that the gland mass should be removed and this was done. Resolution of the lesions of granuloma annulare soon followed. Carbon-arc light baths were given and after six months all trace had gone. But the eruption of lichen scrofulosorum persisted. It was suggested that these lesions were actually commencing granulomata, but they never proceeded as such; the eruption remained stationary. I think in this case it was quite clear that the aetiology is likely to have been tuberculous, in view of the satisfactory response to therapy.

Another case of granuloma annulare showed a tuberculous dactylitis in the vicinity of the lesion, which was on the hand. The granuloma responded well to contact X-ray by Chaoul's method. Treatment was left to a radiotherapist, who gave a larger dose than is our habit. In my last six cases this method of treatment has been employed; there has been slight telangiectasis in some, which is regrettable, but all have shown a good response.

Dr. R. Klaber: We have allowed ourselves to become obsessed with the question of the tuberculous aetiology. It seems to be generally agreed that there is a small percentage of cases which do show such a definite tuberculous background, just as there is a similar percentage in cases of lupus erythematosus. But we have already passed the stage when we have been content to consider only that possibility in lupus erythematosus. If there is no such evidence we look elsewhere. It is very interesting that Dr. Barber and Dr. Forman should already be looking for streptococcal foci and it may be that these will be forthcoming in a further percentage of cases. But in our present ignorance we might well look still further afield and consider other possibilities, however rare or remote.

Two years ago Dr. Brain showed a case here which had occurred in a diabetic (*Proc. Roy. Soc. Med.*, 32, 1403 (Sect. Dermat., 79)). I have seen two cases in diabetics. So far as I am aware, there is no literature on the possible connexion between diabetes and granuloma annulare. There is a further diabetic interest in the occasional clinical and constant histological resemblance between granuloma annulare and necrobiosis lipoidica diabetorum. All the early cases of necrobiosis lipoidica were associated with diabetes, but there is now a long list of cases without this association.

I have recently seen a case of granuloma annulare which occurred at the site of rupture of a finger tendon, soon after the occurrence of this injury. Although in most cases of granuloma annulare no history of trauma is obtained, it does seem possible from the predilection of the lesions for the knuckles and other bony points that minor traumata might play some role in causation.

Dr. H. W. Barber: Granuloma annulare must be regarded as belonging to a group of eruptions that includes erythema multiforme, erythema annulare centrifugum, and lupus erythematosus. One may regard each of these eruptions as a specific entity and due to an infection as yet undiscovered, or that it is a non-specific reaction to circulating bacteria or toxins which differ in different cases.

Although in a considerable proportion of cases erythema multiforme is due to streptococcal sensitization, it is frequently associated with recurrent herpes simplex, and some of my cases have led me to suppose that it may be at times a generalized reaction to the herpes virus. The association was pointed out in a paper by Dr. Forman and Dr. Whitwell some years ago (*Forman, L., and Whitwell, G. P. B. (1934), Brit. J. Dermat. & Syph.*, 45, 309). The case I have in mind is a woman who for long has been subject to recurrent attacks of the two eruptions. I saw her once in an attack, and again about a fortnight later by which time one of the lesions of erythema multiforme had become transformed into a typical patch of granuloma annulare. Dr. Gray confirmed my observation. The same transformation has occurred occasionally since then.

activity of pathogenic organisms which would otherwise lie dormant. I think treatment might usefully take the form of applying hot acid solutions, e.g. liq. alumin. acet. B.P.C., 1 in 20 aq., followed by a shake lotion containing 35 p. zinc oxide, 15 p. glycerine and 2% liq. alumin. acet. ad 100 p. That gives a very good lotion which adheres to the nail fold and leaves behind a powdered film covering and protecting it. If this combined treatment is applied twice a day it lasts very well in the majority of cases, though in some cases X-ray treatment must be used in addition. It is important to instruct the women not to practise manicure while undergoing treatment.

Dr. Elizabeth Hunt: This condition is not confined only to refugee women who are now doing housework for the first time in their lives, for I have found a number of cases of this kind in women at home who have been forced as a result of the war to undertake numerous household tasks to which they have hitherto been unaccustomed. My worst case was in a woman who was doing all her own housework for the first time. Working women accustomed to run their own homes, frequently I find, use a scrubbing or very hard brush for their nails, after washing up kitchen utensils, and damage the cuticle in this way, leaving a portal for infection. One interesting case who did housework and much sewing in her leisure hours had recurrent trouble in the nail of the middle finger on which she wore her thimble. When she tried wearing the thimble on her ring finger, the trouble developed there also. The difficulty in treating some of these cases is that they cannot attend regularly at hospital. I advise them to use Milton, and soak the finger nails at night, reducing the strength of the solution as the trouble disappears.

The President: There seems to be general agreement that this condition is really a traumatic one, and in view of that we should consider the question of abandoning the name "monilia infection of the nail fold" altogether. It seems to be a question of maceration, which we can compare with the results of maceration leading to intertrigo in other parts of the body. The essential thing is to instruct these patients to avoid the cause of their trouble. They must be instructed not to put their hands in water, and that is a criticism of the last speaker's suggestion of soaking in Milton. I have found that any method which involves wet treatment predisposes to the perpetuation of the condition.

ECZEMA OF THE EXTERNAL AUDITORY MEATUS

Dr. F. Hellier: Discussing eczema of the external auditory meatus is rather like considering psoriasis of the left elbow or lichen planus of the leg, for eczema is an abnormality of the whole body and not a localized lesion. As Brocq said: "*il n'y a pas un eczéma mais eczémateux*". There are, however, certain anatomical peculiarities about the external auditory meatus which distract one's attention from this fundamental concept and my endeavour to-day is to try and indicate the relative importance of the local and general approach.

First one must consider exactly what one is dealing with in an eczematous lesion of the ear. A doctor consulted me about a year ago with an irritating and scaling rash of the aural orifices. He told me that the condition had cleared whilst he had been away on holiday owing, as he thought, to the improvement in his general condition but it returned when he got back to his practice. I was able to demonstrate that he was sensitive to the bakelite ear pieces of his stethoscope and a change to rubber ones cured his rash.

Another type of eczematous rash, of which the few cases I have seen have been in men, is a form of neurodermatitis due to the patient's habit of fiddling with his ear. This responds in the same way as a neurodermatitis elsewhere to a tar paste and X-ray treatment. But the majority of cases which one meets are really manifestations of seborrhœic eczema with or without lesions elsewhere.

What part does infection play in the production of such an eczema of the external auditory meatus? The frequent association of otitis media with lesions on the ear and scalp has led some dermatologists to overemphasize the infectious aspect. But if one approaches the condition from the point of view of the otologist, one finds that in the very large number of cases of discharging ear which he sees, it is only the exceptional case that develops eczema of the meatus; in other words it is only in the predisposed patient, the patient with the seborrhœic make-up, that an eczema occurs. In such a

Treatment.—The only early example that I have seen, where the nail folds had been affected for less than a week, healed rapidly when treated with zinc ionization, but this method has failed in chronic cases. When there are painful exacerbations patients generally resort to fomentations and at this stage some form of heat is perhaps the most soothing remedy. I have not tried short-wave therapy. For chronic cases my routine is to show the patient how to carry out the treatment with a wisp of wool round the end of a match stick cut to a chisel edge dipped in phenol or liq. iodi mritis diluted one in four with industrial spirit. The space below the fold is well mopped out. If the side of the nail plate is involved this is cut or scraped away and the exposed areas painted also. Finally the groove is sealed with collodion so that the patient can carry on her work without getting dirt or liquids into the spaces. This treatment is carried out daily until the inflammatory stage has subsided, and then, if necessary, four applications of X-ray, $\frac{1}{3}$ pastille B, are given to flatten the unsightly swellings. The time necessary to cure the patient is probably about three months.

Dr. A. C. Roxburgh : These cases are at least as often due to a pure culture of *B. coli* or the staphylococcus or streptococcus, or a mixed infection, as to monilia. My own practice is to give a $\frac{1}{4}$ pastille dose of X-rays once a week, or a $\frac{1}{2}$ pastille dose once a fortnight, up to a total of 2 pastilles spread over two months. I also prescribe Castellani's fuchsin paint which is pushed down with an orange stick to a point short of causing pain. The condition in my experience nearly always clears up, but it is essential for the patients to keep their hands out of water during treatment.

Dr. C. H. Whittle : I started collecting cases about five years ago, and I believe trauma to be the major factor in their production. The first ones which I encountered were in youngish women. I agree with what Dr. Bamber has said about the sex incidence. I have never seen a case in a man. After a long struggle with various measures, including antiseptics and X-rays, it suddenly dawned on me that I was not really dealing with any infection at all, but with a deliberate or semi-deliberate trauma of the fold. I satisfied myself on the point by strapping up the fold with elastoplast, marking it, and watching it for a fortnight, and as I expected, when the elastoplast was removed, the condition had nearly healed. In that case I learned afterwards that the patient had had to go into a mental home. She recovered and when I saw her later she admitted that she had deliberately, or semi-deliberately as a habit, constantly been worrying the nail fold. It happened in this case—and I have noticed the same thing in two or three other cases—to be the ring finger which was involved, and the second case was in a woman unhappily married. I present that to the psychologists for explanation. In both these cases I was impressed by the fact of trauma.

Another group, which Dr. Bamber mentioned, is a diffuse condition affecting most of the fingers. In the cases I have just referred to only one or two fingers were affected, but in what is known as "washerwomen's" cases the condition involves nearly all the fingers. As to the treatment of the "washerwomen's" cases, the dermatitis of the hand which involves the fold as well, I have always been in difficulties, and I am grateful for Dr. Bamber's suggestion to seal the groove with collodion. If that can be kept on I think it will solve the problem.

Incidentally, the first case I had was one of infection with *B. proteus*. That was just at the time when monilia was beginning to be thought of as a prominent cause of paronychia. Since then I have encountered a variety of organisms including staphylococci, coliform bacilli, monilia, the nature of the infective agent appearing to be a matter of small moment.

Dr. F. W. Jacobson : Many refugees who are now engaged in domestic service and are using their hands for washing and other housework, to which they have hitherto been unaccustomed, are showing this condition. They dip their hands into more or less strong alkaline solutions, and there are cases in which the "acid mantle" is destroyed by the alkali, so that ubiquitous micro-organisms, previously more or less dormant, suddenly become pathogenic. Many of these women, although engaged in household service, still manicure their hands and try to keep up a pleasing appearance of the fingers and finger nails, and thus we have a combination of the factors of trauma and the arousing into

activity of pathogenic organisms which would otherwise lie dormant. I think treatment might usefully take the form of applying hot acid solutions, e.g. liq. alumin. acet. B.P.C., 1 in 20 aq., followed by a shake lotion containing 35 p. zinc oxide, 15 p. glycerine and 2% liq. alumin. acet. ad 100 p. That gives a very good lotion which adheres to the nail fold and leaves behind a powdered film covering and protecting it. If this combined treatment is applied twice a day it lasts very well in the majority of cases, though in some cases X-ray treatment must be used in addition. It is important to instruct the women not to practise manicure while undergoing treatment.

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What part does infection play in the production of such an eczema of the external auditory meatus? The frequent association of otitis media with lesions on the ear and scalp has led some dermatologists to overemphasize the infectious aspect. But if one approaches the condition from the point of view of the otologist, one finds that in the very large number of cases of discharging ear which he sees, it is only the exceptional case that develops eczema of the meatus; in other words it is only in the predisposed patient, the patient with the seborrhœic make-up, that an eczema occurs. In such a

predisposed patient, however, infection is of the utmost importance both as a precipitating and as an aggravating factor, and as long as the infection remains there will be little chance of clearing up the eczematous condition permanently. Moreover this abnormal make-up extends beyond the skin to the mucous membranes and these patients are peculiarly prone to catarrhal conditions in and around the nasopharynx. It is surprising how often one discovers infected antra, &c., not only in those cases with an actual perforation of the drum and an otitis media but in those whose drums are quite sound, and I always transilluminate the antra of all patients with seborrhœic manifestations on the head. We therefore must conclude that if we are to treat a case of seborrhœic eczema of the auditory meatus fundamentally, we must not only treat his seborrhœic make-up but also deal with any sepsis which may be found in the ear, nose or throat. Unfortunately one does not always get much assistance from the aural surgeon as despite numerous operations, the infection often persists and the patient is no better. I believe that the infection in these cases is difficult to clear from the very fact that they are seborrhœics with unduly vulnerable mucous membranes. The treatment of aural and nasal infections is beyond my sphere but I have had several cases with chronic otitis media who have benefited from ionization of the middle ear.

I cannot enter fully into the general treatment of the seborrhœic, but will just mention that a reduction of carbohydrates and fats is indicated in severe cases, a precaution that Lord Woolton has already enforced on all of us. Much more important are environmental conditions and I have seen the most intractable cases clear when they have been sent into the country to lead a healthy, fresh-air life. Failing this one should try daily artificial sunlight to the whole body and give extra vitamins in the diet.

Turning to local treatment, it is important to clear up any eruption on the face or scalp and here quinolor compound ointment is often of great value. In the external auditory meatus, however, I have seen many patients suffering from the too enthusiastic use of strong antiseptics and I also believe that ointments should be avoided as far as possible. Recently gentian violet has become very popular in the treatment of acute eczema and I consider this to be one of the most important advances in local therapy of recent years. I assume that it acts in part as an antiseptic and in part by precipitating the proteins in the serous exudate. Anyhow it is my first choice in a moist eczema of the ear, used as a 1% watery solution. As an alternative one can use silver nitrate which acts in the same way, either as a 2% aqueous solution or in sp. aeth. nit. If the condition is so acute that the meatus is almost occluded, I use glycerine and ichthyol which reduces the œdema. In more chronic, dry cases I employ calamine liniment to which is added 1% carbolic acid or 5% of ichthyol. If the above fail and one is driven to use an ointment, this should be introduced sparingly on a wick of gauze which is retained in the ear till the next dressing. If this is not done the external auditory meatus gets clogged with a sticky mass which is difficult to remove without irritating the skin. I use various ointments but I prefer a not very stiff tar paste containing 2% benzoic acid. Eventually one often has to recourse to X-rays. It is difficult to see how they affect the deeper part of the meatus but their action is definite and one reason may be that the eczematous process starts in most cases in the outer part of the meatus and only reaches the deeper part when the condition becomes very severe or chronic. It is therefore important to treat these cases early and use X-rays whilst they are most likely to be of assistance. Lastly one must add that these cases often resent the blandest applications so that one cannot lay down exact rules, and in actual practice one's therapy is frequently guided more by subconscious dermatological instinct than by any rule of thumb.

Dr. J. D. Rolleston: Although Dr. Hellier has mentioned the importance of regarding infections as a cause of eczema of the external auditory meatus, I am surprised he did not mention a particular infection, namely, diphtheria. I have seen so many cases of young children with diphtheria in which the external auditory meatus has been affected that I think something ought to be said about it [1]. The condition may occur with diphtheria of the throat or it may be an isolated condition localized to the meatus. I should suggest that, particularly in the case of young children, before any further measures are taken, a culture should be obtained and then antitoxin could be given if necessary and the condition may be expected to clear up rapidly.

In the *British Journal of Dermatology* in 1916 [2] I related how a boy was treated for

a long time for a troublesome whitlow and then it occurred to me that I might cultivate the whitlow for diphtheria bacilli. I found a pure culture, and after an injection of antitoxin it cleared up in a few days, showing that diphtheria of the skin may simulate other lesions.

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Dr. Agnes Savill: Dr. Alfred Friel did great pioneer work with zinc ionization for chronic otorrhœa. He had numerous clinics for this ionization in connexion with the L.C.C. When this discharge leads to impetiginization of the auditory canal, ionization is indicated. Indeed it is often dramatic how, in one or two sittings, the swelling which blocks the entrance and causes deafness can be reduced. A dose of about 5 milliampères for four minutes is usually sufficient.

Dr. Twiston Davies: I am glad to hear of a satisfactory treatment for cases complicated by perichondritis. I am impressed with the frequency with which eczema of the auditory meatus is a sequel of impetigo. The best treatment consists of the insufflation of a powder—2 drachms of tincture of iodine to an ounce of boracic powder—after syringing out debris and drying the meatus. The patient is instructed to roll a piece of paper into a tube, dip one end into the powder and get a friend to blow it down his ear—very unhygienic, but efficient and cheap. If further treatment is necessary, thorium X often succeeds. The best form in which to use it is the ointment, 1,000 units per gramme. A thin smear is not enough; a quarter of a gramme must be put in to get any result. I have never seen any resulting reaction from this ointment.

Dr. P. C. P. Ingram: Like Dr. Davies, I have noticed the association of this condition with impetigo among soldiers whom I have had under my care recently.

EXFOLIATIVE ERYTHRODERMIA

Dr. R. Klaber: This condition is scarcely a clinical entity, only a clinical picture. It is the end-result of several distinct disease processes. Therapeutic considerations require its division into some of its known types. In the first of these groups, one would clearly place those cases resulting from irritative treatment of an eczema or psoriasis. Next, there are those cases which may follow the injection of arsenicals (especially N.A.B.) and of the heavy metals. Then there are those cases associated with disorders of deeper tissues. Finally, we have the primary forms described by Hebra, and by Wilson and Brocq. And to those well-recognized groups, I might add a fifth, where the exfoliation and erythema seem to be secondary to generalized hyperidrosis and pruritus.

The treatment of the first two groups, namely, those due to external irritation or internal medication, is largely preventive. With reference to the reticulo-endothelial group, a differential blood-count and sternal puncture may sometimes, if rarely, provide useful information. I think, however, we should go a little further than that. The lymph nodes are very commonly enlarged and if in all such cases a gland were removed and referred to Dr. Robb Smith's Lymphnode Registry at Oxford, it is possible that some increased knowledge might result.

In the light of our present knowledge—or rather our present ignorance—of the causes of this condition, treatment must usually be empirical and symptomatic. I believe Dr. Adamson used to have some faith in a lotion of glycerine and rosewater. With regard to general treatment, I have read papers in which the writers have been impressed by the results following intravenous glucose, or sodium thiosulphate, or liver extracts. More recently, various vitamins have been employed, in the hope of influencing the course

predisposed patient, however, infection is of the utmost importance both as a precipitating and as an aggravating factor, and as long as the infection remains there will be little chance of clearing up the eczematous condition permanently. Moreover this abnormal make-up extends beyond the skin to the mucous membranes and these patients are peculiarly prone to catarrhal conditions in and around the nasopharynx. It is surprising how often one discovers infected antra, &c., not only in those cases with an actual perforation of the drum and an otitis media but in those whose drums are quite sound, and I always transilluminate the antra of all patients with seborrhoeic manifestations on the head. We therefore must conclude that if we are to treat a case of seborrhoeic eczema of the auditory meatus fundamentally, we must not only treat his seborrhoeic make-up but also deal with any sepsis which may be found in the ear, nose or throat. Unfortunately one does not always get much assistance from the aural surgeon as despite numerous operations, the infection often persists and the patient is no better. I believe that the infection in these cases is difficult to clear from the very fact that they are seborrhoeics with unduly vulnerable mucous membranes. The treatment of aural and nasal infections is beyond my sphere but I have had several cases with chronic otitis media who have benefited from ionization of the middle ear.

I cannot enter fully into the general treatment of the seborrhoeic, but will just mention that a reduction of carbohydrates and fats is indicated in severe cases, a precaution that Lord Woolton has already enforced on all of us. Much more important are environmental conditions and I have seen the most intractable cases clear when they have been sent into the country to lead a healthy, fresh-air life. Failing this one should try daily artificial sunlight to the whole body and give extra vitamins in the diet.

Turning to local treatment, it is important to clear up any eruption on the face or scalp and here quinolor compound ointment is often of great value. In the external auditory meatus, however, I have seen many patients suffering from the too enthusiastic use of strong antiseptics and I also believe that ointments should be avoided as far as possible. Recently gentian violet has become very popular in the treatment of acute eczema and I consider this to be one of the most important advances in local therapy of recent years. I assume that it acts in part as an antiseptic and in part by precipitating the proteins in the serous exudate. Anyhow it is my first choice in a moist eczema of the ear, used as a 1% watery solution. As an alternative one can use silver nitrate which acts in the same way, either as a 2% aqueous solution or in sp. aeth. nit. If the condition is so acute that the meatus is almost occluded, I use glycerine and ichthylol which reduces the oedema. In more chronic, dry cases I employ calamine liniment to which is added 1% carbolic acid or 5% of ichthylol. If the above fail and one is driven to use an ointment, this should be introduced sparingly on a wick of gauze which is retained in the ear till the next dressing. If this is not done the external auditory meatus gets clogged with a sticky mass which is difficult to remove without irritating the skin. I use various ointments but I prefer a not very stiff tar paste containing 2% benzoic acid. Eventually one often has recourse to X-rays. It is difficult to see how they affect the deeper part of the meatus but their action is definite and one reason may be that the eczematous process starts in most cases in the outer part of the meatus and only reaches the deeper part when the condition becomes very severe or chronic. It is therefore important to treat these cases early and use X-rays whilst they are most likely to be of assistance. Lastly one must add that these cases often resent the blandest applications so that one cannot lay down exact rules, and in actual practice one's therapy is frequently guided more by subconscious dermatological instinct than by any rule of thumb.

Dr. J. D. Rolleston : Although Dr. Hellier has mentioned the importance of regarding infections as a cause of eczema of the external auditory meatus, I am surprised he did not mention a particular infection, namely, diphtheria. I have seen so many cases of young children with diphtheria in which the external auditory meatus has been affected that I think something ought to be said about it [1]. The condition may occur with diphtheria of the throat or it may be an isolated condition localized to the meatus. I should suggest that, particularly in the case of young children, before any further measures are taken, a culture should be obtained and then antitoxin could be given if necessary and the condition may be expected to clear up rapidly.

In the *British Journal of Dermatology* in 1916 [2] I related how a boy was treated for

expresses any special anxiety she may happen to be feeling for them, in this manner. With regard to treatment, erythrodermia is high on the list of conditions which are better not treated at all, at any rate locally. One difficulty of course is that these patients generally feel the cold and are also intolerant of contact with bedclothes. I find that they appreciate an arrangement of fracture cradles with electric light bulbs inside. I treated three cases, which I happened to have under my care simultaneously, with liver extract and it made all of them very much worse. With the same series I tried every kind of diet I could think of, and came to the conclusion that they did much better if allowed to choose themselves what they wanted to eat including even alcohol. They usually have enormous appetites, and they react badly if restricted. I should like to know a little more about treatment by rest. I had a patient not long ago who was just beginning an erythrodermia and I advised rest. This did not prevent generalization, and after six months in bed he was not making any progress. He then announced that whatever happened he must go back to his business which entailed travelling up to town every day and standing in a shop. He did so, and within about four weeks the eruption had disappeared. One wonders if it is really worth while to rest these patients.

Dr. A. M. H. Gray: The majority of these patients after a period of rest insist on going back to work. My own experience is that it does not make much difference whether they are in hospital or at work.

Dr. P. C. P. Ingram: In cases of toxic dermatitis I attach considerable importance to the use of injections of calcium thiosulphate repeated daily for several days or even longer if necessary. It is well tolerated and it seems to do a definite amount of good.

Dr. H. W. Allen: Most of the cases of erythrodermia due to idiosyncrasy to metal-containing drugs are the result of the combined action of drug and pre-existing skin infection, the drug acting as a precipitating factor where, usually, seborrhœa is the pre-existing condition. As for local treatment, calamine liniment, b.d., and some substitute for olive oil for the removal of scales, are practically a *sine qua non*. Increasing percentages of ichthyol are added to the liniment at a later stage if the pityrosporon is present.

Dr. John Franklin: I have recently had a man in hospital with N.A.B. dermatitis, and although he was quite ill, his appetite was phenomenal. The sister complained that on ordinary diets she could not give him enough to eat.

Dr. W. N. Goldsmith: I wonder if it is the common experience that the vast majority of these cases of obscure erythrodermia are in men. There is a certain type seen in old men with great œdematous thickening of the skin. In one case extensive biochemical investigations were made on blood and urine but no explanation was found for the œdema. In this and several similar cases most benefit was derived from injections of salyrgan. Perhaps in these old people there is some deficiency in the action of the kidneys.

THE TREATMENT OF HERPES RECURRENS

Dr. Hugh Gordon: Herpes recurrens is a condition with a known ætiology—a virus. Virus diseases as yet do not respond to known immunological rules, and therefore although we might expect to have a specific treatment, none has yet been firmly established. Most of us will remember some six years ago a most interesting paper by Dr. Brain at Sheffield on specific desensitization in herpes recurrens. The methods he reported seemed too complicated for routine treatment of the disease, and I fear that I have no data on this subject which I would care to record. I hope, however, that some other members will be able to tell us of their experiences in producing active or passive immunization.

Ordinary Jennerian vaccination has long been recommended as a treatment. My experiences in this respect are disappointing. One case previously unvaccinated appeared to be cured, though her reaction was worse than the disease. Three others previously unvaccinated did not benefit. I tried a small series on those previously vaccinated by repeated small vaccinations without any success.

of exfoliative dermatitis. It seems to me that some patients, like those described by Wilson and by Brocq, get better after a considerable time, while others, like those seen by Hebra, after a varying period, slide gradually or suddenly "downhill".

Dr. Elizabeth Hunt : I have had a severe case of exfoliative dermatitis following medication with stilboestrol. The interesting point of the case was that the condition started with loss of the axillary and pubic hair. My most recent case could not be classified under any of the categories mentioned by Dr. Klaber. This was a woman of over 50 with an acute eruption involving the whole of her body and accompanied by oedema of the limbs. No physical cause for her eruption was discoverable and treatment with increased vitamins and other nutritional methods had but little effect. Then I came across a paper in which the writer suggested that these cases are due to pancreatic insufficiency and stated that he had treated a number of cases successfully with insulin. The glucose tolerance test revealed a very slight deviation from normal; there was at no time glycosuria, but the patient was put on small doses of insulin, and forthwith the skin condition began to improve and within a few weeks it was possible to discharge her to a convalescent home. Shortly after her return to her own home when the skin was quite well this unfortunate woman took her life because she thought she was not wanted. She had been very distressed by her appearance when in hospital and depressed at times. It is possible that an unstable mental condition in this instance had been affected adversely by war conditions and that the skin affection had a psychical origin.

Another of my cases was a young woman in her twenties with an acute generalized eruption whom I treated unsuccessfully for months in hospital, until a mild epidemic of influenza broke out in the wards. The other patients recovered, but this girl developed a high temperature with a cough and on examination of the sputum tubercle bacilli were found. No clinical signs of pulmonary tuberculosis had been discovered by physical examination.

With this new complication the skin eruption resolved in the most marvellous manner in a very few days. I have in consequence asked myself if tuberculin would have helped this case.

Dr. A. M. H. Gray : I do not know what to do with people with the third type of erythrodermia, which has often been going on for several years before they are seen. They may have a normal blood-count or perhaps some increase of white cells. It is extremely rare to find any of these cases associated with frank leukaemia, though there is no doubt that such association occurs. The vast majority of these cases, those unassociated with any preceding eczema or psoriasis, never seem to show any characteristic signs of disease at all except in the skin. Some of them occasionally clear up by themselves, but the vast majority are very intractable.

Dr. H. W. Gordon : With regard to treatment of exfoliative dermatitis from metallic poisoning, removal of 200 c.c. of blood was recommended in Vienna. In other erythrodermias sometimes injections of liver have seemed helpful—perhaps from an underlying avitaminosis.

Dr. Wigley : The intravenous injection of T.A.B. vaccine has been useful in some cases. If a temperature of 103° to 105° is produced in a patient two or three weeks running the skin does seem to clear sufficiently to get them out of hospital, but there is little doubt that they relapse sooner or later.

Dr. Twiston Davies : This condition is becoming more frequent, though perhaps not in its classical forms. The two varieties I see most often are: one in which only part of the surface is affected, for example the entire lower extremities and nowhere else, or symmetrical areas of varied distribution; and another in which a universal eruption develops acutely and clears up in a few weeks or months. The latter type is often associated with physical or emotional trauma. One of my patients is constantly falling and breaking her leg, and each time she does so she gets an attack, on at least one occasion preceded by enlargement of the inguinal glands. Another, who has two sons in the war,

definite benefit is obtained, the course can be repeated once every year up to a total of 900 R. for any one area, spread over three to four years.

All other local treatment is palliative and does not pretend to be preventive. Dabbing the early lesion with spirits of camphor or eau-de-Cologne is usually recommended. I am indebted to Dr. Clara Warren for the suggestion of 1/1,000 adrenalin. If the lesions do not dry up quickly or tend to become purulent, they are best treated on the lines of impetigo, since secondary infection is common.

Dr. J. D. Rolleston : This subject has interested me for many years. In 1907 [1] I wrote a paper on herpes facialis in diphtheria in which I made the observation that herpes occurred in 4% of diphtheria cases, and in 1910 [2] I wrote on recurrent herpes in scarlet fever which I found to be present in 6.5%. What I should like to know is why herpes recurrens should be so frequent in certain infections and so rare in others. The cases in which herpes recurrens is frequent are cerebrospinal fever, malaria, and pneumonia, but in typhoid fever it is practically non-existent. I have brought up to show to the Section the illustrations of two cases of herpes recurrens in the acute stage of diphtheria [these were handed round]. One of these was reported by Dr. C. I. Wright [3] on herpes recurrens accompanied by bullæ, in a girl of 8 years of age who during the three previous years had had attacks of herpes recurrens. The other was a case which I showed in the Children's section [4] of a boy aged 13, who during the acute stage of diphtheria developed herpes, and had had numerous attacks of herpes recurrens previously.

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Dr. A. C. Roxburgh: A German writer once claimed to have treated 100 consecutive cases of recurrent herpes successfully by inoculating the serum from a young herpetic lesion on to some site in the arm. I have tried this method in a few cases and, much to my surprise, two or three of them did "take" and the patients stated later that they had been clear for two to three years afterwards. It is essential to use the serum from a perfectly fresh vesicle.

The President: It is some years since I had the opportunity of treating a case of facial herpes recurrens by the auto-inoculation method, and in that instance the result was dramatic. Some three days after the primary inoculation in the forearm a typical herpetic vesicle developed in situ, and from this I inoculated the other arm with a similar though less active response. A third transfer proved negative. Each inoculation was carried out intradermally. To minimize the risk of coccal infections an early vesicle, containing the presumably more active virus, should be secured. Goldsmith ("Recent Advances in Dermatology", p. 377) quotes the classical case of Plesch, who cured himself by this means, and refers to an extensive series of successful results published by Hruszek, *Derm. Zeit.*, 1933, 68, 27 (Abs., *Brit. J. Dermat. & Syph.*, 1934, 46, 296).

Miss Lewis : I have tried auto-inoculation, but could not obtain a vesicular response. I have also prepared and used a formalized guinea-pig vaccine in a small series of cases, but for a true assessment of the value of these methods, a properly-controlled, large-scale investigation is indicated.

Dr. F. W. Jacobson: In some patients this herpeticiform rash gives the appearance of erythema multiforme. A well-known causative agent is phenolphthalein, and nine-tenths of all the purgative drugs on the market contain phenolphthalein.

Dr. Elizabeth Hunt : I had a case of severe herpes recurrens on the face in a hospital sister, due to sepsis at the apex of one of her canines, completely cured by extraction of the tooth. So far I have not come across any case of recurrent herpes of the female genitalia at the menstrual periods. I have had one case of repeated attacks on the lip at

There are, I think, some more or less well-marked clinical types of the disease. Since the virus factor is presumably the same in all of them their different characteristics may bring to light independent activating factors which might be of some importance in treatment. They are thus worth considering.

I would classify these types as follows:

(1) *Herpes recurrens of childhood*.—The lesion is usually localized to approximately the same area of the cheek. It occurs usually once or twice a year from the ages of 3-12 years. It appears to cure spontaneously at about the time of completion of the second dentition.

These facts might suggest some association with the second dentition either from infective or pressure causes. I do not believe that this has been proved. Personally I have found no benefit from referring these cases to the dental surgeon.

(2) *Herpes recurrens facialis of adults*.—The lesions in this group are usually round the lips or nasal margins, and more rarely on the cheek. Attacks occur from intercurrent infections, most typically the common cold. They may, however, be associated with no obvious activating causes. I have been struck by the frequency with which young women complain that the herpes occurs on the eve of some important social function. It may be considered fanciful to suggest a psychological background in these instances. I feel, however, that this is at least possible.

In other patients climatic factors are important, such as a cold wind, or sudden exposure to strong sun, i.e. the first few days of a Swiss holiday.

(3) *Herpes recurrens genitalis*.—In males, penile lesions are rarely so recurrent that they come for treatment on this account. In my experience trauma, chemical irritants, and psychological reasons such as a guilty conscience may be presumed to be factors in producing the eruption.

In women, herpes recurrens occurs on the buttocks, and I have noticed occasionally a tendency for the eruption to be associated with the monthly period. In a few instances it disappeared with gynaecological treatment.

Before discussing treatment in general, one must emphasize that the disease is benign—the eruption usually lasts a week. On the face it is a cosmetic blemish which at the worst can leave a faint scar. Therefore no treatment is justified which is dangerous.

Treatment can be considered under two headings: (1) general; (2) local.

General treatment.—It is established beyond doubt that the virus has a high biotrophic index. Its activities are frequently exalted by intercurrent infection, the common cold being the classical example. Other infections are, of course, well known to be capable of producing the disease. Under these circumstances it would be logical to conclude that focal sepsis might be a factor. In my experience results of treatment from this aspect have been very rarely successful. It remains, however, true that if one can prevent the patients from having colds, one can frequently greatly diminish their attacks of herpes.

Vitamins have been in vogue for the treatment of this disease, as in many others. Vitamin C was reported to be specific in some American publications some years ago. I well remember a chronic case of twenty years' standing whom I had treated unsuccessfully, who claimed to be cured by taking betaxan. I have been unable to repeat this success by giving vitamin B or any other vitamin.

My experiences with drugs such as arsenic have been similarly unsuccessful.

Local treatment.—X-rays are by far the most effective treatment, though the proviso of no risk is specially applicable. Their action is presumably to increase the resistance of the epidermal cell to the virus, and they are more effective the more localized is the lesion to one area. I would welcome suggestions as to the most effective technique. In my experience treatment has to be minimal and is usually better filtered through one or even two millimetres of aluminium. Three doses of 100 R. should produce at least six months' immunity, or they are not worth repeating. If some very

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the periods and think a possible reason for this association may be the occurrence of subnormal temperature in the day before a period. Herpes of the lip often follows exposure to cold winds and chills.

Dr. F. Parkes Weber: The literature does not seem to be suggestive of any great frequency of herpes simplex recurrens, but if physicians who were mostly treating cases not dermatological, had collected statistics regarding patients who had casually mentioned that they also occasionally had an attack of recurrent simple herpes, one would be astonished at the relative frequency of the ailment in question, including, of course, recurrent herpes of the penis and prepuce in men, recurrent gluteal or femoral herpes in women, as well as recurrent herpes of the lips and other parts of the face.

Section for the Study of Disease in Children

President—A. G. MAITLAND-JONES, O.B.E., M.D.

[October 24, 1941]

DISCUSSION ON PYLORIC STENOSIS IN INFANCY

Dr. Donald Paterson : Congenital pyloric stenosis.—The problem of medical as opposed to surgical treatment of pyloric stenosis should be examined firstly from the results obtained by each method, and secondly from the availability of each method throughout the country. For instance, there are many places in country districts where surgery is not obtainable, and medical treatment would therefore naturally be tried first.

Results of surgical treatment.—I have before me such figures as those of C. F. Harris and E. Keynes (1937) where there was only one death in 50 consecutive infants, and David Levi (1941) who operated on 100 consecutive breast-fed infants, without a death. In 1939, out of 92 pyloric cases admitted to Great Ormond Street for operation, there were 6 deaths, a mortality of 6.5%. It is true the hospital is most modern, and each infant occupied a small wardlet of its own, but when one remembers the poor state in which some of the infants arrived, it is a remarkably low figure. In the two previous years the mortality was 10% and 9% respectively. From the above figures it seems clear that pyloric stenosis can be treated surgically with a very low mortality, even in hospital, and the infants can be restored to normal health in a period of two weeks or less.

In my private practice, out of a series of 60 surgically treated cases (50 males and 10 females) seen since 1925, there were two deaths only, and both of these children were admitted to hospital, where they contracted gastro-enteritis after the operation. This is a mortality of 3.5%.

Results of medical treatment.—Although medical treatment has been attempted for more than twenty years, there has been a much greater interest taken in it during the last few years. This is particularly true since the outbreak of the war, and the almost complete closure of most of the children's hospitals in our big cities. At first, medical treatment consisted in encouraging breast-feeding, but if the infant had been weaned, in giving a humanized dried milk, or other feed in which the curd was small. In addition, a daily stomach wash-out, with normal saline, was given, together with one drop of a 1:1,000 solution of atropine sulphate before each feed.

Eumydrin (atropine methyl nitrate) is a crystalline substance said to be $\frac{1}{50}$ as toxic as atropine, and much stronger relatively in its action. It was first used by Usener, in 1926, and Paul Drucker introduced it the following year in Copenhagen. In 1935 Elizabeth Svensgaard wrote a paper in the *Archives of Disease in Childhood* recommending its use, and since then various papers have been written on the successful use of this drug. In 1936 and 1941 MacKay, in 1938 Braithwaite, 1939 and 1941 Dobbs, and 1939 Vertue and Lightwood, have all described cases treated by eumydrin and the combined general mortality has been approximately 12% or 13%.

How it acts.—The pylorus is normally relaxed by the actions of the vagus and contracted by the sympathetic. The normal action of atropine is to inhibit vagal activity, thus atropine should constrict the pylorus by cutting out the vagal action. This action of atropine is seen in connexion with the splanchnics. These vessels are constricted by the sympathetic, and relaxed by the vagus, and are actually constricted when atropine is administered. Now, in pyloric stenosis when atropine is used, inhibition of the vagus and constriction of the pylorus does not take place; actually atropine causes relaxation of the pylorus, and it appears that atropine must act directly on the muscular cytoplasm itself, because this action is observable even when the vagi are severed. The degree of relaxation

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CONCLUSIONS

From the facts elicited, the following conclusions may well be drawn:

(1) Surgery must still remain the method of choice for the treatment of all but selected cases of congenital pyloric stenosis. This is particularly so in very young infants who are progressively losing weight.

(2) In those cases seen after the eighth week and where the weight has been maintained, or there has been a slight gain, and the nursing is good, a trial of eumydrin should always be made. This is more likely to be successful in female infants than in males.

(3) If eumydrin be used, it is best given as lamellæ, or in alcoholic solution in a concentrated form.

(4) Better facilities for the isolation of infants from ward infections must be provided if we are to equal the statistics of many of the continental schools, especially in Scandinavia.

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Dr. Richard Dobbs: The medical treatment of pyloric stenosis is not new; it was used because there was no alternative in the earliest case that has come to light. In 1646 Fabricus Hildanus (Kellett, 1933) cured an infant of a few weeks, who had vomiting and absolute constipation, by reducing the volume of feeds and exerting a policy of expectant patience. This gives the clue to the first principle upon which medical treatment is based. The disease is, we know, self limiting in nature and, we shrewdly suspect, functional in origin. As the child may die in the meantime if left to cure itself, medical treatment is designed to hasten the natural process. The third principle I will come to later.

TABLE I.—THE RESULTS OF MEDICAL AND SURGICAL TREATMENT. REPORTED SERIES.

| Treated medically | | | | | Treated surgically | | | | |
|-------------------|------------|--------------|--------|-----|--------------------|-----------|--------------------|--------|------|
| Date | Authors | Cases | Deaths | | Date | Authors | Cases | Deaths | |
| | | | No. | % | | | | No. | % |
| Before 1910 | Heubner | .. Total of | 5 | 7 | Before 1910 | | Mortality over 50% | | |
| | Bloch | 71 | | | | | | | |
| | Starck | | | | | | | | |
| | Bendix | | | | | | | | |
| 1910 | Hutchison— | | | | 1913 | Roland | .. 10 | 5 | 50 |
| | Private | .. 20 | 2 | 10 | | | | | |
| | Hospital | .. 64 | 50 | 78 | | | | | |
| 1914 | Holt .. | .. 28 | 14 | 50 | 1914 | Holt .. | .. 29 | 17 | 58 |
| 1922 | Haas .. | .. 39 | 1 | 2.8 | 1914 | Richter | .. 22 | 4 | 18.1 |
| 1923 | Ibrahim | .. 52 | 2 | 3.8 | 1918 | Strauss | .. 63 | 3 | 4.8 |
| 1928 | Monrad | .. 228 | 10 | 4.4 | 1937 | Jewesbury | 303 | 38 | 12.5 |
| | | | | | | and Page | | | |
| | | | | | | Private | .. 16 | 1 | 6.2 |
| | | | | | | Hospital | .. 287 | 37 | 12.9 |
| 1935 | Svensgaard | .. 61 | 2 | 3.3 | 1936 | Levi .. | .. 50 | 1 | 2 |
| 1940 | Wallgren | .. Not given | | 1% | 1937 | Donnovan | .. 243 | 1 | 0.4 |

* Quoted by Hutchison (1910).

is relative to the size of dose of atropine given; that is, larger doses of atropine cause greater relaxation.

Methods of administering eumydrin.—Svensgaard (1935) suggested giving from 1 to 3 c.c. or more of freshly prepared 1:10,000 solution of eumydrin a few minutes before each feed, and increasing the amount if necessary. Most of the papers published are based on this method of administration. The obvious objection to this is that the solution of eumydrin may pass into the already full stomach and lie unabsorbed or be vomited later. Arvid Wallgren (1940) suggested that a 0.6% alcoholic solution of eumydrin (eumydrin 0.3 + concentrate alcohol 50) containing approximately 0.1 mg. i.e. about $\frac{1}{10}$ gr. per drop, should be administered instead. The alcoholic drops should be placed on the child's tongue or gums where it is rapidly absorbed, and he found that three to five drops were required daily to relax the pylorus. By this method there is no doubt about the absorption of the eumydrin, and it cannot be lost by vomiting.

For the past year or more, it has been the practice at the Westminster Hospital to administer eumydrin in the form of lamellæ, each containing $\frac{1}{10}$ gr. These are given ten to fifteen minutes before the feed, and the lamella is placed on or under the tongue, or in the child's cheek, where it rapidly melts, and is absorbed. A very great advantage with this preparation is that it keeps indefinitely, and is most suitable for home use as the mother cannot make a mistake about the dose. This preparation can now be obtained as "Pylotropin". Cases have now been successfully treated by these lamellæ in Great Ormond Street Hospital. The eumydrin can also be administered in one or two drops of glycerine quite satisfactorily, but, of course, in any other vehicle than the lamellæ it does not keep longer than one week.

Results of cases seen privately.—In a series of 40 cases (26 males and 14 females) seen since 1925, and treated medically, one infant died, and this one case had been admitted to hospital, and there contracted gastro-enteritis—a mortality of 2.5%. The average age of this series was 8.2 weeks. Of these one received no medicinal treatment at all, being given gastric lavage only; 26 received atropine sulphate—one drop of a 1:1,000 solution before each feed; and 13 were given eumydrin. Prior to 1940, this was administered as $\frac{1}{10}$ gr. (0.1 mg.) of eumydrin in 10 minims of water, before each feed. After 1940 the same dose of eumydrin was given in two drops of glycerine, and later in 25% alcohol. Finally, lamellæ, described previously, were exclusively used.

Length of stay in hospital.—We must ask ourselves whether even if we had the most perfect hospitals and nursing we would wish to keep the infants there for many weeks undergoing treatment, and whether that is economically and socially a sound procedure. It can be taken that the average case of surgically treated congenital pyloric stenosis remains in hospital from ten to fourteen days. Attempts have often been made to send the infant home on the second or third day, particularly if breast-fed, but distances are often great, the mother inexperienced and relapses occur, or the wound may become infected. On the whole, this is not a satisfactory practice. Discharging the infant too soon is merely a confession that hospital surroundings are unsatisfactory and dangerous rather than the way to meet the problem. It ought to, and must, be possible to keep an infant safely in a hospital wardlet, free from infection, for a period of a fortnight. With eumydrin treatment, the length of stay in hospital is, as a rule, much longer than in the operated cases. With Svensgaard it was as long as seventy-seven days, whereas in the Guy's Hospital series (Vertue, 1939) the average stay in hospital was eighteen days. The ideal would be to treat these cases as out-patients, but that is not possible, except in the very mild ones, or where a first-class children's nurse can be continually in attendance.

Failures of eumydrin.—Of the last ten consecutive cases seen by me, all were put on eumydrin. Seven of these were rapidly successful, but in three cases the loss of weight and vomiting did not cease, and ultimately all three had to be operated on. It was noticeable that all of the three infants were below the age of 8 weeks, and showed symptoms during the first two weeks with progressive loss of weight and dehydration.

It would be giving the medical profession, particularly the general practitioner, a completely wrong idea if it was thought that all cases of pyloric stenosis could be put on eumydrin, and provided the dose was properly regulated the case would be cured. Too much stress has been placed on the action of the drug, and too little stress on the careful nursing and feeding and general management. Certainly the nursing care is quite as important as the drug.

CONCLUSIONS

From the facts elicited, the following conclusions may well be drawn:

(1) Surgery must still remain the method of choice for the treatment of all but selected cases of congenital pyloric stenosis. This is particularly so in very young infants who are progressively losing weight.

(2) In those cases seen after the eighth week and where the weight has been maintained, or there has been a slight gain, and the nursing is good, a trial of eumydrin should always be made. This is more likely to be successful in female infants than in males.

(3) If eumydrin be used, it is best given as lamellæ, or in alcoholic solution in a concentrated form.

(4) Better facilities for the isolation of infants from ward infections must be provided if we are to equal the statistics of many of the continental schools, especially in Scandinavia.

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Dr. Richard Dobbs: The medical treatment of pyloric stenosis is not new; it was used because there was no alternative in the earliest case that has come to light. In 1646 Fabricus Hildanus (Kellett, 1933) cured an infant of a few weeks, who had vomiting and absolute constipation, by reducing the volume of feeds and exerting a policy of expectant patience. This gives the clue to the first principle upon which medical treatment is based. The disease is, we know, self limiting in nature and, we shrewdly suspect, functional in origin. As the child may die in the meantime if left to cure itself, medical treatment is designed to hasten the natural process. The third principle I will come to later.

TABLE I.—THE RESULTS OF MEDICAL AND SURGICAL TREATMENT. REPORTED SERIES.

| Treated medically | | | | | Treated surgically | | | | |
|-------------------|----------------------------------------------|----------------|--------|-----|--------------------|--------------------|--------------------|--------|------|
| Date | Authors | Cases | Deaths | | Date | Authors | Cases | Deaths | |
| | | | No. | % | | | | No. | % |
| Before 1910 | Heubner } Bloch } Starck } Bendix } | .. Total of 71 | 5 | 7 | Before 1910 | | Mortality over 50% | | |
| 1910 | Hutchison—Private | .. 20 | 2 | 10 | 1913 | Roland | .. 10 | 5 | 50 |
| | Hospital | .. 64 | 50 | 78 | | | | | |
| 1914 | Holt .. | .. 28 | 14 | 50 | 1914 | Holt .. | .. 29 | 17 | 58 |
| 1922 | Haas .. | .. 39 | 1 | 2.8 | 1914 | Richter .. | .. 22 | 4 | 18.1 |
| 1923 | Ibrahim .. | .. 52 | 2 | 3.8 | 1918 | Strauss .. | .. 63 | 3 | 4.8 |
| 1928 | Monrad .. | .. 228 | 10 | 4.4 | 1937 | Jewesbury and Page | 303 | 38 | 12.5 |
| | | | | | | Private | .. 16 | 1 | 6.2 |
| | | | | | | Hospital | .. 287 | 37 | 12.9 |
| 1935 | Svensgaard .. | .. 61 | 2 | 3.3 | 1936 | Levi .. | .. 50 | 1 | 2 |
| 1940 | Wallgren .. | .. Not given | | 1% | 1937 | Donnovan .. | .. 243 | 1 | 0.4 |

* Quoted by Hutchison (1910).

Early in this century medical treatment produced results which, in some hands, were not so very different from those of the present day. Table I shows some of the collected results of both methods of treatment from the beginning of the century to the present time, and illustrates that it was not until after 1910 that surgical results were in any way comparable. About this time, while Rammstedt was attempting a submucous pyloroplasty, an operation devised a few years earlier by Fredet and Dufour, and independently by Weber, the stitches tore away from the muscle. The child looked ill and Rammstedt closed the abdomen. Though convalescence was rather stormy, the child did not die. At the next opportunity Rammstedt purposely only took the operation to the stage of muscle incision. This child fared even better, and made an uninterrupted recovery. How successful Rammstedt's operation is can be seen in the table. It is so successful because it does precisely what is needed in the shortest possible time. It follows the second principle upon which medical treatment is also based; it hastens the natural tendency of the disease to cure itself. The spasm is relieved, in this case suddenly, and the muscle has perforce to relax. The tumour is not removed by the surgeon; finding, so to speak, nothing to keep it in training, and feeling, perhaps, rather redundant, it gradually fades away. It does so, as Wollstein (1922) so beautifully showed from post-mortems on cases dying from two days to several weeks after operation, during the course of three to four weeks, after which the circular muscle joins again by fibrous union, the eventual result being anatomically almost perfect and indistinguishable from normal. And that brings me to the third principle upon which treatment is based. Once the spasm has been overcome, either naturally, by surgery, or medically, the functional result is perfect. Medical treatment would be useless, however immediately successful, if relapse occurred in later years. This is not the case. The two methods of treatment are essentially similar. Relieve the spasm, and the stomach and pylorus return to normal. The results of both methods are comparable, and can scarcely be improved upon. There are two other sides to the picture. The incidence of pyloric stenosis is, I fancy, fairly evenly distributed throughout the world, and is not confined, for instance, to the urban population. Though the operation is comparatively easy, it needs considerable skill and experience to achieve consistently good results, and there is a mortality directly due to surgical interference. Even in published series this often amounts to 5%, and is probably frequently higher. Secondly, there is in hospitals in this country the toll levied by gastro-enteritis.

TABLE II.—COLLECTED CASES (RAMMSTEDT).

| Date | Treated medically | | | Treated surgically | | |
|---------|-------------------|--------|------|--------------------|--------|------|
| | Cases | Deaths | % | Cases | Deaths | % |
| 1918-28 | 1,345 | 219 | 16 | 497 | 112 | 22.5 |
| 1929-33 | 1,675 | 150 | 9 | 757 | 110 | 14.4 |
| Total | 3,020 | 369 | 12.2 | 1,254 | 222 | 17.7 |

The dangers of hospital treatment in this country do not apply to such countries as Scandinavia, where gastro-enteritis hardly ever occurs even during admissions of eleven and twelve weeks. The new factor introduced by the use of eumydrin is that out-patient treatment can be carried out in a large number of cases and after an initial phase.

Table III gives relevant details of 46 cases of pyloric stenosis treated with eumydrin. The group has all the characteristic features of age, sex distribution and interval of good health. Peristalsis was visible in all, and the pylorus was felt in all but one, who later recovered after Rammstedt's operation. Eumydrin was tried in each case for ten days. If, at that time, vomiting was undiminished operation was decided on. If, on the other hand, vomiting was less, medical treatment was continued. The mother was instructed in the routine of treatment for one or two days, and as soon as it was thought safe the child went home and was treated as an out-patient. This entirely arbitrary plan was carried out only to avoid prolonging medical treatment indefinitely, in the hope that improvement would eventually take place. It did, however, lead to definite results. A premium was set on speedy cessation of vomiting and the cases fell consequently into three groups; the first, a group of seventeen cases, who responded to eumydrin in the course of a few days and went home within ten days; a second group who responded more slowly, yet sufficiently by the end of ten days to avoid operation; and a third group who failed to respond in the required time and were handed over for the relief of their spasm to the surgeons.

TABLE III.—DETAILS OF 31 CASES SUCCESSFULLY TREATED BY EUMYDRIN, ARRANGED ACCORDING TO LENGTH OF STAY IN HOSPITAL.

| | Case | Sex | Place in family | Days in hosp. | Age at onset Wks. | Age on admis- sion Wks. | Weight change since birth lb. oz. | Maximum dose of eumydrin each feed c.c. | Weeks given |
|-------------------------------------|------|-----|-----------------------|---------------------|----------------------------|-------------------------------------|-----------------------------------------------|-----------------------------------------------------|----------------|
| Group A in hosp. 10 days or less | 24 | M. | 1 | 0 | 4 | 12 | 1 0 | 2.5 | 6 |
| | 26 | F. | 1 | 0 | 8 | 10 | 2 9 | 3 | 8 |
| | 39 | M. | 2 | 0 | 6 | 9 | 1 2 | 4 | 6 |
| | 33 | M. | 1 | 5 | 7 | 8 | 1 0 | 3 | 9 |
| | 34 | M. | 1 | 5 | 6 | 7 | 0 9 | 3 | 10 |
| | 40 | M. | 1 | 5 | 2 | 3 | 0 5 | 3 | 5 |
| | 12 | M. | 1 | 6 | 6 | 8 | 1 2 | 5 | 7 |
| | 15 | M. | 2 | 6 | 4 | 7 | 0 5 | 4 | 11 |
| | 4 | F. | 1 | 7 | 2 | 4 | 0 6 | 2 | 9 |
| | 41 | M. | 3 | 7 | 5 | 6 | 1 4 | 2 | 7 |
| | 28 | M. | 1 | 8 | 1 | 1½ | 0 4 | 3 | 6 |
| | 42 | M. | 1 | 8 | 6 | 10 | 0 9 | a | 8 |
| | 45 | M. | 1 | 8 | 3 | 4 | 0 3 | a | 5 |
| | 5 | M. | 2 | 9 | 6 | 8 | 1 4 | 4 | 9 |
| | 18 | M. | 1 | 9 | 4 | 5 | 0 3 | 5 | 15 |
| | 31 | M. | 1 | 10 | 5 | 6 | 1 2 | 3 | 4 |
| | 46 | M. | 2 | 10 | 5 | 6 | 0 4 | a | 6 |
| Average in 17 cases | | | | 6 | 4.7 | 6.7 | 0 17 | 3 | 7.6 |
| Group B in hosp. 10 to 20 days | 9 | M. | 2 | 13 | 2½ | 4½ | 0 8 | 4 | 8 |
| | 11 | F. | 1 | 13 | 3 | 6 | 0 5 | 6 | 10 |
| | 2 | M. | 2 | 14 | 2½ | 5 | 0 5 | 2.5 | 10 |
| | 13 | M. | 1 | 14 | 5 | 7½ | — | 3 | 9 |
| | 32 | F. | 3 | 14 | 2½ | 5 | 0 2 | 5 | 8 |
| | 10 | M. | 1 | 15 | 2½ | 4½ | 3 0 | 4 | 11 |
| | 16 | F. | 1 | 15 | 2 | 3 | 1 14 | 6 | 11 |
| | 17 | M. | 3 | 15 | 2½ | 3 | 0 2 | 4 | 12 |
| | 18 | M. | 1 | 18 | 3 | 4 | 1 3 | 5 | 11 |
| | 19 | M. | 1 | 19 | 3½ | 4 | — | 4.5 | 14 |
| Average in 10 cases | | | | 15 | 2½ | 4½ | 0 7½ | 5 | 10½ |
| Group C in hosp. over 30 days | 21 | M. | 2 | 30 | 3 | 7 | 1 1 | — | 12 |
| | 29 | M. | 1 | 32 | 8 | 9 | 2 13 | — | 12 |
| | 1 | F. | 1 | 34 | 1 | 3 | 1 0 | — | 13 |
| | 6 | F. | 1 | 70 | 3 | 6 | 1 2 | — | 9 |

a = Alcoholic eumydrin.

The successfully treated medical cases are divided in Table III into a group of seventeen sent home within ten days, and a second group of ten, sent home within twenty days. There is a third group of four cases who were all in for thirty days or longer. All but one were kept in for reasons other than vomiting, and it will be noticed that there are no intermediate cases. The next table compares the three groups, i.e. the two responding to treatment, and the group operated on, with each other in relation to age. The age at the onset of vomiting and the weight gained or lost before presenting for treatment had clearly a bearing on the degree to which the spasm responded to the drug. The first three cases in Table III had an average age of six weeks when vomiting started, and had gained 28oz. since birth when they were first seen. Of the twelve cases eventually operated on (Table V) none was over 4 weeks old at the onset of vomiting (only three were in fact that age), and all were boys, though seven of the whole series were girls.

A very important question is, does failure to operate immediately increase the operative risk. Or how much is the operative risk increased, since there is no gainsaying that ten days have been wasted and vomiting has gone on. There was loss of weight in fact in

TABLE IV.—CASE HISTORIES COMPARED.

| | <i>Av. age at onset Weeks</i> | <i>Av. age on admission Weeks</i> | <i>Av. weight loss or gain since birth Oz.</i> |
|-------------------------------------------|---------------------------------------|-------------------------------------------|------------------------------------------------------------|
| Group A (Disch. within 10 days). 17 cases | 4.7 | 6.7 | +17 |
| Group B (Disch. within 20 days). 10 cases | 2½ | 4½ | -7½ |
| Group D (treated by operation). 12 cases | 2½ | 4½ | +½ |

only three, with some gain in the rest, and convalescence after operation was rapid. Although the heavy mortality, four deaths in the cases operated on, might be attributed to the pre-operative medical treatment, this was not so. One died of purulent peritonitis thirty-six hours after operation and a perforation of the duodenal mucosa was found at post-mortem; another died soon after operation of massive pulmonary collapse, and a large milk curd was found impacted in the right main bronchus. These must be counted as the hazards of surgical treatment. Two died of gastro-enteritis. No. 37 was doing well after operation until No. 38 was admitted. Unfortunately owing to war-time ward space and nursing difficulties they had to be nursed in the same open ward. In No. 38 the child was ill with gastro-enteritis when admitted and operation was delayed for only a few days whilst measures to combat dehydration and the generally poor condition were undertaken.

TABLE V.—DETAILS OF 12 CASES (ALL MALES) OPERATED UPON AFTER EUMYDRIN HAD FAILED.

| <i>Case</i> | <i>Place in family</i> | <i>Age at onset</i> | <i>Length of history</i> | <i>Birth weight</i> | <i>Weight on admission</i> | <i>Weight at operation</i> | <i>Max. dose of Eumydrin at each feed</i> | <i>Length of treatment</i> | <i>Operation</i> | <i>Result</i> |
|-------------|----------------------------|---------------------|------------------------------|---------------------|--------------------------------|--------------------------------|---------------------------------------------------|--------------------------------|----------------------------|---------------------------|
| | | <i>Wks.</i> | <i>Days</i> | <i>lb. oz.</i> | <i>lb. oz.</i> | <i>lb. oz.</i> | <i>c.c.</i> | <i>Days</i> | | |
| 7 | 1 | 3 | 7 | 7 4 | 7 10 | 7 12 | 6 | 27 | Relapse | Disch. 12 days |
| 8 | 2 | 2 | 7 | 8 0 | 7 6 | 7 6 | 5 | 10 | Contd. vom. | " 7 weeks |
| 20 | 1 | 1 | 1½ | 8 12 | 7 6 | 6 12 | 7 | 8 | Contd. vom. loss of wt. | " 8 days |
| 22 | 1 | 4 | 14 | 6 8 | 6 11 | 7 1 | 4 | 8 | Contd. vom. | " 3 weeks |
| 23 | 1 | 3½ | 14 | 8 0 | 8 3 | 8 6 | 5 | 8 | Contd. vom. | Death (Peritonitis) |
| 25 | 1 | 3 | 14 | 7 0 | 8 5 | 8 6 | 5 | 10 | Contd. vom. | Disch. 7 days |
| 27 | 1 | 1½ | 2½ | 6 6 | 5 15 | 8 3 | 6 | 105 | Relapse | Disch. recov. 12 days |
| 30 | 1 | 4 | — | 8 0 | 8 12 | 8 2 | 4 | 6 | Contd. vom. loss of wt. | Disch. 2 weeks |
| 35 | 1 | ½ | 17 | 7 5 | 6 11 | 6 15 | 7 | 13 | Contd. vom. | Death (Pulm. collapse) |
| 36 | 2 | 3 | 17 | 6 0 | 6 10 | 7 3 | 5 | 33 | Relapse after disch. | Disch. 7 days |
| 37 | 1 | 1½ | 24 | 6 13 | 6 15½ | 6 8 | 4 | 12 | Contd. vom. | Death (Gastro-ent). |
| 38 | 1 | 4 | 21 | 7 12 | 7 4 | 7 5 | 5 | 5 | Contd. vom. | Death (Gastro-ent). |

The dangers of open ward treatment are well illustrated in this series. There were four fatal cases of gastro-enteritis, these two and two medically treated, which did not appear in Table III. Until the war all the cases were treated either in separate cubicles or in separate rooms at the Queen's Hospital, where they stayed with their mothers. After the war began this became impossible, and at the same time nursing facilities became less adequate. The last fifteen cases were treated after September 1939, and of these four died of gastro-enteritis and two others had milder non-fatal attacks. The only case in the first 31 was admitted with the condition, and was successfully treated medically.

With regard to the use of eumydrin by out-patients: Firstly, the aqueous solution usually prescribed as a 1:10,000 solution, rapidly deteriorates, probably losing over half its activity during the first week. The alcoholic solution is, on the other hand, not only considerably more effective but is also comparatively stable. It may actually increase in effectiveness—and therefore also in toxicity—with evaporation of alcohol, so that its use in out-patients may be less safe. The lamellæ, of which I have no experience, should overcome this difficulty as well as that of dosage.

Secondly, it is clear that atropine and its derivatives not only relieve the spasm of the pylorus, but hastens recovery. Left to itself the vomiting gradually subsides during the course of about ten to twelve weeks. Though at first the drug was given for about this time before the dose was gradually decreased, in the later cases of the series eumydrin was stopped earlier. As a result the vomiting recurred when eumydrin was withheld in five cases, after ten days, three weeks, three and a half weeks, four weeks and five weeks respectively. There was no return of vomiting when the drug was stopped, after four weeks in one case, five weeks in two cases, and six weeks in four cases. This places the lower limit of time at about four to five weeks. Information regarding the upper limit is less definite. Vomiting did not return in any case in which eumydrin was withheld after the sixth week, but the cases which would be expected to take longer were operated on. In one case vomiting was never properly controlled, and though satisfactory weight increase took place at home for about three and a half months the child was eventually readmitted for operation.

SUMMARY

Medical treatment with eumydrin can be used with considerable success as a method of out-patient treatment. A considerable proportion of cases can be sent home within ten days to attend weekly, and probably for those, in whom dehydration and wasting is absent, admission will in the future not be necessary. These are cases in whom symptoms start late, probably after the fourth week of life, and possibly females respond more easily than males. Of the rest about half will be home within three weeks, and the other half, more resistant, will either need prolonged medical treatment or operation. Whether operative or medical measures are undertaken in any given case depends amongst other things on the surgical skill and the hospital and nursing facilities available, as well as upon the age, sex and general condition of the patient. The question before the meeting is not so much whether pyloric stenosis should be treated medically or surgically, but in which cases, and under what circumstances should medical treatment be employed, and when should it give place to surgical intervention.

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Sir Lancelot Barrington-Ward: Up to 1917, when Rammstedt's operation was first performed at the Hospital for Sick Children, Great Ormond Street, the mortality of the disease was 80%. From 1917 to 1937, 1,061 cases have been admitted to the hospital. Three were moribund on admission, 68 were treated medically with a mortality of 77%, and the remainder were treated surgically with a mortality of 20.1%, and with every year showing a decrease. I have been hampered by the evacuation of the hospital, and the disturbance of the war, and I regret I cannot bring the general figures more up

to date, but the figures already obtained, representing as they do the work of the whole surgical division of the hospital, and therefore a better test than the statistics of one individual, are sufficiently striking. In the last year reviewed, 1937, the mortality was 8.7%, and Dr. Paterson has now stated that in 1939 the mortality was only 6.5%. How was this reduction of mortality from 8.0% to 6.5% achieved?

In the first place, Rammstedt's operation revolutionized the treatment. After many previous attempts at more complicated procedures, the discovery that simple division of the constricting muscular fibres relieved the pyloric obstruction, opened a new chapter. At first the mortality was considerable, but certain principles had yet to be worked out. Ladd and Lanman of the Boston Children's Hospital were among the first to emphasize that under no circumstances should the operation be looked upon as an urgent emergency, but that great care and sufficient time must be taken in the preparation for operation. Gastric lavage to reduce the gastritis, subcutaneous or intravenous administration of 5% glucose solution to combat dehydration and alkalosis, in the worst cases blood transfusion, became a routine in every children's hospital and the mortality generally declined.

Then came a further advance. It was noted that the most common cause of death was gastro-enteritis, and that this complication was avoided if infants were nursed in separate cubicles with special nurses, to eliminate cross-infection. This explains a fact of which all surgeons are aware that private cases do infinitely better than hospital. At the Children's Nursing Home in London, up to the outbreak of war, I am informed that 186 consecutive cases had been operated upon without fatality. I have operated upon 51 private cases, and of these all recovered save one who, despite steady increase of weight, developed bronchitis at home some weeks after operation and died. It may be argued that the better outlook for private cases is due to earlier diagnosis and more resistant stock. That this is not so, is shown by the fact that infants in hospital, nursed under the same conditions as obtain in private practice, are equally free from those fatal complications associated with large wards and exposure to cross-infection. In the old hospital in Great Ormond Street, with old-fashioned wards, filled with children of all ages and complaints, this cross-infection was rife: in the new hospital with separate cubicles and individual nursing, conditions approaching private practice were obtained and the results improved immediately. Further proof of the influence of segregation and avoidance of infections is provided by our experience in the first year of war. As a precaution against air raids the few children in hospital were removed from their cubicles at night to corridors and such places of greater safety. Segregation and isolation thus broke down. Dr. Levick informs me that of 97 cases treated under these conditions between October 1939 and September 1940, 26 were complicated by gastro-enteritis, and of these 11 died. The total mortality from complications was 13.4%.

In the surgical technique of the operation, probably little improvement can be expected, although I know the danger of making such a statement. Perforation of the duodenum, peritonitis, reactionary hæmorrhage, no longer occur, and the only complication that occasionally follows is the persistence of symptoms due to incomplete division of the constricting muscle.

There is little to choose between local or general anaesthesia, but I have the impression that the infant looks better and does better after general than after local anaesthesia. Under gas and oxygen the operation is shorter, the manipulations are easier and the minimum of toxic material is introduced into the circulation.

It has always been the custom for infants suspected to be suffering from congenital pyloric stenosis to be admitted under the care of the physicians, and the question whether surgical or medical treatment is to be adopted must remain in their hands. If, however, under suitable conditions it is possible to cure 100 consecutive cases without a fatality, the claims of surgery are very strong. I have no experience of eumydrin medication save in those instances where the drug has failed and a debilitated infant is brought to operation as a last resort. No doubt it is good for mild cases and for those late cases in whom symptoms do not begin until the third month which do less well with operation and which tend to cure themselves if they can be tided over a few weeks. In private practice, where time and nursing can be more generously provided, antispasmodic treatment may have a more useful field. The disadvantages lie in the

uncertainty of the method, and the difficulty of deciding how far the general condition of the patient can be allowed to deteriorate before invoking the aid of surgery. Medical treatment takes longer. Svensgaard, according to her original paper, kept her patients in hospital for months. After operation, particularly in the case of breast-fed infants, the patient is discharged home within a few days, and the illness is at an end.

Mr. David Levi: The Rammstedt operation for pyloric stenosis has in my hands been modified in certain respects:

- (1) There is no prolonged pre-operative preparation of the child. Gastric lavage half an hour before the contemplated time of operation is insisted upon.
- (2) No pre-operative medication whatsoever is ordered.
- (3) The theatre is heated to as near 80° F. as possible.
- (4) The operation is performed under local anæsthesia induced by 10 c.c. or less of ½% ethocain solution.
- (5) Nothing is allowed to leave the abdomen apart from the hypertrophic pylorus.
- (6) Feeding by the mouth is commenced three to four hours from the time of operation.

As a result of this procedure, certain facts emerge.

I have recently published (1941)¹ an account of one hundred consecutive breast-fed infants treated without a death.

In the charts of cases 2, 3 and 4 (selected at random), one admitted on Monday, the next on Wednesday, and the third on Friday of the same week in November 1938, there was an immediate improvement and rapid gain in weight. Case 2 gained 11 oz. in one week; case 3 gained 16 oz. in one week; case 4 gained 14 oz. in one week.

It is logical to deduce from that:

- (1) As a result of ten-minute interference, during which the patient never loses consciousness, and is very slightly shocked, he is "cured"!
- (2) There is no operative mortality.

Table II and Table III (*Brit. M. J.*, 1941 (i), 964) show the records of 46 children who were bottle-fed; there are five who died, all from gastro-enteritis.

Chart IV (*Brit. M. J.*, 1941 (i), 963) gives a typical chart of a fatal case. It shows the immediate response to operation. The weight going from 6 lb. 8 oz. at operation to 6 lb. 14 oz. on sixth day. But on the fifth day following operation, the stools are frequent and relaxed; then there is a drop in weight and the rapid death of the child on the eleventh day.

Is this death to be attributed to surgery, or to the fact that pediatricians are unable to feed normal babies artificially in hospitals over a period without killing some of them?

The problem which faces us is how to feed normal infants in institutions, artificially, without killing them from gastro-enteritis, not how to cure pyloric stenosis, because this can be done with relatively little shock by an operation which must be classed among the most successful in the whole field of surgery.

The factors in the prophylaxis of gastro-enteritis fall under three heads:

(1) *The feeding. All babies should be breast-fed.*—The evidence on which this statement is based is:

(a) The tables just shown demonstrate that breast feeding is superior to bottle feeding in children suffering from pyloric stenosis.

(b) There is an increased tendency to bottle-feed infants to-day.

(c) Young infants should not be artificially fed unless there is some definite medical contra-indication which would prohibit the mother suckling her infant. The mother's milk is the best food for all small infants and we have no entirely satisfactory substitute for it at the present time.

I would recommend that we as a body should agitate that this should be taught in all medical schools and that it should be brought to the notice of the obstetricians who are perhaps the worst offenders in violating this axiomatic statement.

¹ Levi (1941), *Brit. M. J.* (i), 964, Table I.

(2) *The building*.—We at the Infants Hospital have been fortunate in having designed the new hospital, and we have been working in it for a number of years. The ward system is unsuitable for infants, and babies have rooms to themselves with wash-basins in each room, sterilizing facilities are readily available for soiled dressings, &c. I have no time to enter into architectural details. The last six years have demonstrated that a good building will reduce the incidence of gastro-enteritis among its inmates, but it will *not* entirely eliminate it.

(3) *The nursing*.—The evidence is as follows: (a) I visited a hospital recently to see an infant. I was taken to the cot by a Red Cross nurse. She removed the faecal-stained napkin and rolled it into a ball and placed it under the child's pillow.

(b) At my own Sector hospital, I am unable to admit infants under 12 months old, because so many die from gastro-enteritis.

(c) I examined nurses for the Final State Examination recently. I asked many of these women how they would change and feed a normal baby. The answers I got would astound you. Here is one: "I would take the baby to the bathroom, change it, and feed it, and take it back to the ward."

"Do you always feed babies in the lavatory?"

"Oh yes, we always do at our hospital. You see it is so convenient. The bathroom is next door to the kitchen."

"Do you wash your hands during this procedure?"

"Oh yes, of course, when I put the baby in its cot."

(d) Gastro-enteritis is prevalent throughout the country.

(e) Even in our friends' homes if you examine the cook's lavatory, you hardly ever find a wash-basin in it. If she does wash her hands she does so at the kitchen sink where our food is prepared.

There appears to be a very regrettable lack of education in hygiene among our adolescent women. And these are the women who become our probationer nurses, and our young mothers. If we could teach them the elements of hygiene at school, our standards of nursing would rise automatically.

I therefore have two further recommendations to place before you:

(1) That this Section officially approach the Minister of Education calling upon him to institute classes in hygiene and baby care in all girls' schools throughout the country.

(2) That a committee of pediatricians be appointed by this Section to supervise and/or produce a film or films on how to feed a baby, and how to avoid giving it diarrhoea, such a film to be an authoritative document, and that these films be distributed by the Ministry of Information to Welfare Centres, Townswomen's Guilds and Women's Societies all over England.

A committee appointed by this Section would be more representative than if the task were tackled by one particular hospital.

We have therefore a threefold task before us:

(1) To emphasize the significance and importance of breast-feeding for young infants.

(2) To endeavour to raise the standards of hygiene among adolescent women.

(3) To guide and advise architects in the planning of houses and hospitals in post-war England.

F/Lt. C. Hardwick said this was an important discussion. Besides Mr. Levi's proposals, which should be seconded and put to the meeting, the value of eumydrin had to be decided.

As had been said it was not a new drug, it had been in use for over five years; in this respect one could hardly imagine a discussion as to the value of sulphapyridine five years after its introduction. The figures for surgery which had been given proved conclusively that Rammstedt's operation was by far the better method of therapy. It could be said dogmatically that eumydrin had no place in the treatment of pyloric stenosis and should be abandoned. Mention had been made of the value of eumydrin in the wilds, when surgery was not available; but in days when it was possible to fly the Atlantic in nine hours there were no wilds in Great Britain. What was a mild case of pyloric stenosis?

Mr. W. Etherington-Wilson asked if anyone had seen spinal analgesia used for the operation. Nobody having had experience he thought it worth while bringing the subject before the meeting.

Seven years ago, a bad risk aged 17 days and weighing under 4 lb. was successfully given a spinal, using his own technique of timed vertical ascent, with percaine. Eleven months later a similar procedure was carried out on the brother of this child, a better risk. These successes were very encouraging and now the number in the small series has reached thirteen, all cases of pyloric stenosis. The original estimation of the dosage and timing, derived from glass tube experiments, for the first baby proved correct and has been used throughout, small allowances being made for the length of the infant's back. Percaine 1-2,500, sp. gr. 1.003 plus seems ideal. Dose 2 to 2.5 c.c. Total time in sitting position fifteen to twenty seconds. As soon as the injection into the fourth lumbar space through a No. 18 hypodermic needle is begun, the seconds-hand clock is started. The 2 c.c. is injected within 5 seconds and this time is included in the total of say 18 seconds used in that particular case. No premedication is given and local analgesia is unnecessary for the lumbar puncture. Lumbar puncture is easy in babies, but occasionally the fluid has to be induced to drip by aspiration. The child is held firmly on the operating table on sorbo blocks, and when the solution has been introduced it is held sitting bolt upright, if not already so, till the seconds have elapsed. Place the child on his back and hold up by the feet in Trendelenburg, till the table has been sloped 10°; this slope is maintained during the operation and journey back and for some twelve hours afterwards.

Analgesia is obtained within ten minutes to the 6th dorsal segment, the child ceases moving, becomes lazy and usually goes to sleep on the warm electric blanket on the table.

It is an advantage deliberately to spin out the operation time, so that forty minutes should have passed since the clock was started, before the Sister carefully carries the babe, sloped in her arms back to a warm sloped cot.

Mr. Etherington-Wilson went on to say that the method was well worthy of consideration, but should only be carried out by an understanding, expert spinal anaesthetist. Such grave operation risks were no subjects for blind experimentation.

The advantages claimed were, no hurry, gentle manipulations, ease of deliberate operating and possibly less trouble with the wound later than if a local infiltration was used. Some cases showed signs of fall in blood-pressure by pallor, but this was not measured and no stimulants were used in these cases. Possibly the well-being of the cases on the table was explained by some lack of development of the vasomotor powers at such early ages.

Dr. Pearse Williams said that he himself was biased towards operation, having competent surgical assistance always at hand. He had not been impressed by eumydrin in the few cases in which he had used it. He and his surgical colleague co-operated fully in diagnosis and aftercare. He drew attention to a further sign, the "pyloric nose" demonstrated to him by Dr. Reginald Miller, in this condition the nose appeared peculiarly prominent and was in fact an outstanding feature. Gastro-enteritis was not due in all cases to induced infection. Gastritis would occur if the feeding after operation was stepped up too rapidly and was probably due to previous irritation of the gastric mucosa by stale food contents. Separate cubicles and careful attention to all nursing details had improved their results at the Willesden General Hospital and he hoped the Section would play its part in future hospital planning where wards for children and infants were concerned. He agreed with the President in denying the existence of pylorospasm as an entity.

Dr. C. Elaine Field: I regret that two members of the Section have cast doubt on the existence of pylorospasm as distinct from pyloric stenosis. Recently, under my care, I had a male infant aged 4 weeks with all the characteristic symptoms of pyloric stenosis including marked peristalsis and projectile vomiting, but no tumour could be felt. At operation there was no enlargement of the pylorus, nevertheless a Rammstedt's incision was performed. The child continued to vomit after the operation, but ceased to do so immediately eumydrin was given.

I feel eumydrin has its uses in doubtful cases where no tumour can be felt or where an experienced surgeon is unobtainable, otherwise I favour surgical treatment.

Dr. Charles Pinckney : Pyloric stenosis, if the nutrition of the child can be maintained, will disappear at the age of 6 months. The essential aim in treatment, therefore, is to maintain an adequate nutrition till this age, when the dysfunction will tend to correct itself.

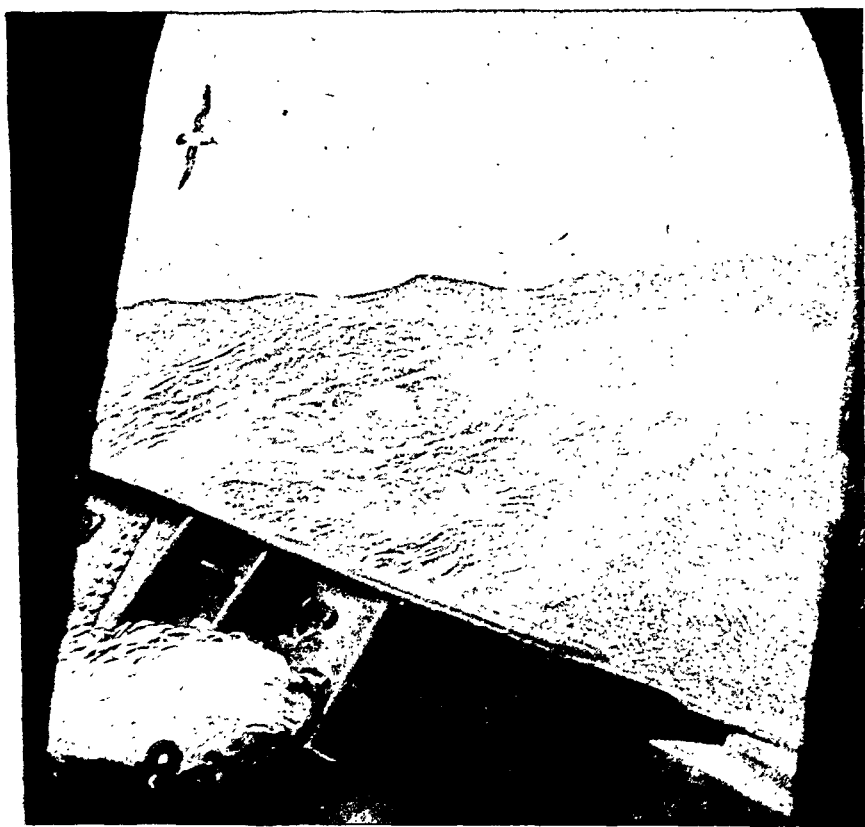
The question of which treatment is the most suitable, medical or surgical, must depend primarily, therefore, on the state of nutrition of each child when first seen.

If the state of nutrition is good and the child is above its birthweight, longer time can be spent in overcoming the dysfunction; so that it is in these cases we have the most favourable opportunity for medical treatment with eumydrin.

If malnutrition is already present, and especially if the child is below its birthweight, the dysfunction must be overcome in the shortest possible time, so as to restore the child to an adequate nutrition once again, and in these cases the Rammstedt operation offers the shortest time in treatment.

The prolonged medical treatment in hospital of cases on eumydrin is often unnecessary, and the last three cases seen, which were in good nutrition without dehydration, have been treated with eumydrin as out-patients from the beginning.

Occasional vomiting—even daily—may continue for some time with eumydrin treatment, but if nutrition is maintained and weight increases, the vomiting will gradually cease; but the eumydrin should be continued for at least a fortnight after the cessation of vomiting.



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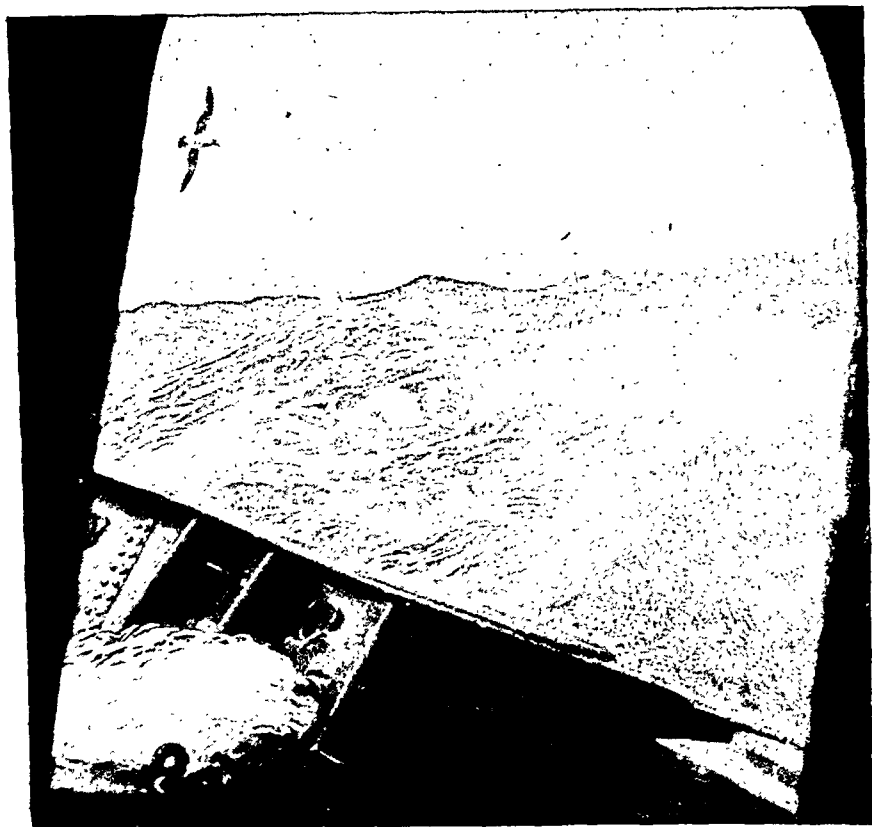
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FROM THE DEPTHS OF THE OCEANS HEALTH COMES TO THE WORLD

DEEP IN THESE SEAS swims the halibut. And within the halibut lies the source of one of the most potent aids to health ever discovered. For the doctors and scientists who search ceaselessly for whatever can benefit mankind discovered that halibut liver oil is one of the most richly concen-

trated sources of the vitamins which protect us from the onslaught of infection and disease. The

Crookes Laboratories are proud to be associated with the work of these men — proud to supply them with the tools to fight disease and to help ordinary people to live happier lives.



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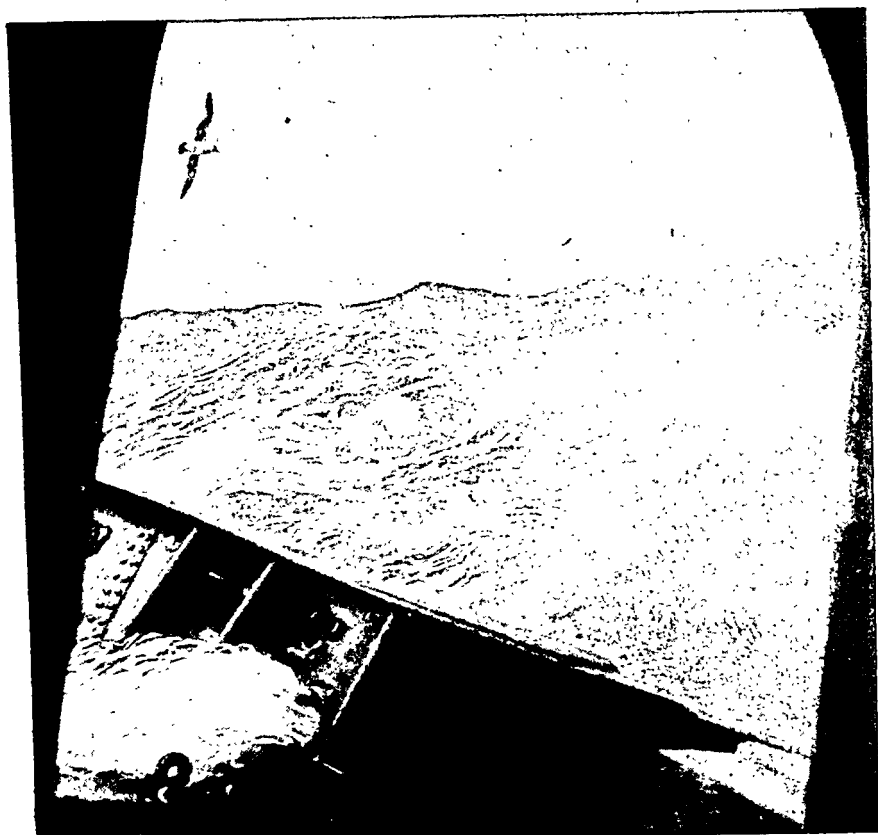
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FOR NERVOUS AND MENTAL DISORDERS

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FOR THE UPPER AND MIDDLE CLASSES ONLY. PRESIDENT—THE MOST HON. THE MARQUESS OF EXETER, K.G., C.M.G., A.D.C.
MEDICAL SUPERINTENDENT: THOMAS TENNENT, M.D., F.R.C.P., D.P.H., D.P.M.

THIS Registered Hospital is situated in 130 acres of park and pleasure grounds. Voluntary patients, who are suffering from incipient mental disorders or who wish to prevent recurrent attacks of mental trouble; temporary patients, and certified patients of both sexes are received for treatment. Careful clinical, biochemical, bacteriological, and pathological examinations. Private rooms with special nurses, male or female, in the Hospital or in one of the numerous villas in the grounds of the various branches can be provided.

WANTAGE HOUSE.

This is a reception hospital, in detached grounds with a separate entrance, to which patients can be admitted. It is equipped with all the apparatus for the complete investigation and treatment of Mental and Nervous Disorders by the most modern methods; insulin treatment is available for suitable cases. It contains special departments for hydrotherapy by various methods, including Turkish and Russian Baths, the prolonged immersion bath, Vichy Douche, Scotch Douche, Electrical Baths, Plombières treatment, etc. There is an Operating Theatre, a Dental Surgery, an X-ray room, an Ultra-violet Apparatus, and a department for Diathermy and High-frequency treatment. It also contains Laboratories for biochemical, bacteriological, and pathological research. Psychotherapeutic treatment is employed when indicated.

MOULTON PARK.

Two miles from the main Hospital there are several branch establishments and villas situated in a park and farm of 650 acres. Milk, meat, fruit and vegetables are supplied to the Hospital from the farm, gardens and orchards of Moulton Park. Occupational therapy is a feature of this branch, and patients are given every facility for occupying themselves in farming, gardening and fruit growing.

BRYN-Y-NEUADD HALL.

The Seaside house of St. Andrew's Hospital is beautifully situated in a park of 330 acres, at Llanfairfechan, amidst the finest scenery in North Wales. On the north-west side of the Estate a mile of sea coast forms the boundary. Patients may visit this branch for a short seaside change or for longer periods. The Hospital has its own private bathing house on the seashore. There is trout-fishing in the park.

At all the branches of the Hospital there are cricket grounds, football and hockey grounds, lawn tennis courts (grass and hard courts), croquet grounds, golf courses and bowling greens. Ladies and gentlemen have their own gardens, and facilities are provided for handicrafts, such as carpentry, etc.

For terms and further particulars apply to the Medical Superintendent ('Phone : 2356 and 2357 Northampton), who can be seen in London by appointment.

Section of the History of Medicine

President—J. F. HALLS DALLY, M.D.

[October 1, 1941]

John Caius and the Revival of Learning

By Sir WALTER LANGDON-BROWN, M.D.

It is typical of the present break with tradition that when a new President was installed at the Royal College of Physicians on the day following Palm Sunday 1941 he was the first for nearly four centuries who did not receive at the hands of the Senior Censor the silver caduceus, the gift of John Caius. That insignia of office, which the President alone may carry, is at present buried in the vault of a bank. What manner of man was this John Caius, repeatedly President of the College of Physicians, the re-founder and later Master of the College which now goes by his name, the author of the first medical treatise in English, who introduced the study of anatomy in the University of Cambridge? What part did he play in the revival of learning?

The more one considers the Middle Ages the more one is impressed by the idea that they were divided by two contemporary events like a chasm, the flight of the Popes to Avignon and the Black Death. The broken, or more correctly the uncompleted bridge at Avignon is symbolic, reaching as it does from the fortified Palace of the Popes to end abruptly in mid-stream of the turbulent Rhone. For this bridge, like the church at Winchelsea, was never completed because of the Black Death. These two events changed the religious, political and social face of Europe, just as it is changing to-day. If it is perhaps extravagant to maintain that this Papal retreat had within it the seeds of the Reformation, it was undoubtedly a blow to religious unity, while the Black Death profoundly affected the whole social system. The reaction to the resulting difficulties then as after the last war was the assertion of ruthless authority; the difference being that now the successors of John Ball have been able to seize the reins instead of the Church. But in both cases a repression of intellectual freedom followed. It is not surprising therefore that at the outset the revival of learning took an authoritarian form. Till recently we have been inclined to attribute too large a share in the Renaissance to the fall of Constantinople; now we realize that the fertilizing seeds that were then broadcast into Western Europe fell on soil already prepared to receive them. That the first harvest from those seeds was strictly a revival is shown by the establishment by Cosmo de Medici of a Platonic Academy at Florence, which his son Piero and his grandson Lorenzo the Magnificent also ardently supported. The teachers and students of that academy are delightfully portrayed on the walls of the old Medici chapel at Florence in Benozzo Gozzoli's fresco *The Magi* with all his characteristic springtime charm. Sir George Newman eloquently says: "It was a gay and pagan world of carnivals, masquerades,

tournaments, revelry and dissipation, in the midst of which a mighty transformation of the human mind was already in progress. Its setting was the garden of Florence. Into this beautiful city of flowers, colour and song there came about 1485 a grave, studious and sober-minded Englishman, Thomas Linacre. . . . He came out of the shades of scholasticism at Oxford into the sunlight of a larger life and a wider purpose."

This is not the occasion to expatiate on the admittedly great services of Linacre to English medicine. It was rather startling, however, when Professor Topley said in the lecture founded by Linacre's own bequest that the only reason he did not do more harm as a physician than he did was that the times were too much for him. Surely this is to be wise after the event. The first necessary step was the preparation of accurate Greek texts and that he saw to; one can hardly blame him if he did not foresee the next step, that of experiment, when so much had to be done to put medicine on what he believed to be a sound basis. That this basis must be authoritarian his whole upbringing would declare.

John Caius was born at Norwich half a century after Linacre and was only 14 years old when Linacre died in 1524. His devotion to Linacre's memory was lifelong and was exemplified by his erecting a monument in 1537 over Linacre's neglected grave in St. Paul's Cathedral. The family name was Keys, and although he followed the prevalent fashion of latinizing the spelling of it, the original pronunciation was retained. He was 19, rather older than the undergraduates of that time when he entered Conville Hall. This College originally stood between Free School Lane and the churchyard of St. Botolph's, a site which is now the Master's garden at Corpus, although at one time it was a plague pit from which bones still turn up. In 1353, only five years after its foundation, William Bateman, Bishop of Norwich, moved the College to where Conville Court of the present college now stands and renamed it the "Hall of the Annunciation of the B.V.M.", though this name seems seldom to have been used. Conville Hall, however, retained land on the other side of Free School Lane, so-called because here was built the school endowed by Stephen Perse, Senior Fellow of the College in 1615. Here it remained until the last decade of the nineteenth century when it migrated and the School of Engineering took its place, to be succeeded in its turn by an extension of the ever-growing Cavendish Laboratory. Caius' original bent was towards theology, to which he made contributions before he was 21. He graduated at the head of the list in 1532 and in the following year was appointed Principal of Physwick Hostel. This hostel, now absorbed into Trinity College, was then a branch of Conville Hall, and was governed by two principals, one chosen from among its own scholars, the other from Conville Hall. It was distinguished by the number of learned men it sent out into the world. In that year he was also elected Fellow of Conville. In 1539 he set out to study medicine under Montana at Padua, that famous university formed by a secession from Bologna in 1222. It may fairly be urged that the modern study of anatomy began with the artists, and those who have seen the frescoes of Signorelli in the cathedral at Orvieto must realize that here was a new approach to the study of the human body, which was carried to a much higher pitch by the great genius of Leonardo. In 1516, three years before the death of Leonardo, there was born in Brussels Andreas Vesalius who was destined to do for anatomy what his contemporary Copernicus did for astronomy. Since a man's talents are so often accredited to his mother, let it be kept on record that the mother of Vesalius was an Englishwoman, whose maiden name was Isabella Crabbe. He went to Paris to study under Sylvius for four years and was then appointed Professor of Surgery and Anatomy at Padua at the early age of 22. Here he discarded Galenic tradition and set his students dissecting the human body for themselves, as Frederick II had enjoined at Salerno three centuries before. As is well known Caius lodged in the same house as Vesalius at Padua and came very much under his influence. He thus obtained a training in direct observation for which Linacre had no such opportunity. Vesalius published his great work, *De Fabrica Humani Corporis* in 1548, the same year as Copernicus published his. It is tragic that subsequently abandoning anatomy he was compelled by the Pope to undertake a pilgrimage to Jerusalem as a penalty for contradicting the authority of Galen; for on his return journey he was drowned off the island of Zante at the early age of 49. Thus he joined the ranks of the martyrs to science, to be followed by Giordano Bruno and Lavoisier, a roll to which the authoritarian of to-day is rapidly adding. Though Archimedes shouted "Eureka" the last word lay with the Roman soldier.

After taking his M.D. in 1541 Caius became Professor of Greek at Padua, a post he appears to have held for only two years, as in 1543 he began a tour all over Italy studying at Florence and Pisa and visiting all the most celebrated libraries to collate MSS., principally those of Galen and Celsus. He enjoyed the hospitality of Cosmo de Medici, and as he writes of a visit to Fiesole it may be assumed that he stayed at the famous Villa Medici there. Urbino and Ferrara yielded him some reward for his researches, Bologna very little and Siena none at all. He found a good manuscript of Pliny at St. Maria Novella in Florence, and studied the Greek manuscripts in the Vatican Library. "When I say books", he remarked, "I generally mean Greek manuscripts". He gave a description of the ruins of ancient Rome, of which there was far more to be seen then than in later years until the modern excavations. Sir Norman Moore tells us that he travelled back through Germany and made one friend who added much to his happiness, Conrad Gesner. This learned and kindly physician, Greek scholar, botanist and writer on natural history, died of the plague in 1565 at the early age of 48. Caius was deeply afflicted and felt his loss more and not less as time went on. This is to anticipate, however, for Caius returned to England in 1547 and was elected F.R.C.P. Venn can find no evidence for the legend that he practised at Cambridge, Norwich and Shrewsbury. In 1551 there was an outbreak of sweating sickness at the last-named town, and Caius certainly went to investigate it; in the following year he published an account of this disease, the first medical treatise in English, though he subsequently translated it into Latin. It must also have been the first medical treatise for a long time that was based on personal observation and not merely a compilation of authorities. His publisher was Richard Grafton who had been Treasurer of St. Bartholomew's. This may well have influenced his choice of abode when he took up his residence in London that same year for he was granted a lease of the house within the precincts of the hospital just inside the Smithfield gate. It faced the Church of St. Bartholomew the Less, occupying part of the site of the present pathological laboratory. It does not seem to have been very commodious except for the hall, which was spacious enough to accommodate the whole College of Physicians at their first college feast in 1556. For the rest it seems to have consisted merely of a kitchen, two or three bedrooms and a garret. Yet Caius must have liked it for he retained its possession throughout his lengthy and repeated absences in Cambridge and he actually died there. Nevertheless though he was in Bart's he was not of Bart's; apart from some minor benefactions to the hospital his relationship to it remained merely that of a tenant towards a landlord. He never entered the wards as far as is known, though he must have walked across to the Little Britain gateway on his way to give his lectures on Anatomy at the Barber Surgeons Hall, which survived the fire of London in 1666 to perish in the German incendiary raid of 1940. Here Caius lectured for nearly twenty years in spite of many other demands on his time. For he had a large and lucrative practice and was physician to three sovereigns in succession, Edward VI, Mary and Elizabeth. By this time he was an "Elect" at the College of Physicians, i.e. one of the eight senior Fellows who withdrew into another room and like a little conclave of Cardinals elected one of themselves, returning to the Comitia to announce the happy tidings. Moreover when a vacancy occurred among the elect it was filled by the votes of the remaining seven. This extraordinary self-propagating method was brought to an end in an interesting manner, as was related to me by Dr. Sidney Phillips, when he was Treasurer. When the General Medical Council was created by the Medical Act of 1858 the College not unnaturally became fearful of the loss of their privileges. So the officials approached Graham, then Home Secretary. He promised to do what he could to conserve their position but on one condition. He told them frankly that he disliked their method of electing their President, and this must be altered if he was to give his support. Accordingly in the amended Medical Act of 1860 it was laid down that the election of President was always to take place on the day following Palm Sunday and carried out according to the bye-laws of the College for the time being. At the same time he made it clear that he expected the method to be adopted of free election by ballot without preceding nominations as it is to this day.

Caius became President in 1555 and was re-elected annually till 1560. He was most industrious in its interests. He collected its annals from scattered papers into a volume, and therein wrote the records on a plan that has been continued ever since. He presented the silver caduceus to which I have alluded, and with the love of symbolism which

tournaments, revelry and dissipation, in the midst of which a mighty transformation of the human mind was already in progress. Its setting was the garden of Florence. Into this beautiful city of flowers, colour and song there came about 1485 a grave, studious and sober-minded Englishman, Thomas Linacre. . . . He came out of the shades of scholasticism at Oxford into the sunlight of a larger life and a wider purpose."

This is not the occasion to expatiate on the admittedly great services of Linacre to English medicine. It was rather startling, however, when Professor Topley said in the lecture founded by Linacre's own bequest that the only reason he did not do more harm as a physician than he did was that the times were too much for him. Surely this is to be wise after the event. The first necessary step was the preparation of accurate Greek texts and that he saw to; one can hardly blame him if he did not foresee the next step, that of experiment, when so much had to be done to put medicine on what he believed to be a sound basis. That this basis must be authoritarian his whole upbringing would declare.

John Caius was born at Norwich half a century after Linacre and was only 14 years old when Linacre died in 1524. His devotion to Linacre's memory was lifelong and was exemplified by his erecting a monument in 1537 over Linacre's neglected grave in St. Paul's Cathedral. The family name was Keys, and although he followed the prevalent fashion of latinizing the spelling of it, the original pronunciation was retained. He was 19, rather older than the undergraduates of that time when he entered Gonville Hall. This College originally stood between Free School Lane and the churchyard of St. Botolph's, a site which is now the Master's garden at Corpus, although at one time it was a plague pit from which bones still turn up. In 1353, only five years after its foundation, William Bateman, Bishop of Norwich, moved the College to where Gonville Court of the present college now stands and renamed it the "Hall of the Annunciation of the B.V.M.", though this name seems seldom to have been used. Gonville Hall, however, retained land on the other side of Free School Lane, so-called because here was built the school endowed by Stephen Perse, Senior Fellow of the College in 1615. Here it remained until the last decade of the nineteenth century when it migrated and the School of Engineering took its place, to be succeeded in its turn by an extension of the ever-growing Cavendish Laboratory. Caius' original bent was towards theology, to which he made contributions before he was 21. He graduated at the head of the list in 1532 and in the following year was appointed Principal of Physwick Hostel. This hostel, now absorbed into Trinity College, was then a branch of Gonville Hall, and was governed by two principals, one chosen from among its own scholars, the other from Gonville Hall. It was distinguished by the number of learned men it sent out into the world. In that year he was also elected Fellow of Gonville. In 1539 he set out to study medicine under Montana at Padua, that famous university formed by a secession from Bologna in 1222. It may fairly be urged that the modern study of anatomy began with the artists, and those who have seen the frescoes of Signorelli in the cathedral at Orvieto must realize that there was a new approach to the study of the human body, which was carried to a much higher pitch by the great genius of Leonardo. In 1516, three years before the death of Leonardo, there was born in Brussels Andreas Vesalius who was destined to do for anatomy what his contemporary Copernicus did for astronomy. Since a man's talents are so often accredited to his mother, let it be kept on record that the mother of Vesalius was an Englishwoman, whose maiden name was Isabella Crabbe. He went to Paris to study under Sylvius for four years and was then appointed Professor of Surgery and Anatomy at Padua at the early age of 22. Here he discarded Galenic tradition and set his students dissecting the human body for themselves, as Frederick II had enjoined at Salerno three centuries before. As is well known Caius lodged in the same house as Vesalius at Padua and came very much under his influence. He thus obtained a training in direct observation for which Linacre had no such opportunity. Vesalius published his great work, *De Fabrica Humani Corporis* in 1548, the same year as Copernicus published his. It is tragic that subsequently abandoning anatomy he was compelled by the Pope to undertake a pilgrimage to Jerusalem as a penalty for contradicting the authority of Galen; for on his return journey he was drowned off the island of Zante at the early age of 49. Thus he joined the ranks of the martyrs to science, to be followed by Giordano Bruno and Lavoisier, a roll to which the authoritarian of to-day is rapidly adding. Though Archimedes shouted "Eureka" the last word lay with the Roman soldier.

handed over nearly all his wealth and landed property including the Manors of Croxley near Rickmansworth, Runciton and Burnham in Norfolk and Caxton in Cambridgeshire. He also bought four houses opposite St. Michael's Church from Trinity for the enlargement of the College. He confirmed the existing Master of Gonville, one Thomas Bacon, as Master of the new College, who, however, proved himself in the words of the College Annals: "Certainly serious, gentle and lovable but useless and negligent as a custodian". Consequently the next year the Fellows unanimously requested Caius to become Master, which he did, but refused to accept any salary while continually enriching the college with gifts of plate and valuable books.

Caius took the greatest personal interest in the rebuilding of the College. His study of Italian architecture enabled him to play a most useful part here. The new Caius Court was placed to the south of the old Gonville Hall, and its south side was to be merely a wall. We have become so accustomed to the idea of a closed quadrangle for a College Court that it is perhaps surprising to learn that no such form was adopted in either University until in 1352 was built the original Court of Corpus which is still standing. Caius believed this arrangement was unhygienic so he only had buildings on three sides of his new court, which also had the Fellows' garden to the east of it and the Masters' Garden to the west. Here we see the medical mind at work. But one of his chief cares, and one in which his love of symbolism is again declared, was the arrangement of the three gates—a small rather austere gate of entrance, the Gate of Humility in Trinity Street, the appropriate beginning to a student's career—an inner more sumptuous one under the tower, which bears the word *Sapientia* on its outer side and *Virtus* on the inner: these representing what he hoped the student would acquire; and the third a most ornate erection—the Gate of Honour by which the student left to take his degree in the adjoining Senate House. These gates are such an important landmark in Cambridge architecture that we must deal with them in some detail. The first Renaissance architecture to be found in this country is a Chantry in Christchurch Minster, of the date 1500. Though there is some Tudor architecture which has affinity with the Renaissance style. Professor A. E. Richardson holds that the first real Renaissance architecture in Cambridge is represented by these three gates. The Gate of Humility was removed by Waterhouse in 1869, but was fortunately preserved by someone who had more feeling for tradition than he had, and it can now be seen at the foot of the Masters' garden, the less interesting side being visible to the general public on going along the first staircase to the left after entering the Gate of Honour. There are engaged Ionic pilasters on either side with *Humilitas* incised on the lintel, and a more ornate pediment. Under this is a simple arch. The Gate of Virtue is a noble work; it has been ascribed to John of Padua, a plausible ascription in view of Caius' old association with that city. Professor Richardson points out however that the design follows even in minute details of mouldings one in the book of designs published by Gianbattista Alberti. An architect friend of mine tells me that the book of Alberti's designs had in their day as widespread a popularity as that of Chippendale two centuries later. The foundation stone of this gate was laid with great pomp and ceremony in 1565 by Caius himself and the Gate of Humility was built at the same time. The Gate of Honour was not built till 1574, the year following the death of Caius, who had, however, left the most precise plans for its construction. It is extremely ornate and has been regarded as being with the fountain in Trinity Great Court the best examples of Elizabethan Renaissance which the University possesses. To me it is delightful, but it is rather too exotic for the taste of many moderns. It has two storeys of the Italianized Ionic and Corinthian orders, while the arch of the doorway is in the Tudor style with classical mouldings. Over this is the word "*Honoris*" and the whole is surmounted by a solid cupola of stone. Originally it was even more decorated for there was a weathercock on the top made in the shape of a serpent and dove. The commentary accompanying the plans shows that in every detail this gate was of symbolic significance to Caius. Perhaps the most beautiful ceremonial use of the gate now is on the occasion of the funeral of a Fellow. The Gate is closed, until at the end of the service the bier is brought up to it; the choir ranged on either side chants the *Nunc Dimittis*; the gate is thrown wide open and the body is borne to its last resting place, thus leaving its former abode with honour.

A decorative feature of the court that Caius built which has now completely vanished was in the words of the College Annals a pillar and a stone "of exquisite and wonderful

characterized him explained that this sceptre of silver indicated that the sway of the President should be mild, while the serpents at its head inculcated sagacity in rule and in act. He also presented a cushion on which it was to repose in honour, a book of the statutes bound in red velvet and decorated with silver, together with a seal "the token and support of fidelity". His election for the year 1560 was postponed till December 22 because of his absence in Cambridge, to the distraction of the Electors, as he said when he duly inscribed the story of his delinquency in the Annals. Perhaps it was to teach him a lesson that next year they elected Dr. Richard Master on St. Luke's Day. Master had been a beneficed clergyman but resigned because he felt he was not qualified by inclination or knowledge of the Bible, but also, as he said, because popery however abandoned in name flourished here in reality. So he applied himself successfully to the study and practice of physic, becoming physician to Queen Elizabeth two years before receiving the presidential caduceus. Whether he filled that office with distinction is not known, but at any rate Caius was forgiven and re-elected in 1562 and 1563. Then they reverted to Robert Huicke, who had previously been President in 1551 and 1552, and physician to Henry VIII. This could hardly have been an improvement for when he appealed against a suit of divorce, the Privy Council dismissed his appeal in 1546 with these words: "We have never in all our lives had matter that more pitied us; so much cruelty and circumvention appeared in the man, so little cause ministered by the woman." It does not speak well for the Elects that they should have chosen such a President three times after this open condemnation of his conduct.

Caius did not become President again till 1571, the ninth and last time, for his duties in Cambridge had become more imperative. Of those who occupied the Presidential Chair in the interval the most interesting is Richard Caldwell who was elected to the Fellowship and a Censorship on one and the same day. Only eleven years later he became President in 1570. The great debt the College owes him is his joining with Lord Lumley in founding the Lumleian lectures in 1582, and the importance of this gift was recognized by the erection of a suitable theatre. Yet no one could have anticipated that this gift would give the opportunity to William Harvey, who had attended Caius' anatomical demonstrations at Cambridge as we learn from his own notes, to make his great researches and that this theatre would witness the exhibition of an immortal discovery.

Of that last presidency urged upon Caius against his own wishes we know little, except that in the following year he was permitted to resign, if only he would come to the College for the quarterly comitia when any specially important business was to be discussed. It is sad to report that the Annals of the College which he had so scrupulously kept except for part of the time he was so busy in Cambridge are a complete blank for nine years after his resignation. Then Dr. Marbeck was appointed Registrar, since when there has been no gap in the recording, though the volume of that record for 1771-1781 is missing, having been abstracted by a Treasurer who presumably destroyed it because it contained the statement of his rejection by the Censors' Board when he first came up for examination.

One more fact about Caius' work for the College of Physicians which is quoted in Munk's Roll. He was the chosen defender of their privileges when the surgeons claimed the right to give internal remedies for sciatica, syphilis or any kind of ulcer or wound. In this they were supported by the Bishop of London and the Master of the Rolls. "He so learnedly defended the College rights and the illegality of the Surgeons' practice . . . that it was unanimously agreed by the Queen's Commissioners that it was unlawful" for them so to do.

I will now revert to his life at Cambridge; it seemed to me better to treat this separately rather than to attempt a chronological plan. In 1557 he brought before the authorities of Gonville Hall a scheme for an expansion and fresh endowment of the College without revealing that he was the intending benefactor. However, as it was discovered that the plan involved a charter of foundation, the Hall having no letters patent, he was declared a co-founder with Edmund Gonville and William Bateman. Appropriately, Lady Day in the first year of Queen Elizabeth's reign was chosen for the college to be re-dedicated to the Virgin Mary, though its name was now to be Gonville and Caius College. Caius

faculties. It is this which is one of the great distinctions between a university and a technical college. This is also entirely in the spirit of Caius who was interested in theology, and was a profound classical scholar as well as an antiquarian and the most distinguished natural historian of his time. Gesner described him to Queen Elizabeth as the most learned physician of his age. He contributed articles on rare animals and plants for Gesner's *Historia Animalium*, and wrote a treatise on British dogs which was included in Pennant's *British Zoology*. He was a protagonist for what we now call the modern pronunciation of Latin and Greek from which England, and England alone, had departed.

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The legend of the vast antiquity of Cambridge had one curious survival. Close by the orchard of St. John's College is an ancient building known as the School of Pythagoras, and in the past it was pointed out as the most ancient academic building in Cambridge. Actually it was never so called until the time of Caius, who labelled it thus in support of his contention. In point of fact it is well known that the original Norman building was merely called the Stone House, which belonged to the family of Dunning, believed to be the forerunners of Sir George Downing of Gamlingay, who founded Downing College and whose grandfather gave his name to the famous street in Whitehall. Walter de Merton purchased the Stone House in 1270 not long after he had founded his "Hall of Scholars" at Oxford. Becoming alarmed that the issue of the Barons' War against Henry III would imperil his Oxford foundation, he held this Cambridge property in reserve ready to transfer his scholars hither. The crisis never came and so the Stone House was never used for any academic purpose, except curiously enough from 1872 to 1874 as the original home for Newnham College for Women. To-day it forms part of Merton Hall, the residence of Lord Rothschild.

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It is sad to record that despite all he had done for learning and for the College he refounded, Caius' tenure of the Mastership was not a peaceful one. His relations with the fellows of the College became strained; according to Venn the fellows were narrow-minded and bitter, very young, none of them in 1564 being 25 years old—and Caius certainly expelled some of them. At the instance of the fellows the dispute was referred to the Archbishop of Canterbury who blamed both parties, the master the more severely. Yet the fellows quite inconsistently refused to accept the finding and carried the matter to Lord Burghley, the Chancellor. The question of depriving Caius of his mastership was

workmanship, bearing 60 dials (horologia) placed upon it . . . and adorned with the arms of those who were at that time resident in the college and given by him to the college as a memorial of his good wishes towards it." Presumably this curious and interesting object fell a victim to an earlier Waterhouse. The College Annals kept as scrupulously as those instituted by Caius at the College of Physicians give a complete account of the expenses of Caius' buildings—they amounted to £1,834 4s. 2d., a considerable sum for those days—"besyde the expence ommytted by negligence and expences also yet to come for the perfection of the buyldynge of the College and payynge of the courts of the same". In these accounts one may notice the distinction drawn between the sums paid to the freemasons, the rough masons and the labourers.

It will be convenient here to deal briefly with the chief architectural extensions of the college since the time of Caius. His immediate successor Thomas Legge, who died in 1607, left money for a range of buildings facing St. Michael's Church while Stephen Perse in 1615 bequeathed some more for a block at right angles to it, i.e. facing Trinity Lane. Thus Tree Court was formed, but the Gate of Humility and the adjacent garden were spared. In the later part of the seventeenth century the Chapel was rebuilt and then in 1869 Waterhouse destroyed the quite inoffensive buildings of Legge and Perse, removed the Gate of Humility and erected his extraordinary version of a French chateau which obtrudes itself on the eye of every beholder coming up King's Parade, overpowering the classical outlines of Gibbs' Senate House. A Fellow of Caius criticizing some recent additions to Peterhouse to the late Lord Chalmers while he was Master of that College, was met with the rejoinder "Those who live in Waterhouses should not throw stones". Of no one is it truer than of architects that the evil men do lives after them. Yet tastes change and as someone recently remarked, in another thirty years the vanguard of the highbrows may be holding this building up to admiration. Quite possibly, for I note that in Le Keux's *Memorials of Cambridge* published in 1847, the classical perfection of Gibbs' Fellows' building of King's College is referred to as mean and incongruous while the sham Gothic veneer of stucco imposed on the late Elizabethan red brick buildings of Sidney Sussex is highly praised.

In the '90s of last century the houses on the inner curve of Rose Crescent were altered and added to the College as St. Michael's Court. And now that has been extended to the Market Place in the best style of factory architecture with a row of shops underneath. Symbolical no doubt, but not exactly in the tradition of Caius' symbolism!

As already stated, Caius was mainly responsible for introducing the study of anatomy into England. This he did, not only at the Barber Surgeons Hall in 1546 but at Cambridge in 1557. In 1565 this was regularized by a formal annual grant of two bodies of criminals or unknown strangers for dissection in Caius College. After his time the Regius Professors of Physic were responsible for the teaching of anatomy, a duty that was often neglected by them even including the famous Glisson. It was not until 1707 that a Chair of Anatomy was instituted, and ill fortune still followed, for George Rolfe the first occupant was after unheeded warnings deprived of his Chair for neglect of his duties.

It must not be imagined that although Caius was a medical man he intended his college to be devoted solely to the study of medicine; his interests were far too wide for that. Moreover until after 1870 the number of Cambridge medical graduates was quite small. Nevertheless his college has always had a reputation in medicine. Of the 21 Regius Professors of Physic 7 were Caius men originally, while the present occupant of the Chair was incorporated into that college. One of these, Glisson was also President of the College of Physicians. St. John's College comes next with 5. Strange to say Trinity, so rich in other professorships, has never filled the Chair of Physic.

When the medical school began to grow so rapidly towards the end of last century this medical reputation of Gonville and Caius attracted so many medical students as to embarrass the then tutors. These two powerful personalities, E. S. Roberts, afterwards Master, and J. S. Reid afterwards Professor of Ancient History, were determined that the college should not become regarded as solely or even mainly medical. In this they were quite right if sometimes rather ruthless. For obviously the great advantage of the collegiate system is the opportunity it gives for the mixing socially of students in the different

faculties. It is this which is one of the great distinctions between a university and a technical college. This is also entirely in the spirit of Caius who was interested in theology, and was a profound classical scholar as well as an antiquarian and the most distinguished natural historian of his time. Gesner described him to Queen Elizabeth as the most learned physician of his age. He contributed articles on rare animals and plants for Gesner's *Historia Animalium*, and wrote a treatise on British dogs which was included in Pennant's *British Zoology*. He was a protagonist for what we now call the modern pronunciation of Latin and Greek from which England, and England alone, had departed.

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discussed but finally nothing was done. Caius having been accused by his fellows of being an atheist was later accused of being a papist. Pitseus gives it as his opinion that he had no very determinate ideas in religion and observes that he was always of the same religion as the reigning monarch. My own feeling after studying his career is that his religion was that of all sensible men, that is the one which no sensible man ever tells. He must have been inclined to cry "a plague on both your houses", but I think he had a decided leaning to the æsthetic and ritual side of Catholicism. Certainly he kept a great number of Catholic vestments and ornaments for the altar with other things in order to save the College the cost of buying more should it please the sovereign to change the religion of the state again—for after all Caius had seen four alterations within twenty-five years. This came to the knowledge of Dr. Sandys, Bishop of London, who with Dr. Byng, Master of Clare, and Vice-Chancellor of the University organized a raid on Caius' lodge and found in the Bishop's elegant language "much popish trumpery". "It was thought good", he continued, "by the whole consent of the heads of howses, to burne the books and such other things as served most for idolatrous abuses and to cause the rest to be defacid." This outrage was carried out in the presence among others of Whitgift, then Master of Trinity and afterwards Archbishop of Canterbury, who had already ejected Cartwright from his Chair on the opposite charge of Puritanism, and thus laid the foundation of a reputation for bigotry and persecution which he so consistently maintained as Primate of all England.

This blow seems to have broken Caius. He went to London, returning six months later to resign his mastership in favour of Legge on June 27, 1573, and took up his quarters in rooms over the Gate of Virtue and Wisdom for the short time that remained, for he predicted that he would die on July 29 and had his grave prepared in the college chapel early in that month. His death actually occurred on that date while in his little house at Barts, he being not quite three months short of 63 years of age. His body was disembowelled, those viscera being interred in the Hospital Church but the rest of his remains were brought to Cambridge according to his testamentary directions. At Trumpington Ford his coffin was met by Whitgift who was then Vice-Chancellor and who I hope had some feelings of remorse, as well as by the Master and Fellows of his college and many others. Thus he returned for the last time "in honourable procession" with as Matthew Parker said "the greatest funeral pomp" to his beloved College where little more than seven months previous they had done their best to break his heart. Mindful of his own latter end he had prepared the inscription for his tomb and it is interesting to note that he used the phrase he had placed on Linacre's tomb in St. Paul's "Vivit post funera Virtus" but whereas that formed part of a long epitaph, in his own case he simply added with a brevity as dignified as it is pathetic, "Fui Caius".

His grave was twice disturbed during alterations to the chapel, once in 1719 and again in 1891. On the latter occasion Professor Macalister estimated from the length of the femur that his height did not exceed 5 foot 1 inch. One is reminded of the child's comment in *Cavalcade* at the sight of Queen Victoria's coffin, "She must have been a very little lady". But stature in cubits does not accord with character, and the world has often been much indebted to those who lacked inches. That Caius became eccentric in his latter years seems probable and I have sometimes wondered whether the Dr. Caius of *The Merry Wives of Windsor* was based upon the memory of his curious ways. True the play was written more than twenty years after the death of John Caius, and the physician in it is a Frenchman. What views have Shakespearian scholars on this?

I believe that Caius was a man who was afraid of personal relations. He never married and his only intimate friend was Conrad Gesner. The way he grieved over the loss of Gesner for the remaining eight years of his own life suggests why he was unwilling to lend his heart to be torn by bereavements. But if friends die, institutions do not when carefully tended, and so he gave his heart to the College of Physicians and to his college at Cambridge. He adorned them both with gifts like a lover. Here his repressed affections found outlet. And then—the bitterness of it, his college turned against him, his University despoiled him. Perhaps even a worse grief was that he began to doubt his wisdom in supporting the new learning, when he saw the excesses to which men were led when they had thrown off the shackles of authority. Walter Raleigh expressed the point finely when he said: "That great movement of the mind of man brought with it the exhilara-

tion of an untried freedom and the zest of unlimited experiment; but it took the human soul from its station in a balanced and rounded scheme of things to deliver it over to every kind of danger and excess; . . . [man] was like a child out of school, trying his strength and resource in all kinds of fantastic and extravagant attempts." That I think is what Caius felt in his later life, just as Fairfax came to feel about the attitude of Parliament to Charles I and Wordsworth about the French Revolution. Emancipation brought disillusionment in its train. Is not the same feeling in the air to-day, as we witness the overthrow of moral standards which had seemed to our fathers to be absolute? And can we not sympathize with Caius as he watched his college drifting towards the unknown, when we are watching institutions to which we have devoted much of our lives in peril of change? What the future holds for them we know not, but we suspect it will hardly accord with the hopes we held. Still the adaptability of man is extraordinary and the brave new world to come will doubtless have virtues all its own. If Caius returned he would find his college flourishing beyond his wildest dreams. "Fui Caius", but the past tense loses its poignancy in the living present which truly proclaims of him "Vivit post funera virtus". For his name is not "one that is writ in water".

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Paracelsus: Personality, Doctrines and His Alleged Influence in the Reform of Medicine

By H. P. BAYON, M.D., PH.D.

(St. Catharine's College, Cambridge)

AUREOLUS PHILIPP THEOPHRAST BOMBAST AB HOHENHEIM—commonly known as Paracelsus—died in Salzburg on September 24, 1541; the 400th anniversary of his death may be commemorated by an essay to evaluate the historical significance of his doctrines in relation to the development of medicine and modern science.

PARACELSIAN LITERATURE

From contemporary testimony it is known that Paracelsus was a copious author, but that, notwithstanding his efforts, only few of his writings were printed and published during his lifetime. After his death, Paracelsian writings were collected and edited, but there is reason to believe that among these several supposititious works made their appearance, for imitators were numerous.

For example, Johann Thölde published the *Triumph Wagen Antimonii* (Leipzig, 1604), which purported to be the MS. of a fourteenth century Benedictine monk, Basil Valentine, but in reality consisted of transcriptions from various Paracelsian works. This book gave rise to considerable controversy, because until Basil Valentine was shown to be a forger, it seemed that Paracelsus had copied from this MS.

Sudhoff undertook to edit the genuine writings in *Bibliotheca paracelsica* (Berlin, 1894) and traced 23 publications *intra vitam Paracelsi*, including several *Prognosticationes* relative to judicial astrology. He then had the extant MSS. printed in *Paracelsus-Handschriften, gesammelt und besprochen* (Berlin, 1899). Later, Sudhoff and Matthiessen published several of the religious writings of Paracelsus in *Archiv für Reformationsgeschichte* 1917-8. Lastly, Sudhoff collected all medical works in fourteen volumes under the title *Theophrast von Hohenheim, genannt Paracelsus* (Munich-Berlin, 1922-33) and incorporated the results of his studies in a small readable book: *Paracelsus* (Leipzig, 1936). Even those who disagree with Sudhoff's conclusions about the merits of Paracelsus, must admit that the investigations of the Leipzig professor were prolonged and extensive; yet he failed in achieving the recognition of the genuine writings of his hero. It is ascertained that the only major work that appeared in the lifetime of Paracelsus was *Die grosse Wundartzney* (Augsburg, 1536), Steyner.

There is a large and widespread Paracelsian literature in many languages, but it is not captious to say that a great deal is based on erroneous assumptions. Too often the appraisement of Paracelsus has been founded on incorrect statements about his activities; so that a thumbnail sketch of his life is necessary to sift from the assertions of his admirers or detractors much that is irrelevant or grossly inaccurate.

BIOGRAPHICAL NOTES AND COMMENTS

Paracelsus was born at Einsiedeln, Canton Schwyz, in the latter part of the year 1493, as son of the licensed medical practitioner Wilhelm Bombast ab Hohenheim, whose wife appears to have been a nurse or midwife; she died during the infancy of her only child. In 1502 Wilhelm ab Hohenheim moved to Villach in Carinthia, where mines belonging to the Fuggers of Augsburg were situated; here Paracelsus became interested in technical chemistry, but nothing definite is known of his youthful studies, if any; at the age of 22—that is in 1515—he visited the Fugger mines at Schwatz in Tyrol and was there about a year. Then he began his travels and—if his statements can be trusted—visited the High Schools of Germany, Italy, France; he was also in Granada in Spain, Lisbon in Portugal, Barcelona in Catalonia, England, Denmark, Prussia, Latvia, Poland, Hungary, Rumania, Croatia, Dalmatia, Sicily, Constantinople, Rhodes, Samos, Candia, Alexandria in Egypt. It is, however, definite that in August 1524 he resided in Salzburg, which he left in May 1525 and appeared in Strassbourg in 1526, as a famous practitioner of medicine, so that he was appointed town-physician in December of the same year. He soon left for Basel, having been called to treat Johann Fröben, the well-known book-publisher, at whose house Paracelsus met Erasmus, Oecolampadius and, no doubt, several other theologians. Such men could not fail to influence Paracelsus—and indeed echoes of their opinions are found in his writings.

On the recommendation of Erasmus and Frobenius, Paracelsus was appointed Basel Town Physician, one of whose duties it was to lecture on Medicine at the University: the announcement of his lectures was dated June 5, 1527; in this he declared himself as: *Bombast ex Hohenheim Heremita, utriusque medicinar doctor ac professor*. The tone was moderate—when compared with what Paracelsus said in his books—and had he abided by his programme he would have scarcely aroused so much hostility. But barely three weeks after, on June 24, he threw a medical book—not clearly defined by him as *Summe der Bücher*—into a bonfire the students had lit on Midsummer's night. This incident was later greatly magnified and considered as an outstanding event in the history of medicine; the volume was said to have been a tome of Galen or an even more ponderous Avicenna; while in reality it must have been a small compendium and the deed was inspired by Luther burning the Papal Bull at Wittenberg on December 10, 1520. Then Paracelsus lectured in German, no doubt in the style we know from his writings—*grob Schwyzer-Dütsch*—and obtained a much increased number of hearers—some thirty or more, instead of the half-dozen of his predecessor. The medical profession in Basel cannot be blamed for objecting to such antics and they prepared a counterblast in Latin entitled *Nomes Galeni adversus Theophrastum, sed potius Cacophrastum*, had it printed and posted in various public places. Copies still exist and in the text it was doubted whether Paracelsus was worthy of feeding the swine of Hippocrates or carrying his urinal. Paracelsus complained about the libel to the magistrates, became involved in a squabble about fees

with a patient, Canon Lichtenberger, and left the town in anger early in February 1528. Thus his academical activity was brief, even if lively, corresponding to what might be called one semester.

Meanwhile, on May 21, 1527, in a signed document, he had stated that he was a Doctor of Medicine of the "Praiseworthy High School of Ferrara" and henceforth frequently added to his signature *beider Artzney Doctor* = Doctor in both medicines; this may have meant Medicine and Surgery. There is no concrete evidence that he ever passed an examination at Ferrara and indeed the physicians at Basel denied that he was qualified to teach. It seems that either his extensive travelling was exaggerated or that in the five years between 1516 and 1521 he could not have covered so much land and yet attended a medical course leading to a degree; unless it was granted *Honoris causa*. Sudhoff ("Paracelsus," Leipzig, 1936) pieced together the further wanderings of Paracelsus, who was at Colmar end of February 1528, in Nuremberg during 1529 and henceforth resided, or visited, several Swiss, German or Austrian towns, such as St. Gall, Regensburg, Innsbruck. He seems to have travelled to patients by whom he was consulted; it is recorded that on one such occasion he called the ordinary medical attendants "Arschkratzer" and prescribed minced live earthworms as a dressing for a phlegmon of the hand; the treatment was successful. He reached Salzburg late in 1540, dying there next year following an undiagnosed illness. His will has been found and its several bequests show that he must have obtained a modest competence.

THE PHYSIOGNOMY AND PERSONALITY OF PARACELSUS

A portrait of Wilhelm von Hohenheim still exists and shows a certain resemblance to some of the paintings or engravings supposed to depict his son. In my opinion, the pictures which represent Paracelsus as a stately handsome, bearded person are fictitious, for he was a rather small, blond man of the "eunuchoid" type, to use a contemporary expression. Sudhoff (1936) reported that the skull examined in the grave at Salzburg showed the stigmata of rhachitis, but I should like to suggest a different aetiological explanation, which agrees with many recorded observations viz.: Congenital syphilis. The diagnosis is supported by his eunuchoid type—testicular atrophy; early baldness and premature senility; square cranium; death at the age of 47-48; it has been suggested by Sudhoff (1936) that Paracelsus was jaundiced during the last year of his life; if that could be ascertained it would be additional evidence.

It would appear that Paracelsus was a heavy drinker; the evidence is a letter of Johannes Herbst (Oporinus), the renowned book-publisher, who had been his "famulus" at Basel in 1527-8. Writing November 26, 1555, to Johann Weyer, Oporinus said that Paracelsus, even when drunk, could dictate speeches in German (to be translated into Latin) which a sober man could not have improved; that he would return home at midnight, throw himself on his bed fully dressed and then get up waving his sword. Sudhoff—and others—have tried to contradict such evidence, but the letter of Oporinus is a clear description by a reliable contemporary witness of drunken conduct and is moreover confirmed by other letters which refer to Paracelsus as a convivial toper and agrees with the notes of Reuchlin about the behaviour of Paracelsus at St. Gall.

WHO WERE THE TEACHERS OF PARACELSUS?

Sudhoff (1936) wrote in the preface l.c.:

Seit über fünfzig Jahren habe ich mich mit dem Werken und Wirken dieses grossen, urdeutschen Mannes beschäftigt, der die Bindungen der Naturwissenschaften des Mittelalters an antike Vorstellungen löste und seine ärztliche Tätigkeit und sein Wissen allein aus dem Selbst-Forschen ableitete.

It is not quite correct that Paracelsus derived his knowledge and his medical practice from personal investigations alone; apart from the utter impossibility of such a process, it can be shown that he was original in few matters and in those he was abysmally wrong.

Paracelsus stated that he intended relying on his own observations and experience—*Alterius non sit qui suus esse potest*. He however acknowledged as teachers in the *Adepta philosophia* first of all his father then a Bishop Erhart, the artisans of the noble Sigmund Fugger of Schwartz and the Abbot of Sponheim. The latter is usually said to have been Johannes Trithemius (1462-1516), Abbot of Sponheim in Nahegau; but Sudhoff (1936), p. 13, asserted that instead Bruno, Graf von Sponheim was meant. Sudhoff's arguments

do not sound very convincing, because Abbot Bruno lived in the thirteenth century, while Trithemius dabbled in alchemy, expressed Neo-platonic views and said that when an ignorant man became a doctor, it was like putting a barrel outside a house where there was no wine to sell. There are many echoes of such opinions in Paracelsian writings.

Then Paracelsus stated that he had discussed philosophical and medical questions with doctors, barbers, bath-attendants, learned physicians, [old] women, magicians, wherever they may have been, in convents, in the company of noblemen or commoners, among the wise or foolish; from these he learnt that medicine was a dubious art, which could not promise a cure with any degree of certainty and moreover was mostly taught by those who did not know the first thing about the subject. Incidentally, this can be found much more entertainingly presented in the writings of Bernard Shaw, author of the apothegm: "He who can, does; he who cannot, teaches." Numerous other quotations could be made from the writings of Paracelsus which show that his medical outlook was that of the irregular practitioner of medicine—I avoid saying "quack" because it is an uncertain term. At the same time it is not denied that unorthodox practitioners of medicine have introduced valuable therapeutical means and methods; for example, the old wise woman who made Withering adopt digitalis.

A REMARKABLE DOCUMENT FROM FERRARA

The poet Lodovico Ariosto (1474-1533) of Ferrara, wrote *L'Erholato*—the word having a meaning similar to "The Herbarium": in this strange literary product, Maestro Antonio Faentino proclaimed that he was the pupil and heir of Nicola da Lonigo, i.e. Leonicens; that he had been made a Doctor of the Famous College of Ferrara, a Knight of the Golden Spur and was entitled to bear arms; moreover praised his own medical skill, for his fame had spread "nell'ingegnosa Alenagna, ducato d'Austria, Sassonia e Selesia, Fiandra col Brabant e nell'isola d'Olanda; Francia, Inghilterra, Scozia, Albania, la Bosina, la Romania, La Morca, l'Arcipelago e tutta la Grecia, la famosa città di Constantinopoli, Candia, Rodi, Cipro, Cairo, Jerusalem, Damasco, Soria". Faentino also boasted that his personal experience was superior to classical learning and recommended a never-failing, marvellous Electuary. *L'Erholato* was written about 1530 and the literary critics I have consulted were unable to say more than it was obviously a skit on the speech of a charlatan, probably meant to lampoon Antonio Cittadini of Faenza, who taught at Ferrara about the year 1474; but Cittadini was a serious professor of classics and medicine and no mean author; not the expansive boaster apparent in the text of Ariosto. The resemblance to what Paracelsus wrote is so striking, that it seems as if it was intended to be an echo of some of his pronouncements, that may either have been made in Ferrara or have been imitated by someone called Faentino. A chance discovery of some document may yet explain this strange resemblance, which is not without relevance in ascertaining the true personality of Paracelsus.

L'Erholato may help to answer the question whether Paracelsus obtained or was granted a medical degree at Ferrara. In his time, eminent teachers were at the University of Ferrara, e.g. Nicola Leonicens (1428-1524), the renowned commentator of Galen and critic of Pliny; Gian Battista del Monte (1498-1551) who recommended bedside teaching in academical medical study; Celio Calcagnini (1479-1541) who taught at Ferrara circa 1519, and by some, is thought to have anticipated Copernicus. Sudhoff (1936) p. 139 did quote Paracelsus writing in the dedicatory preface of the first edition of the *Grosse Wundartzney*: "... der treue lobwürdige Johann Manardus von Ferrara, den uns Gott nicht vergönnen wollte." This allusion did not appear in the following edition and affords no evidence of academical study. Giovanni Manardi (1462-1536), a great medical authority, followed Leonicens from 1526 to 1536. It does seem strange that Paracelsus never mentioned, were it only in a slighting manner, these luminaries of medicine in Ferrara; though it may be recalled that Harvey, who was so warmly attached to Padua, never referred in print to Galileo who taught there in his time. Thus anyone reading the *Grosse Wundartzney*, must find it difficult to believe that Paracelsus had really pursued a serious study of academical medicine as it was then known.

HIS RELIGIOUS BELIEFS

Paracelsus wrote a great deal about religious subjects, even if relatively little was published. Father Raymund Netzhammer (*Paracelsus: das wissenwerthe . . . und die*

neueste Forschungen, Einsiedeln, 1901) said, p. 128 "Far more in the domain of theology than even in medicine, does Paracelsus who sometimes called himself Doctor of Sacred Scripture, seem to recognize no authority, but to consider his own thinking and philosophizing as authoritative for him." This would be the standpoint of a devout Roman Catholic, but Schubert and Sudhoff (1887-9) concluded that Paracelsus, while opposing Roman hierarchy and its external forms of worship, also rejected all dissenting religious communities as "sects". My opinion is that Paracelsus remained outwardly a Roman Catholic, but inwardly felt that some Church Doctrines required restating in terms consonant with recently acquired knowledge of natural phenomena. This is evident in the fact that he accepted Baptism and Communion as the two principal roads that led to Heaven, but added what may be called a "chemical" explanation to both Sacraments. A similar intellectual attitude is noticeable in Miguel Serveto, who in his *Christianismi restitutio* (1553) gave a biological explanation of the miracle of the Annunciation. The religious views of Paracelsus seemed clearer when he pleaded for a Divine origin of the Healing Art and stated that physicians can heal either by faith or skill in medicine. The curing by faith could only apply to Christians, but the new foundations of medicine, as taught by him, were valid for all others; his conclusion was: "The physician is the servant of nature and God is the Master of Nature."

Paracelsus has been called the "Luther of Medicine" and though such designations are usually intended to be arresting and picturesque—nothing more—still the inference is that since Luther was the central figure of "The Reformation" so Paracelsus induced a reform of medicine. It is true that both were exponents of coarse German invective; but Luther attacked the Pope and Popery on the basis of theological knowledge and the Sacred Writings; whilst Paracelsus condemned both Galen and Avicenna as scribbling fools, abused all practitioners of academical medicine and praised his own personal skill. In relation to religious matters, differences are also noticeable; Luther propounded his own interpretation of pristine Christian beliefs, while Paracelsus—in so far he is understandable—would have substituted Roman Catholicism by Paracelsian religious doctrines which would have altered nearly all existing dogma.

[In the discussion after this lecture, Dr. R. O. Moon mentioned that Matthew Arnold had said that Cromwell was the Philistine in politics, Luther in religion and Bunyan in literature; it appeared—added Dr. Moon—that Paracelsus was the Philistine in medicine.]

PARACELSUS IN MEDICAL THEORY AND PRACTICE

A redeeming feature of the writings of Paracelsus is the high and noble concept he propounded of medical practice; it remains to be shown, indeed it can be doubted—if we are to judge from some of his letters—whether he succeeded in guiding his conduct by the light of such ideals. He stated that the four pillars on which medicine should rest were: philosophy, astrology, alkemy and *Virtus*; the latter was rather undefined, but corresponded to what is now understood as pharmaco-dynamics; accordingly Paracelsus mentioned a "Religion of medicaments". The recommendation of the study of astrology can be variously interpreted, but he certainly continued that neglect of anatomy, physiology and clinical observation, which had proved so disastrous for the progress of medicine, from the time of Salerno onwards. Then in place of the ætiological significance of the four humours of Galen, Paracelsus proposed five entities or *entia*, of which only the *ens veneni* seems to have any concrete foundation; the other four could be easily lumped together: *ens astrale, naturale, spirituale, deale*. Where the neglect of clinical observation becomes painfully evident in Paracelsus is in his description of the diseases of women, which consists in an exsiccous disquisition about the *Matrix*, not the anatomical uterus, but a nebulous concept, fit subject for endless chatter. This chapter is far inferior to the so-called Trotula of Salerno or Soranus. Paracelsus is often praised for having recommended the use of mercury in syphilis in place of guaiac; but this had already been proposed by Caspary Torella of Valencia in 1497. If the age and opportunities are taken into consideration, it does not seem that Paracelsus reveals any advance on what Roger Bacon (c. 1214-c. 1292) said in *De erroribus medicorum*. Thus John Ferguson wrote in the *Enc. Brit. XIVth edition*:

"... with Paracelsus' lofty views of the scope of medicine it is impossible to reconcile his ignorance, his superstition, his erroneous observations."

PHILOSOPHY AND ALCHEMY

Paracelsus has been considered a Neo-platonist, because he was an adherent of the Macrocosmos-microcosmos doctrine, hence his support of astrology; however as taught by Plotinus, and his direct disciples, Neo-platonism was a system of philosophy, the extent of which was unknown to Paracelsus.

In alchemy he propounded the existence of three elements in all bodies: Mercury, representing liquidity and volatility; Sulphur, the principle of combustibility; Salt, the nucleus of all that resisted fire and was permanent; these elements were noticeable in wood when it was burnt. Admittedly, no better opinions were presented before van Helmont's books were published and Robert Boyle (1627-91) performed his experiments; but then it can be said that they were true pioneers and not Paracelsus.

In relation to technical chemistry of metals his work did not reach the practical standard of the *De la Pirotechnia* (Venice, 1540), of Vannoccio Biringuccio (1480-c. 1538), or still less that of *De re metallica* (Basel, 1556), of the physician Georg Bauer [*Agricola*] (1494-1555). It must be mentioned that Paracelsus was the first to describe "Bergsucht" or miner's phthisis.

These were the views propounded during his lifetime; after his death books were published of which he was stated to be the author and several paracelsians arose and became known as Iatro-chemists; the most eminent would be Johann Baptista van Helmont (1577-1644), Oswald Croll (1580-1609), Franciscus Sylvius (*De le Boe*) (1598-1679); all three, be it noted, were University graduates.

THE ROMANTIC "ROSA ET CRUCE" LEGEND

The religious views of Paracelsus influenced his medical theories and these with his philosophical outlook and alchemical disquisitions, inspired the formulation of the Rosicrucian romantic legend, which caused a great stir in certain intellectual circles during the seventeenth to eighteenth centuries. A few words may be said of this offshoot of Paracelsian doctrines, which is relevant to the conflict between Religion and Science.

A protestant teacher, Valentin Andreae (1586-1654) of Herrenberg wrote a small book, published in 1615, relating that a German nobleman—Christian Rosencreutz—had lived in the fifteenth century; had travelled through Europe to Egypt and Arabia and returned with all the profound knowledge of the East. He thereupon obtained the collaboration of twelve unmarried adepts and they retired into a building called the "House of the Holy Ghost", there to meditate and philosophize. Rosencreutz died at the ripe age of 106 years, was interred but it was intended that after one hundred and twenty years his sepulchre should be reopened. His adepts added to their numbers by the admission of suitable candidates.

This little story was implicitly believed, but in 1619 Andreae admitted that it was fiction. This was held to mean that Andreae was not allowed to divulge anything about the Fraternity and the controversy continued more actively than ever. In England the physician Robert Fludd (1574-1637)—who had travelled in Germany—took up the defence of the Rosicrucians against the accusation of magic and dealings with the Devil and gave the first coherent account of Rosicrucianism. However, Fludd was unable to assert where the Fraternity existed, but there is reason to believe that his information was obtained from William Fitzer, who published Harvey's *De motu cordis* (1628) and was an "adept" through one of the De Bry's, into whose family he married. Further ramifications cannot be discussed now, but if the main doctrines of the Rosicrucians are examined, they are found to consist in the study of natural philosophy leading to the revelation of the hidden secrets of Nature, among which were the transmutation of baser metals into gold, the production of an Elixir to prolong human life, together with the practice of medicine for the gratuitous healing of the poor. Johann Thölde, the publisher of the *Triumph Wagen Antimonii* (Leipzig, 1604), was a reputed Secretary of the Order; the monk Basil Valentine, like Rosencreutz, was also a fictitious person.

It has already been said that Paracelsus revealed in his religious beliefs the impact of newly acquired knowledge on orthodox religious dogma; this is again very marked in

Fludd and consequently is noticeable in most Rosicrucian writings, which plead for an explanation of religious mysteries on a scientific basis.

PARACELSIAN DOCTRINES HISTORICALLY CONSIDERED

An appreciation of the historical significance of Paracelsus requires a consideration of his doctrines in relation to orthodox medicine, alkemy-chemistry, Roman Catholic religion, philosophy and natural science; for in all these fields of human endeavour he expressed his views in no uncertain terms. A closer acquaintance with the writings which can be accepted as his, shows that he was violently destructive, only rarely critically constructive and never original, if ever right.

That the observation of Nature was preferable to the acquisition of mere learning had already been propounded by Roger Bacon (1215-92), D'Autrecourt (c. 1346), Cusanus (1401-64), in face of opposition much more serious than Paracelsus ever had to encounter.

Natural philosophy and its successor—modern Science—arose after a more accurate conception of the universe and the laws governing natural phenomena were obtained in the manner propounded by Copernicus, Galileo, Gilbert, Descartes, Francis Bacon.

It can be said that Paracelsus marked the transition between mystical alkemy and constructive, productive chemistry, but he did not contribute to the progress linked with the names of van Helmont, Libavius, Boyle.

In general intellectual outlook, Paracelsus resembled Bernard Palissy (1510-90), who was also keenly interested in technical chemistry, propounded the study of Nature from nature, attempted academical ruition, indulged in polemical sallies against medical dogma and was a fervid religious disputant. When the two are compared, it seems that Palissy was the greater man and deserves to be considered a pioneer of modern natural science; though his medical activities were scanty and not distinguished by therapeutical success.

EPICRITICAL OBSERVATIONS

The myth of the greatness of Paracelsus seems based on the argument *Post hoc, ergo propter hoc*. It can be recalled that the diffusion of printing with movable types was the outstanding event that really produced a reform of classical learning and the revival of natural science. The astounding effects of the printed words and repeated accurate illustration became clearly noticeable in the lifetime of Paracelsus and acquired additional impetus soon after. Thus, to the more fervid admirers of Paracelsus, it seemed that his attack of scholasticism, caused it to fade out. In reality scholasticism persisted in academic circles more than a century after his death; one of its last—and most absurd—exponents, being Guy Patin (1601-72) of Paris University.

One may reasonably ask: Had Paracelsus not lived, what then? The outstanding figures of modern science—Vesalius, Galileo, Gilbert, Harvey—would still have garnered their harvest of actual observations and controlled experimental results.

It is now easy to perceive why and how the practice of medicine in the sixteenth century required reformation; in the first place, it was not the acceptance of ancient texts that was at fault, but rather the blind adherence to scripts containing passages interpolated, muddled and garbled by copyists; one example I can recall is *sugendo* instead of *inungendo*. Then there was the practice of a fatuous uroscopy, apart from bedside observation, which preserved a traditional, mostly ineffective, polypharmacy; then inane pulsology together with senseless bleeding. It is true that the examination of urine, the study of the pulse, several drugs recommended in ancient times, or even blood transfusion, all these are still most usefully employed, but in a manner quite different from that advocated by Paracelsus. Progress in medical matters was induced by men like Nicola Leonicensis (1428-1524) of Ferrara, Thomas Linacre (1460-1524) of Oxford, John Caius (1510-73) of Cambridge, Jean Fernel (1506-88) of Paris, who revised classical texts, encouraged anatomy—which Paracelsus despised—and furthered Hippocratic clinical observation and Galenic therapeutics. Medicine, in all its branches, began to get into its stride when Vesalius dissected, observational and experimental biology was assiduously cultivated by Gesner, Aldrovandi, Harvey,

and Morgagni (1682-1771) combined pathology with anatomy. A great deal of fruitful work was performed apart from Universities, in learned societies all over Europe but in every case by men who had obtained a University training. The type of medical practitioner exemplified by Paracelsus was not connected with any lasting medical improvement. Indeed, from the time of Boerhaave (1668-1738)—who enforced the teaching of medicine at the bedside, it was the irregular practitioner of healing that continued the practice of uroscopy, astrology, polypharmacy by correspondence or at a distance from the patient.

Therefore, it cannot be said that the abusive rantings of Paracelsus contributed to the general progress of science and medicine that began in the sixteenth century, principally as the outcome of the diffusion of accurate knowledge by means of printed books. For he was a rude, circuitous obscurantist, not a harbinger of light, knowledge and progress.

Section of Physical Medicine

President—MAJOR G. D. KERSLEY, R.A.M.C.

[October 4, 1941]

MEETING HELD AT OXFORD

A Demonstration of the Study of Sweat Secretion by the Quinizarin Method.—L. GUTTMANN, M.D. (Freiburg).

In recent years it has become evident that a study of sweat secretion is valuable as a diagnostic method. Details of the sudomotor nerve fibre pathways both in the central and peripheral nervous system can be demonstrated, and purely local physiological activity of the sweat glands estimated. In addition, pathological changes in the nervous system which involve the sudomotor pathways and local damage to the sweat glands themselves can be studied. The method is objective and does not depend like sensory tests upon statements made by the patient. Dye tests: in particular the quinizarin method, have proved the most satisfactory.

The sodium salt of quinizarin (2-6-disulphonic acid) is the colour indicator. It is a red-brown dye and a derivative of anthraquinone similar to alizarin, purpurin and anthraruphin all of which are used in the dye industry. For application to the human skin the quinizarin must be mixed with sodium carbonate in order that the colour change with moisture shall take place whatever the pH of the sweat secretion: rice starch is also added to economize in the amount of dye that has to be used. The following mixture is most suitable:

| | |
|---------------------------------|----------|
| Quinizarin 2-6-disulphonic acid | 35 g. |
| Sodium carbonate (powdered) ... | 30 g. |
| Rice starch | 60-70 g. |

On occasions it has proved useful to change these proportions, and the powder has even been mixed with olive or paraffin oils (not glycerine) for special investigations. The powder form, however, has proved satisfactory for all clinical purposes. On account of the affinity of quinizarin for water the test powder must be stored in absolutely dry and air-tight containers. When performing a test the necessary quantity of powder should be removed from the container which should be resealed at once. It has been found that a slight change in colour from red-grey to blue-grey which occurs after a time does not interfere with the usefulness of the mixture.

Although the application of the powder to the skin is simple a standardized technique is desirable. If possible the test should be carried out in a room set apart for this purpose, particularly if a sensory test is to be carried out immediately following the sweating test. The powder is dusted over the skin with a pad of cotton-wool and moderate pressure is used to ensure that the orifices of all the sweat ducts become filled with powder. This procedure must be done thoroughly and the powder evenly distributed. Application of the powder with an atomizer was found to be unsatisfactory owing to the lack of pressure. Special care should be taken in powdering the face in order not to evoke tears or sneezing. It should be mentioned that the powder is quite harmless and tests can be carried out repeatedly at short intervals; this is a special advantage of the quinizarin method over other dye tests, for instance the iodine starch method. Sweating is usually induced by heat, in which case one or two cups of hot tea and 5-10 gr. of aspirin are given before the powder is applied. The patient is then placed under a radiant heat cradle for a variable time, usually from fifteen to forty-five minutes, depending on the readiness with which sweating is produced. During this period careful observations should be made of the onset, amount and distribution of sweat secretion. Continuous observation of the patient

during the test is necessary to guard against errors of interpretation due to "running" of the sweat over anhydrotic areas.

A special sweating chamber was demonstrated which has been designed to fit standard hospital beds. It is portable and can be transferred from bed to bed. It is also divisible into two parts and furnished with two windows on either side and two on top, which facilitate observation of the patient from all sides. Each half of the box can be used separately if necessary, both ends of each half being closed by curtains.

When testing the effects of pilocarpine, mecholyl, insulin or other drugs on the secretion of the sweat glands, the drug is injected after powdering.

When the secretion of visible sweat commences the hydrotic areas of skin take on a dark blue-violet colour, and the opening of the individual sweat ducts appear as small dark dots. The anhydrotic areas remain unchanged in colour. The colour change is easy to photograph for record purposes. At the end of the test the dye can be removed with soap and water, if necessary a weak solution of acetic acid or vinegar can be used in addition.

A case was demonstrated of a left lateral popliteal nerve lesion which followed a dislocation of the knee. Photographs indicating the results obtained in other peripheral nerve injuries were also shown. In general the area of sensory loss corresponds to that of the anhydrotic area in complete peripheral nerve interruptions. The dye tests can thus be usefully employed as a guide for sensory tests in peripheral nerve and plexus lesions. Photographs illustrating the successive stages of recovery of sweat secretion compared with that of sensibility during the course of nerve regeneration following nerve sutures and lesions in continuity were demonstrated. Photographs illustrating the recovery of sweat secretion in two skin grafts were also demonstrated. The success of operations involving the removal of portions of the sympathetic nervous system can be exactly controlled by this method and photographs illustrating this were shown. Finally photographs of disturbances of sweat secretion following spinal cord and brain lesions were demonstrated.

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Demonstration of Apparatus for Recording Muscle and Nerve Action Potentials.—

G. WEDDELL, M.B., B.S., and R. E. PATTER, B.A.

A demonstration was given of a method by which muscle action potentials are being recorded photographically.

Concentric needle electrodes similar to those described by Brown (1937) are coupled to the input of a differential amplifier through carefully screened leads, the amplifier output being fed into a cathode ray oscilloscope unit essentially the same as described by Schmitt and Schmitt (1940). The apparatus is sufficiently sensitive to record the action potentials originating in denervated muscles, and the screening is adequate to eliminate all interference from stray fields surrounding the patient in the particular building in which the apparatus is situated (fig. 1).

The machine is being used to investigate the electrical activity of muscles which have been partially or completely deprived of their nerve supply and to follow the changes which occur during the process of re-innervation. The work of Denny-Brown and Pennybacker (1938) has shown that "fibrillation" ceases when a muscle becomes re-innervated. It is hoped that the recording of muscle action potentials at intervals over a period of time from patients with nerve injuries may be helpful in determining the early onset of recovery and may also indicate the pattern of recovery in individual muscles.

An electrode carrier was also shown (fig. 2) which can be used to conduct stimuli to, and pick up action potentials from, nerves exposed during operation. The use of a circuit by which the stimulus may be synchronized with the sweep enables standing waves of nerve action potentials to be obtained and the differential amplifier obviates the need for special screening of the patient. Photographs of nerve and muscle action potentials were shown.

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FIG. 1.

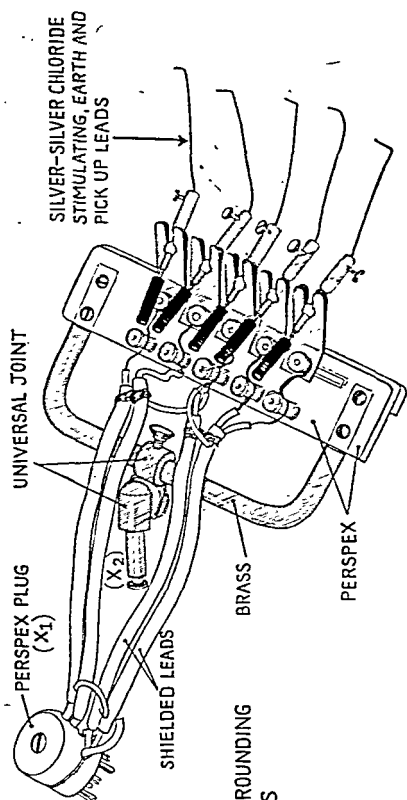
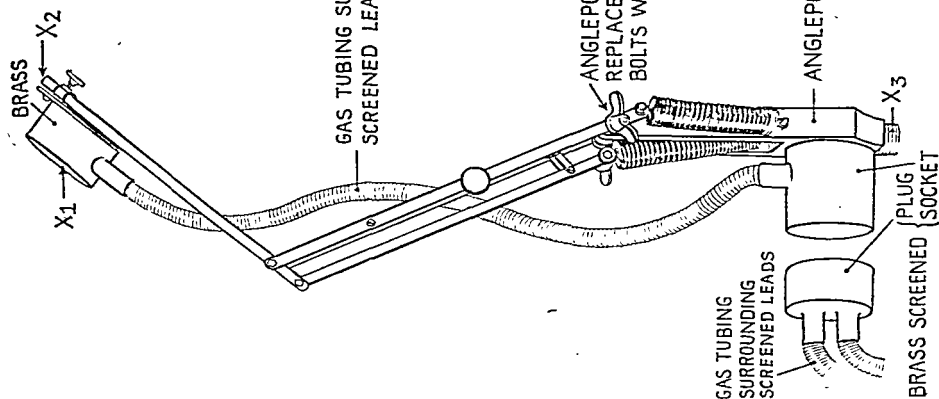


FIG. 1.—Illustrates the manner in which an anglepoise lamp stand has been adapted as an electrode carrier for use either on an operating table or elsewhere. X_1 is the point at which the plug carrying leads from the sterilizable electrode mounting (see fig. 2) is inserted. X_2 is the point at which the electrode mounting is attached (see fig. 2). X_3 is the point at which the electrode carrier is screwed either into the operating table or into the standard anglepoise lamp base.

FIG. 2.—Shows details of the sterilizable electrode mounting.
[The electrode carrier and mounting were designed and executed by Mr. P. Peade of the Department of Anatomy, Oxford.]

Demonstration of Electric Shock Therapy.—Lieut.-Colonel R. W. ARMSTRONG, M.D., D.P.M.

Colonel Armstrong described briefly the history of shock treatment in mental disorders and the various methods which had been used to produce remission or recovery in schizophrenia. The advantages of the electrical method first described by Cerletti and Bini were enumerated, and it was pointed out that this form of treatment was of value not only in schizophrenia but also in affective disorders and in some psychoneuroses.

A portable convulsant apparatus was described and the technique of producing an epileptiform convulsion was first described and then demonstrated on two psychotic patients.

[November 1, 1941]

MEETING HELD AT BATH

Some Orthopaedic Procedures Employed in the Treatment of Arthritis

By JOHN BASTOW, M.D., F.R.C.S.

ABSTRACT.—(1) A series of six cases of ankylosing spondylitis was shown to illustrate the benefit gained, even in very long-standing cases, by the following regime:

- (a) Complete rest in a plaster shell.
- (b) Gradual correction of kyphosis by adding thin layers of padding under the dorso-lumbar junction, and subtracting similar layers of padding from under the head, to increase extension in the spine.
- (c) Deep-breathing exercises practised constantly while the patient is lying in a plaster shell.
- (d) Daily physiotherapy.
- (e) The fitting of a Goldthwaite spinal brace to maintain the position of maximum correction that has been obtained.

The improvement was manifested in all cases by alleviation of symptoms, improvement in posture, and increased chest expansion.

(2) A series of six cases of arthritis of the knee was shown, including rheumatoid arthritis, infective arthritis and osteo-arthritis, illustrating treatment by rest plasters, followed by:

- (a) Arthroscopy and lavage—where swelling and effusion persist, and
 - (b) Bone drilling—where bone changes are marked and there is constant pain at rest.
- Improvement was manifested by relief of symptoms, subsidence of signs of inflammation, and restoration of a considerable degree of movement.

(3) A case of arthritis of the tarsal and metatarsal joints, illustrating relief of symptoms afforded by the application of a plaster boot.

(1) A series of six cases of spondylitis ankylosans was shown to illustrate the routine of treatment carried out at the Royal National Hospital for Rheumatic Diseases, from an orthopaedic point of view.

All cases on admission are supplied with a plaster bed, which extends from the vertex to the sacrum in cases where the disease is confined to the spine itself, and extending to the knees or mid-calves in cases where the hips are affected.

These plaster shells are applied after sedation, with the patient lying prone, and the spine extended as fully as his condition will permit. They are cut away freely behind the shoulders, but extend to the mid-axillary line on either side, below the axilla.

The plaster is then dried and lined with stockinet, and each patient is encouraged to lie supine in his shell for as long as possible, with the aid of regular sedatives, until, in a very short time, he spends the whole of the twenty-four hours in his plaster, except for the period during which he is undergoing physiotherapy. Gradual improvement of posture is obtained:

- (a) By the regular performance of deep-breathing exercises.
- (b) By the insertion of thin strips of woollen material under the spine, wherever the ankylosis is not complete, and especially at the dorsolumbar junction. By this means, a surprising amount of correction of the kyphosis can be obtained within a few weeks, and the chest expansion improves considerably.

Persistent local pain in resistant cases is treated by applications of deep X-ray therapy, when ordinary physiotherapy fails to bring relief.

When it is judged that the maximum degree of correction in a particular case has been obtained, the patient is fitted with a Goldthwaite spinal brace, and allowed up for gradually increasing periods. He spends the night in his plaster shell, and takes it home with him; with instructions to continue its use until long after all symptoms have disappeared, and to return to its use at the first sign of recurrence of pain or increase in deformity.

The following table illustrates the cases shown at the meeting:

CASES OF SPONDYLITIS ANKYLOSANS.

| Name (Males) | Onset | Previous health | Previous treatment | Condition on admission | Treatment and duration | Suspension stability (corrected) | | Present condition |
|----------------------|---------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------|----------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------|-------------------------------------|--------------|---------------------------------------|
| | | | | | | Hamatocrit Per cent. | Per cent. | |
| (1) J. 36 yrs. | 3 mths. very sudden. Pain in neck, hips, shoulders and lumbar region within a week | Rheumatic fever, 1932. Pneumonia, 1934. Appendix removed and operation for adhesions in 1939 | None. Rest in bed. | Constant acute pain. Emaciated. Chest expansion 1½ in. Rise temp. K.J. + + ; A.J. + + | Plaster shell. Deep breathing. Goldthwaite brace. Massage. Short wave diathermy. Cod-liver oil. Fersolate. 8 weeks. | 41 now 43 | 58 now 58 | Much improved. Chest expansion 2½ in. |
| (2) E. G. 47 yrs. | 10 mths. gradual, 4 mths. acute | Severe burn foot 1934-7. Rectal polyp removed 1941 | None | Great loss of weight. Stiffness in rising and bending. Chest expansion 1 in. K.J. + ; A.J. + | Plaster shell. Deep breathing. Goldthwaite brace. Fersolate. 4 weeks | 42 | 67 | Improving. Chest expansion 2 in. |
| (3) L. G. 25 yrs. | 2 yrs. gradual, 5 mths. acute | Rheumatic fever 1934. No other illnesses; played Rugby till onset of stiffness. | None. Some rest in bed | Very ill on admission. Rise temp. 3 wks. Emaciated. Pain acute. Chest expansion ½ in. K.J. + + ; A.J. + + | Plaster shell. Deep breathing. Goldthwaite brace. Short-wave diathermy. Adexolin. Fersolate. 7 weeks | 37 now 44 | 73 now 63 | Much improved. Chest expansion 1½ in. |
| (4) S. 30 yrs. | 3 yrs. gradual, 2 yrs. acute extending to neck | Pyorrhœa. Appendix removed 6 mths. ago | Teeth extracted 1939. Deep X-ray. Gold injections. Blood transfusion | Very acute pain. Spasm of hips. Chest exp. ½ in. K.J. + ; A.J. + | Plaster shell. Deep breathing. Goldthwaite brace. Deep X-ray. Adexolin. Fersolate. 3 weeks | 37 | 81 | Improving. Chest expansion 1½ in. |
| (5) D. P. 34 yrs. | 18 mths. gradual, 6 mths. acute | Good | Massage. Infra-red rays | Constant aching pain—lumbar. Loss of weight. Chest expansion 2 in. K.J. + ; A.J. + | Plaster shell. Deep breathing. Deep X-ray. Fersolate. Adexolin. 6 weeks | 44 now 41 | 64 now 65 | Much improved. Chest expansion 3½ in. |
| (6) G. B. 52 yrs. | 5 yrs. gradual, 2 yrs. acute | Good. Diarrhœa severe at times | None, except medicine for diarrhœa | Loss weight. Chest expansion 1½ in. Stiffness | Plaster shell. Goldthwaite brace. 4 weeks | 42 | 61 | Much improved. Chest expansion 2½ in. |

X-rays confirm diagnosis in all cases.

(2) *Treatment of arthritis of the knees by (a) Arthrotomy and joint lavage. (b) Bone-drilling.*

A series of six cases of arthritis of the knee was shown, illustrating the type of case suitable for surgical treatment.

(a) In certain cases of arthritis of the knees, whether rheumatoid arthritis, infective arthritis or osteo-arthritis, signs and symptoms of inflammation persist in spite of rest. In this hospital practically all cases are supplied with rest plasters on admission, and these are worn continuously, only being removed once daily for physiotherapy or hydrotherapy to prevent stiffness.

Such cases, where swelling of the joint is a prominent feature, benefit considerably by the operation of arthrotomy and joint lavage. The procedure is as follows:

A general anæsthetic is employed in all cases, and after the application of a tourniquet, a 2 in. incision is made over the suprapatellar pouch, on either the inner or the outer side, and the quadriceps muscle is split in the line of its fibres. The capsule of the joint is incised, the contents of the joint are expressed, and the synovia inspected.

In a large proportion of the cases the joint is almost completely filled with large masses of solid fibrinous exudate of the consistency of the white of a poached egg, which it is obviously quite impossible to remove from the joint by aspiration.

Occasionally this fibrinous exudate is adherent to the surface of the synovial membrane, but is easily separated by gentle dissection with gauze moistened with saline. Enlarged and congested synovial fringes are usually present at the articular margins, but it is only in a few cases that these are of sufficient bulk to account for any degree of joint swelling in themselves.

The joint is next gently washed out, using a soft rubber catheter, with a dilute solution of warm eusol, and is flexed and extended to ensure that all compartments of the joint are thoroughly irrigated. The small wound is then closed in layers, and the rest plaster reapplied and fixed to the leg by means of a pressure bandage over wool.

Within twenty-four to forty-eight hours the relief of symptoms is very noticeable. The patient is instructed to practise quadriceps exercises from the second day, but the knee is not taken out of the plaster until the sutures are removed on the tenth day, after which physiotherapy and active exercises are resumed.

It is remarkable how quickly heat and muscle spasm subside after this operation, although in some cases a serous effusion recurs, which occasionally requires aspiration, but soon subsides when faradism is applied to the quadriceps. No patient is allowed to resume weight-bearing until all signs of active inflammation in the joint have subsided.

The following table illustrates the cases shown at the meeting:

CASES OF LAVAGE—KNEE-JOINTS.

| Name | Date of admission | Onset | Condition on admission | Treatment | Present condition |
|--------------------------|-------------------|-----------|---------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------|
| (1) T., male, 64 yrs. | 10.10.41 | 2 years | Redness, pain, heat, spasm, fluid ++. Hæmatocrit 41%. Suspension stability (corrected) 62 % | Rest plasters. Awaiting operation. | |
| (2) S., male, 47 yrs. | 5.2.41 | 13 months | R. knee, pain, heat, some spasm. L. knee affected later. Hæm. 33%. S.S. 66% | Rest plasters. 5.3.41: Operation R. knee. Synovial membranes injected, cedematous; early erosion; turbid yellow fluid. 19.4.41: Operation, L. knee. Synovial membranes injected and congested, fibrinous exudate adherent to synovia. No gross erosion. Slight pannus formation. 12.8.41: Myocresin. Total 97 g. 18.9.41: Lavage repeated. | "Itching to get up." R. knee moves 180-60 degrees. L. knee moves 174-96 degrees |

CASES OF LAVAGE—KNEE-JOINTS (*contd.*).

| Name | Date of admission | Onset | Condition on admission | Treatment | Present condition |
|-------------------------------------|-------------------|------------------------------------------|------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------|
| (3) A. W., female, married, 54 yrs. | 31.7.41 | 10 years | Both knees, especially left, heat and fluid: Ankles, toes, L. elbow and shoulder affected. Hæm. 29%. S.S. 64% | Rest plaster. 16.9.41 : Operation, both knees distended, semi-solid material, much fibrinous exudate. 25.10.41 : Fluid accumulated L. knee, aspiration 100 c.c.—very sticky yellow fluid. Rest plasters. | Much relief. R. knee moves 160–78 degrees. L. knee moves 180–90 degrees |
| (4) S.E., male, 23 yrs. | 10.9.41 | 12 years with remissions till March 1941 | Heat, acute pain, swelling. Limited movement, flexion spasm. Previous benefit from streptococcus vaccine. Hæm. 35%. S.S. 66% | 22.10.41 : Operation, both knees. Extreme gelatinous œdema. No gross erosion of the articular cartilage | Very comfortable. R. knee moves 180–110 degrees. L. knee moves 164–108 degrees |
| (5) G. D., female, married, 45 yrs. | 18.9.41 | 7 years with remissions | Pain, heat both knees, especially left. Wrists and finger-joints also affected. Hæm. 36%. S.S. 86% | Rest plasters. 21.10.41 : Operation, left knee. Oily fluid, slight fibrinous exudate. Adhesions numerous between suprapatellar pouch and upper patella. Much synovial œdema. Synovia œdematous and pale. Early marginal erosion of joint cartilage. | Feels very well. L. knee moves 180–148 degrees. |

Hæm. = Hæmatocrit. S.S. = Suspension stability.

(b) In certain cases where changes of osteo-arthritis have supervened, the prominent feature is not effusion but constant pain in the joint, that is not relieved by rest and persists during the night. In such cases, considerable relief is obtained by the operation of bone-boring.

This consists simply of drilling a hole through the cortex of the upper end of the tibia and of the lower end of the femur, with a $\frac{1}{4}$ in. drill, and of passing a Steinman's pin through the hole in the cortex into the cancellous bone in several directions, so that all areas of the condyles are tapped.

The skin is then closed, and the limb put back into a rest plaster until the sutures are removed. Weight-bearing is again not allowed until all inflammatory signs have subsided.

There is usually complete relief of pain within twenty-four to forty-eight hours, and the patients are in all cases extremely grateful. An illustrative case appears below:

CASE OF BONE BORING.

| Name | Date of admission | Onset | Condition on admission | Treatment | Present condition |
|---------------------|-------------------|-------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------|
| (1) Mrs. H. 66 yrs. | 9.9.41 | Sudden. Of 2½ years' duration | Acute pain at night in knees. Shoulders, hands, wrists, metacarpophalangeal joints, and ankles also affected. Hæmatocrit 33%. S.S. (corrected) 61% | Rest plasters, hands and legs. Light general massage. Infra-red rays to knees. 25.10.41 : Operation. Bone-drilling, R. tibia, and lower femur. Lavage of joint, which was disorganized by adhesions and erosion of articular surfaces. | No pain from second night after operation. R. knee moves from 180–154 degrees |

(3) Cases of arthritis of the foot, in which pain and swelling of the tarsal joints and plantar fascia persist in spite of rest and physiotherapy, are often found to respond well to the application of a plaster boot.

Deformities, such as inversion of the forefoot, dropping of the metatarsal heads, or a valgus position of the heel, are corrected as far as possible by manipulation under an anæsthetic, and a plaster boot is then applied, with the arches well moulded up and the plaster extending from beyond the toes to just below the knee.

The patient is rested in bed for twenty-four to forty-eight hours, and then encouraged

to walk in the boot, which is worn from four to eight weeks, depending on the severity of the case.

Considerable relief from pain is obtained by the wearing of such a plaster, and after its removal an arch support is fitted and physiotherapy instituted.

An illustrative case was shown at the meeting.

I am much indebted to Mr. C. Kindersley, F.R.C.S., Surgeon to the Hospital, who is in charge of the Orthopaedic Department, and is responsible for the routine followed in the treatment of the cases of spondylitis, for permission to show these cases. My thanks are also due to Dr. Ross for preparing the résumé of the cases, and to Sister Geere, who is responsible for the application of the plaster splints.

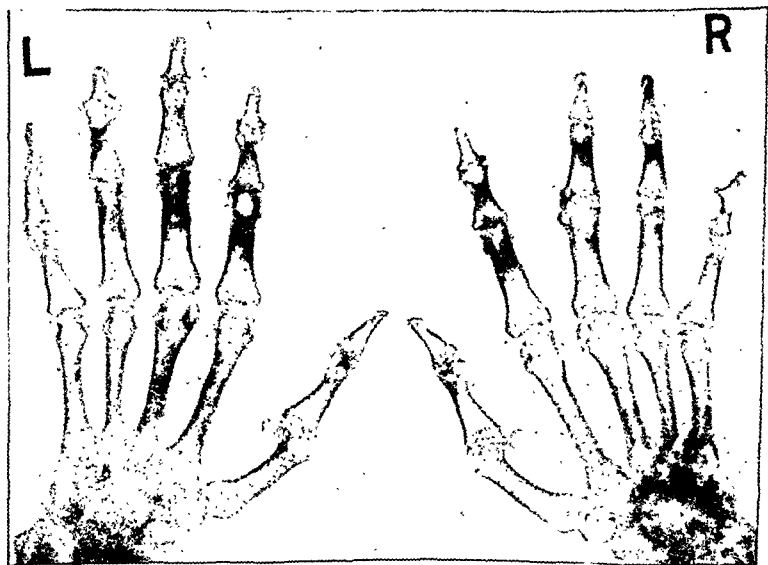
THREE CASES BY J. BARNES BURL, M.D.

(1) Tophaceous Gout.

Collier. Aged 46.

Points of special interest. (a) Good example of light areas in bone. (b) Variations of sedimentation rate. (c) Three tablets of atquinol ($7\frac{1}{2}$ gr.) per diem for five consecutive months with beneficial results.

Previous history.—First attack of gout 1926 following dental extraction with bleeding from gums, sudden onset of severe pain in the knees, elbows, hands and feet (not big toes). Off work eight months; complete recovery after the attack.



CASE 1.—Tophaceous gout.

Subsequent attacks every year since, usually commencing in February, onset sudden, duration some weeks. After attacks his hands are weak, so that he has not worked for seven years. Rest of joints recover completely. On some occasions hands have discharged white matter.

Joints now affected: Shoulders, elbows, wrists, hands and fingers, knees, ankles. Tophi in the ears.

Sedimentation rate 1937 (during acute attack) 50% in one hour; (six weeks later) 92% in one hour. 1938 (during acute attack) 58% in one hour.

Urinary calculus passed by the patient consisted of pure uric acid.

Discussion.—Typical case of tophaceous gout with characteristic variation of the sedimentation rate during and between attacks.

X-rays: Light areas in knees, punched-out areas in hands and big toes. (See fig.)

Blood uric acid: 1937, 5.7 mg. per 100 c.c.; 1938, 6.2 mg. per 100 c.c.; 1941, 5.7 mg. per 100 c.c.

Has taken atquinol tablets, three per day for five months, with excellent results.

For one year after leaving hospital patient kept well.

Readmission (7.10.41).—Severe attacks since January 1941. Urinary calculi passed September 1941, with severe hæmaturia. Blood uric acid 5.7 mg. per 100 c.c. Hæmatocrit 40%. Corrected suspension stability 73%.

(2) Large Cyst Formation directly associated with Rheumatoid Arthritis.

P. M., hotel manageress, aged 31.

Points of special interest.—(a) Circular cysts the size of half a crown have developed just above the right and left acetabulum; both hips show serious damage of the cartilage, together with small cyst formation.



CASE 2.—Large cyst formation directly associated with rheumatoid arthritis.

(b) During a period of nine years this patient has had four courses of gold. After each course the patient improved markedly and was able to return to work.

Onset, gradual, hands, elbows, knees, ankles.

Admission 1932, 1933, 1934, 1935, 1937 (returned to work each time), and has been working up till admission August 1941.

Treatment.—Four courses gold. Sedimentation rate improved by 20%.

On readmission (August 1941).—Condition much worse. Pain acute in hips, knees, wrists. Had finished fourth course gold May 1941. New radiographs show large cysts above the hip-joints. (See fig.)

The X-rays taken in 1935 show a cystic area commencing to form above the left hip. No obvious abnormality above the right hip-joint. Blood calcium and phosphatase normal.

to walk in the boot, which is worn from four to eight weeks, depending on the severity of the case.

Considerable relief from pain is obtained by the wearing of such a plaster, and after its removal an arch support is fitted and physiotherapy instituted.

An illustrative case was shown at the meeting.

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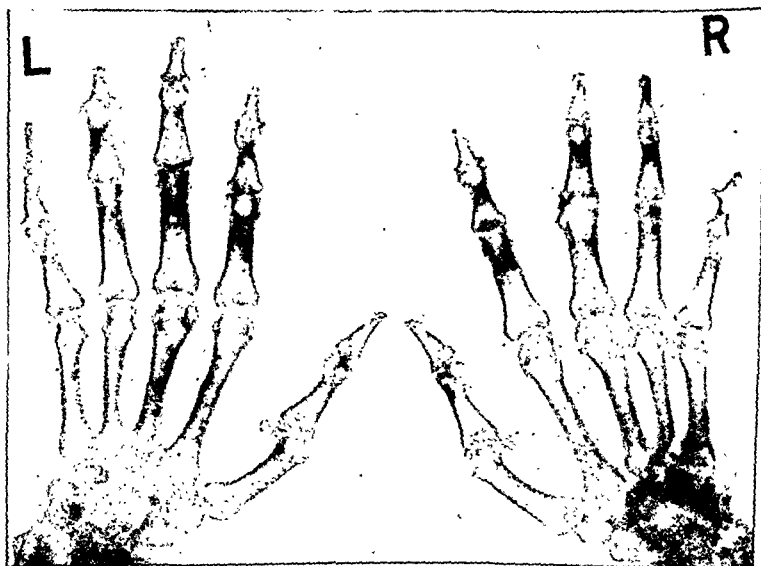
THREE CASES BY J. BARNES BURT, M.D.

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Urinary calculus passed by the patient consisted of pure uric acid.

Section of Surgery

President—E. ROCK CARLING, F.R.C.S.

[November 5, 1941]

DISCUSSION ON STREPTOCOCCAL CROSS-INFECTION IN WARDS

Professor A. A. Miles: The natural reservoir of *Strep. pyogenes* is the upper respiratory tract of man, and in cross-infection of surgical patients we have to consider mainly the upper respiratory tract and wounds. The infection of the respiratory tract is not primarily a surgical problem, but its existence in patients or ward staff must greatly increase the risk of wound infection unless proper precautions are taken.

War-wounded tissue is obviously more susceptible to infection than the deliberately made operation wound, especially where, as in burns, there is extensive destruction of the skin; and the admission to wards of large numbers of primarily infected war-wounds results in a menace to the uninfected wound that is absent in peace-time. Thus, without postulating any deterioration of the prophylactic techniques, hospital infection may leap into war-time prominence. The change has taken place in the risk, and the peace-time technique has been inadequate to meet it.

The streptococcal menace to clean wounds.—In actual ward conditions, we may summarize the streptococcal menace to the clean wound diagrammatically (fig. 1). The "clean wound" in the figure should more exactly be called the "non-streptococcal wound", since many healthy war wounds in their early stages contain a variety of more or less saprophytic bacteria. It is subject to contamination from two main reservoirs. The more natural reservoir is the upper respiratory tract of man. Various observers find from 2-20% of persons carry hemolytic streptococci in the nose or throat (War Memo. No. 6, 1941). Cross-infection in a community of nurses, patients and doctors is probably constant, but only in a small proportion is the infection accompanied by warning signs of sore throat, or "cold in the head". The majority of carriers are infected without any sign; nevertheless, they may be as prolific a source of *Strep. pyogenes* as a person with acute tonsillitis. The streptococci in the upper respiratory tract are showered in liquid droplets, as finer droplets that rapidly dry into streptococcal dust (the droplet nuclei), and leave the upper respiratory tract on the hands and handkerchiefs of the carriers. Sometimes there is an intermediate self-infection of a cut finger or a badly manicured nail: the hand ceases to be an entrepreneur and goes into production as a manufacturer of streptococci.

(3) Still's Disease.

G. B., aged 9.

Points of special interest.—(a) Illustrates the value of gold treatment in Still's disease. (b) Subluxation of the right hip due to weakness of muscles and complete restoration within twenty days by weight extension.

On admission.—Swelling, heat, redness of fingers, wrists, elbows, ankles, knees. Subluxation right hip (recovered after weight extension).

Treatment.—Rest, plaster rest splints. Myocrisin 0.01 g. Total 0.07.

Readmission (21.10.41).—Great improvement. Walking. No pain except slight in right knee. Tendency eversion right foot.

Hæmatocrit 38%, formerly 33%.

Suspension stability (corrected) 62%, formerly 65%.

Pathological Demonstrations.—H. J. GINSON, M.D., D.P.H.

A. Microscopic preparations.—(1) Large subcutaneous nodule from a case of rheumatoid arthritis showing the characteristic histological features. (2) Synovial villi from knee-joint in rheumatoid arthritis showing perivascular lymphocyte aggregations. (3) Urate crystals expressed from gouty tophus. (4) Large subcutaneous nodule from a case of gout. (5) Small cutaneous tophus showing the cytological features of gout.

B. Naked-eye specimens mounted in glycerin gelatin to retain natural colours. (1) A series of four knee-joints illustrating progressive rheumatoid changes from early marginal erosion of cartilage to complete fibrous ankylosis. (2) A series of preparations of tissue from cases of gout illustrating tophi, a subcutaneous nodule and a toe in which the terminal phalanx was completely replaced by uratic material.

C. The "Spa Hospitals Method" of estimating the blood sedimentation rate (as described by Collins, D. H., Gibson, H. J., Race, J., and Salt, H. B., 1939, *Annals of Rheumatic Disease*, 1, 333).

The methods used for estimating the rate and for charting the results of serial tests on the same patient were shown, together with details of the technique of correction for variations in packed red cell volume as estimated by the hæmatocrit tube.

never touches the patient, or any soiled bandages or dressings removed from him, or his bed. (b) A dresser and a part- or whole-time assistant. Their chief concern, from the infection point of view, is to treat each bed as a separate unit; all possibly infected material is carefully disposed in special buckets; everything they have handled during the dressing is sterilized; and the two workers wash before going to the next dressing. The dresser may use a "no-touch" technique, with dry hands, or dress the wound in-dry, sterile rubber gloves; but dressing with "scrubbed up", wet hands should be avoided.

The efficacy of precautions.—Dr. Joyce Wright and I assisted Mr. McKissock in his attack on hospital infection in a neurosurgical unit. Our streptococcal watch established that the incidence of *Strep. pyogenes* in wounds, throats and air was similar throughout an eight months' period of observation. For instance, there were 15 streptococcal wounds, either infected before admission, or hospital infected, in both periods; and at one time or another, about 10% of the patients and personnel in the wards were in each period carriers of *Strep. pyogenes*. Four months constituted a control period, and a second four months the period when improvements in nursing and dressing techniques were tried.

In the first period, 3 air-raid casualties were admitted with infection already established; 32 air-raid casualties were free from *Strep. pyogenes*, but 10 became infected during treatment. At the same time 2 of 46 "clean" operation wounds became infected; making the total hospital infection rate 15.4%. The revision of technique included institution of a quiet period before and during dressing; compulsory masking of dressing team; establishment of a dressing team of four, with carefully defined duties, a rigid separation of the "clean" nurse and her dressing trolley from the dresser and the "dirty" nurse, and insistence on a thoroughgoing "no touch" technique; segregation of bed linen, &c., of infected cases; and the sterilization after use of communal baths and wash-bowls.

In the second four-month period, 11 persons were admitted with already infected wounds; of 46 head wounds free from *Strep. pyogenes* on admission, only one became infected, and all of 49 "clean" operation wounds healed by first intention. Thus, in spite of the prevalence of *Strep. pyogenes* in the second period, the revision of dressing technique was followed by a reduction of the total hospital infection rate from 15.4% to 1.1% (McKissock *et al.*, 1941).

The results are encouraging; but they were achieved by a blunderbuss attack on a very complex problem. Every practicable loophole was stopped, and it is impossible to do more than guess the relative value of the improvements that were made. We need many more bacteriological surveys of ward technique and its results, not only to test the efficacy of different prophylactic measures (and incidentally justify much current aseptic and antiseptic dogma) but also to establish unequivocally that hospital infection is a problem demanding solution, even in wards and hospitals where its effects are not obvious.

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 Medical Research Council War Memorandum No. 6 (1941), "The Prevention of Hospital Infection of Wounds". London.

Professor J. Paterson Ross: The foundations upon which a proper method of dressing wounds in wards must be based may be stated briefly as follows:

(1) *Precautions against contamination by dust and droplets.*—These are placed first not because they are the most important, but because they come first in the course of carrying out a dressing.

Since it has been clearly proved that draughts and dust-raising activities in the ward increase the number of organisms in the air, bedmaking and sweeping must be completed at least one hour before dressings are done, and windows should be closed. Doors should also be closed and traffic through the ward reduced to a minimum while dressings are in progress.

The other main reservoir, and probably the most fruitful source of hospital infection, is the streptococcal wound. In attending a patient with such a wound, the disturbance of infected dressings, pillow-cases, sheets and nightgowns showers streptococcal dust into the air. This takes place both from material that is visibly soiled with discharge, and from apparently unsoiled stuff. Fine flakes of dry streptococcal exudate are carried by hands, clothing and instruments to other patients, or are handed from one patient to another on books and newspapers. Ward utensils that are in common use may carry the cocci. For example, the ordinary wash-bowl is infected when the patient washes, though the wound itself is not washed at the time. Handling infected wounds, dressings and bandages is dangerous for the nurse or surgeon with abrasions or cuts of the hand or forearm; and indeed, it is probable that the hand is more often infected in this way than it is from the upper respiratory tract.

Finally, droplet nuclei and infected dust particles may be deposited from the air directly on to the wounded tissues

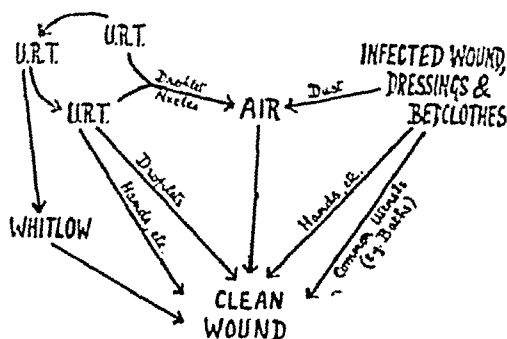


FIG. 1.—Sources and vehicles of *Strep. pyogenes*, and their relation to the uninfected wound. U.R.T. = Upper respiratory tract.

Precautions.—Prophylactic measures are conveniently divided into those indirectly concerned with dressing and treatment of the wound, and those which are incorporated in the technique of dressing the wound. The danger to wounds of infected droplets from nasal and throat carriers is minimized by the wearing of proper masks—a procedure which should help to lower the incidence of upper respiratory tract cross-infection as well. The danger from infected hands is removed by the exclusion of their owners from the ward. Measures to keep down and remove dust, and proper covering of the wound meet the menace from the air. Common utensils, like wash-bowls, are disinfected after use by each patient, and there should be careful segregation of patients' own towels, combs, face-flannels, bath-blankets and bedclothes.

The dressing technique.—The most effective prophylaxis, which is in force for the greater part of the patient's day, is complete covering of the wound. The fully dressed wound is largely impervious to infected particles, however they are transported. The measures taken to prevent hospital infection thus centre on manipulations of the wound when the protective dressing is removed.

With a large expenditure of time, a large supply of sterile instruments and towels, and a good deal of hand washing, one person can dress a moderately complicated wound with safety. But the safety is more certain, and the efficiency greatly increased, if dressings are done by teams in which each member has his own well-defined duties. For one thing, the wound is exposed for a shorter time when dressed by a team, and the risk of air-borne contamination thereby minimized.

The team should consist of (a) a trolley assistant who supplies the sterile and clean materials to the dresser. All sterile material is handed by forceps, and the assistant

operative technique, hæmatoma formation, stitch abscesses and the like, and failure due to cross-infection. I think I may say, though I regret without being able to give statistics, that within the past year there has been an improvement since adopting a stricter régime in my own wards; certainly infections like that with *B. pyocyaneus* which used to spread widely in a ward now occur but rarely and in isolated cases.

I would add, however, that even without the support of statistical evidence we should be prepared to adopt these improvements in our ward technique. Surely nobody can deny that the suggestions are sound and practical, and since there is no difficulty in carrying them into effect I can see no objection to doing so.

The necessity for scrupulous attention to detail is not so apparent with clean sutured operation wounds as it is with traumatic wounds. Even though it may be difficult to infect a post-operative wound, however carelessly it is dressed, this is no excuse for failing to employ what is considered to be a good technique. It is unreasonable to use a good method in one case and a bad one in another—and disasters are not unknown even with the "safest" wounds.

The same considerations apply to dressing wounds at first-aid posts and in the out-patient departments of hospitals—really a much bigger problem and much harder to deal with than ward-dressings—and also to dressing wounds which are already grossly contaminated. It may be suggested that if a wound is to be cleansed by surgical excision there is no need to worry about asepsis when inspecting it beforehand; yet there is no reason to add to the work the surgeon has to do, and there is no guarantee that he will in fact rid the wound of all its organisms by excising it. No wound is so grossly contaminated that it cannot be made worse by additional infection, which may be the last straw in breaking the patient's resistance. There is a serious risk of droplet or contact infection of a wound if it is carelessly "glanced at" during a ward round, or if the dressing is removed without proper precautions, for example before an X-ray examination; the very fact that these manipulations are counted as trivial and not as "proper dressings" makes them all the more dangerous. To achieve success in surgery conscientious attention to detail is essential. Ideals may not be for capture, but we must not grow weary in their pursuit.

It may be argued that these suggestions are all very well in a modern hospital ward with plenty of staff and equipment, but are not generally applicable to conditions all over the country. In point of fact, if suitable adjustments are made in detail to meet local requirements, the essentials of these improved methods of dressing wounds can be put into practice in any hospital, however old-fashioned, in a private house, or in a cottage. Large dressings may have to be done in an operation theatre, using full theatre technique: but the usual ward dressings are done by methods which resemble more closely those of the bacteriologist handling cultures with sterile instruments in "dirty" surroundings and yet avoiding contamination. We look forward to the time when dressings will no longer be done in the ward but in a special room off it especially equipped for the purpose.

The number of assistants employed in doing a dressing will depend on the surgeon, the number of dressings to be done, the number of assistants available, and the team work. When several dressings are to be done they can be got through more efficiently by three persons working together. There should be an adequate supply of the simplest dressing instruments, but this need not mean a large number, as instruments can be re-sterilized in a few minutes. The traditional boiling for twenty minutes is unnecessary and tends to spoil the instruments; clean instruments may be sterilized by boiling for two minutes.

There has been a tendency in the past to dress wounds too often, and this has led some surgeons to see in the "closed plaster technique" the solution of all our problems of cross-infection. But organisms can get in and out of plaster—no efficient method of sealing them has yet been discovered; furthermore, this method carries with it an enormous risk of disseminating infection, as well as adding infection to the wound, when the plaster is changed, unless specially well-thought-out precautions are rigorously enforced.

Surgeons are greatly indebted to the bacteriologists who have demonstrated the sources and channels of hospital infection and have indicated how they may be blocked. The sug-

Organisms in dust are constantly settling down out of the air, and sterile dishes containing instruments, gauze and swabs should all have lids. The buckets provided for soiled dressings should also have lids to prevent dust rising into the air from them. Wounds are to be exposed for only the minimum time required for the necessary examination of and attention to the wound; if there is ever any unavoidable delay in the course of the dressing the wound must be covered by a sterile towel.

The dresser and all those in attendance at the dressing, for instance nurses or students assisting or watching the dresser, must wear masks which are known to be impervious to droplets—this means that they must have a sheet of paper or some other impervious material placed in between the layers of the mask itself. Masks must also be long enough to cover the nose and come well below the chin, and broad enough to prevent droplets escaping at the sides—6 by 8 in. is sufficient.

(2) *Precautions against contact infection.*—The most important sources of infection are other wounds, and throats and noses; and the most important method of spread is by contact—either the dresser's or the patient's fingers, or else contaminated utensils or dressing materials conveying the infection to the wound.

Dressings should be changed entirely with instruments, and fingers must never come in contact with either the wound, the dressing on it, or the skin around it. It is rarely necessary to palpate the skin near a wound, and when this has to be done, sterile gloves must be worn. Some big dressings are better done throughout with gloves—in fact, it is best to do the dressing in the operation theatre with full "theatre technique", which is essentially different from the "ward technique".

Case-to-case infection is avoided either by having a separate packet of sterile dressings and a fresh set of sterile instruments for every case; or else, if a number of dressings are to be done in succession, a clean nurse must pick out instruments and dressings from the stock on the trolley and hand them in her sterile forceps to the dresser who, although his own instruments are also sterile to begin with, is not allowed to handle directly any sterile material on the dressing trolley, as his instruments may have become infected in the course of the dressing.

To avoid infected trickles of water or lotion running off hands or instruments into the wound it is essential that both hands and instruments should be kept dry.

(3) *Methods of sterilization.*—It is most important to review carefully the methods adopted for sterilizing instruments and all materials used in doing dressings. Instruments, even cutting instruments, should be boiled. Spirit is an unsatisfactory disinfectant. When bowls and irrigating cans are being boiled they should be completely immersed in the water; so often one sees them only half-covered. "Sterile water" kept in wards for rinsing instruments and other materials which have been kept in an antiseptic solution is often far from sterile. The antiseptic solutions themselves must be checked to make sure that they are strong enough and that the instruments remain in the solution long enough to ensure safety. The disinfection of baths, and the disposal of their infected contents so as to avoid further spread of infection, require constant attention.

Discussion of practical details.—We are sometimes asked whether these new and elaborate methods are really necessary and effective. In the first place, they are neither new nor elaborate. Before the war there had been a great decline in the standard of aseptic technique, and what is now advocated is really more a revival of a proper interest in asepsis than anything new; furthermore, there is nothing elaborate in shutting windows, wearing masks, and boiling instruments. Certain ideal methods such as the oiling of floors and blankets are admittedly more elaborate, but although they are highly desirable they are not essential.

The necessity for an improvement in our methods is shown by bacteriological demonstration that cross-infection is occurring very frequently under present-day conditions. Good evidence of the efficacy of such an improved technique in dressing head wounds has come from Mr. McKissock and Professor Miles, and we should try to obtain similar evidence from other departments. When doing this it is important to distinguish between the failure of a wound to heal by first intention which may be due to faulty

were not directed to discovering whether reduction in the numbers of air-borne streptococci influenced the incidence of hospital infections, but simply to determining the efficacy of experimental methods of dust-laying.

As regards the reduction of the numbers of bacteria in ward air, all that can be done at present is to give an outline of those methods which have proved satisfactory in laboratory and small-scale ward trials. The determination of their efficacy has depended entirely on counts of β -haemolytic streptococci or of total organisms recovered from the air. The estimation of the numbers of air-borne organisms has been simplified by the use of the slit sampler designed by Bourdillon, Lidwell and Thomas (1941). The sampler collects, with an efficiency of 90% or more, the bacteria from any required volume of air (usually 2-10 cu. ft.) on to solid nutrient media in Petri dishes. For the collection of β -haemolytic streptococci, the use of 1:500,000 gentian violet blood agar (Garrod, 1933) is especially useful, as it inhibits the growth of the majority of air-borne saprophytes without significantly reducing the number of haemolytic streptococci.

Methods of reducing the number of air-borne organisms.—Masks: Hare (1940, 1941) has shown that β -haemolytic streptococci are disseminated by throat carriers in large droplets which settle rapidly and therefore have a short infective range. These large droplets are probably of very great importance in the spread of infection. Not only do they carry large infective doses, but droplets may settle on the bedclothes near the patients' heads, where they dry and from where they may be redistributed into the air when the bed is made. Bourdillon and Lidwell (1941) have shown experimentally that during a single sneeze many thousands of bacteria-laden particles are distributed into the air. The vast majority of these are contained in the larger particles which settle to the floor in a few minutes. The simplest and most effective method of preventing the dissemination of large droplets is masking of the potentially infective persons. Masks of impervious material such as cellulose acetate have been found very suitable. A variety of patterns of mask have been studied. Masks of simple design can be made to suit most purposes. The majority of droplets are collected by a simple mask consisting of a single sheet of cellulose acetate mounted on a wire frame, while a mask of the "box" type will prevent the downward distribution of droplets as well, and could be worn by dressers. It is not suggested that these are the only masks which are satisfactory. Many find gauze masks containing a cellophane sheet more comfortable. They can be as effective in preventing the dissemination of infective droplets, providing that they are impervious. Masks consisting simply of one or two layers of gauze are notoriously inefficient.

DUST-LAYING

It has been known for a considerable time that the greatest bacterial contamination of ward air occurs during times of greatest activity in the ward, and especially during bedmaking and sweeping, when most dust is disturbed. That the degree of infection of ward dust may be considerable has been shown by an analysis of the dust in an ear, nose and throat ward in which some of the experiments to be mentioned in this discussion were performed. One morning's floor sweepings from the ward and its adnexa contained 102 million streptococci (see Thomas, 1941a). Single blankets from patients with streptococcal tonsillitis in this ward were found to contain $\frac{1}{2}$ -1 million of these organisms. As many as 1,000 streptococci have been recovered from 10 cu. ft. of air in a cubicle after the bed had been made. Crosbie and Wright (1941) have shown that *C. diphtheriae* also can persist in large numbers in floor dust.

Treatment of floors.—The treatment of wooden and linoleum floors with spindle oil (approximately 1 gallon per 1,000 sq. ft.) effectively reduces the amount of dust, which can be raised on sweeping (van den Ende, Lush and Edward, 1940; Thomas, 1941b). The method of application of the oil is simple and requires only a flat dish and a household mop. If properly applied and the excess removed after six to twelve hours there need be no excessive slipperiness. The appearance of treated floors though dull is not unpleasant. One drawback is the fact that the oil is not bactericidal and, moreover, inhibits the bactericidal action of the majority of simple antiseptics. Efforts are at present being made to prepare suitable mixtures of antiseptics in spindle oil.

Treatment of bedclothes.—It has already been shown that the treatment of bedclothes with medicinal liquid paraffin will reduce by 90% or more the numbers of bacteria

gestion has been made that in order to maintain a good standard of aseptic technique a special asepsis officer should be appointed to supervise all matters relating to asepsis, antiseptics, disinfection and the general bacteriological control of the hospital as a whole. In a large hospital this may be necessary, yet it is not the ideal solution. A surgeon in charge of a Unit should be responsible for the good conduct of all the procedures undertaken by it, whether in the operation theatre, wards or out-patient department. He must set an example, for no amount of effort on the part of his colleagues or assistants will compensate for his own carelessness, and in dealing with matters of asepsis and disinfection he can always obtain the advice and valuable collaboration of the hospital bacteriologist. Such collaboration has been neglected in the past and would not be encouraged by the creation of this new officer. Medical men and nurses should be educated to acquire an aseptic conscience—a better thing, to my mind, than their being shadowed by an aseptic policeman.

Dr. M. van den Ende: During the last two years a great deal of interest has been shown in methods designed to reduce the incidence of cross-infection in hospital wards. Because of the war, attention is being directed mainly to hospital infection of wounds. It is now becoming apparent that important differences in the mode of transmission probably exist in different wards. Thus, in scarlet fever and ear, nose and throat wards large numbers of streptococci are consistently found on the bedclothes of patients, on the floors and in consequence in the air during bedmaking and sweeping. The numbers of streptococci recovered from the air of wards occupied by patients with war wounds, on the other hand, has been much less striking. It would also appear that the risk of infection of the respiratory tract by air-borne streptococci must be far greater than the risk of wound infection from this source. The respiratory tract retains practically all (90% or more) of the floating particulate matter, including bacteria, from 10 cu. ft. of air during each hour, and only a fraction of the total number present would settle on to a flat surface during the same period (approximately one-thirteenth).

During our own work in general surgical wards, plates were exposed in the wards throughout the day and we were struck by the relative infrequency with which β -haemolytic streptococci were recovered from the air, in spite of the fact that some of the wounds in the ward were infected (van den Ende and Spooner, 1941). McKissock, Wright and Miles found that in a surgical ward only 8 streptococci would settle in an hour on to an area of 1 sq. ft. These results are not strictly comparable with those obtained with a slit sampler in ear, nose and throat wards, but they do suggest that the exposure of wounds to ward air may not constitute the greatest risk of infection. It must be remembered that the number of streptococci which constitute an infective dose for open wounds is not known. If this number should prove to be small, dust as a factor in the transmission of infection may not be so unimportant. The most important single factor in the transmission of infection to wounds appears to be contact. It has been shown that the incidence of hospital infection of wounds can be reduced from 15.4% to 1.1% by a carefully controlled dressing technique (McKissock *et al.*, 1941). But although the common mode of infection may be by contact, measures to prevent air-borne spread of streptococci cannot be neglected, especially for extensive open wounds and large burns. Thus throat carriers of β -haemolytic streptococci are an abundant source of the infective agent. They are distributed from the respiratory tract in droplets which may infect either directly or after having settled and dried on the bedclothes and been redistributed in the dust. The recommendations in the M.R.C. memorandum on the prevention of "hospital infection" of wounds (1941) include advice on the wearing of masks, the minimum disturbance of dust and adequate protection of instrument dishes by well-fitting lids.

Because in scarlet fever and ear, nose and throat wards the numbers of air-borne and particularly dust-borne organisms are considerable, these wards offer the best opportunities for studies on the spread of streptococci in the air. Most of our work has been done in ear, nose and throat wards into which the patients were admitted mainly for tonsillectomy or submucous resection of the nasal septum (Thomas and van den Ende, 1941; van den Ende and Thomas, 1941). The upper respiratory tracts were therefore probably especially susceptible to infection. So far our experiments have not been of long duration and

that the planning of such trials must be careful, in full cognizance of other modes of transmission of infection which, if uncontrolled, may mask any beneficial effect of dust-laying.

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Colonel L. Colebrook described a series of cross-infections which occurred in one of the wards of Sir Harold Gillies' Plastic-Surgery Unit. These infections were disclosed by the work of Major A. E. Francis, R.A.M.C., in conjunction with the late Dr. Fred Griffith, Dr. Dora Colebrook and Dr. S. D. Elliott.

Early in January 1941 a Flying Officer was admitted with very severe burns. He had been treated in another hospital with sulphapyridine by mouth, and a Type 12 streptococcus was isolated just before his admission to the Unit, which proved to be almost completely resistant to the sulphonamide group of drugs. As such resistant streptococci are not common, and their resistance can be readily detected by simple *in vitro* tests, it was possible to recognize, or at least suspect, the arrival of this strain in other patients' wounds. These suspicions were always confirmed by subsequent serological typing of the strains.

Colonel Colebrook then went on to describe briefly the clinical misadventures which had befallen 12 other patients in the course of eight months, as a result of acquiring this sulphonamide-resistant strain of streptococcus in their wounds. [As Major Francis will be publishing this story separately, only a brief summary is given here.] Eleven cases were burns, in various stages of repair, and one an extensive granulating wound resulting from a road accident.

In two instances, although the lesions were almost completely healed, the arrival of this streptococcus was followed by fever and tissue breakdown. A similar, but more severe infection, occurred spontaneously in the clinically healthy granulating wound of a third case.

In five other patients a sharp febrile attack and acute suppuration (uncontrolled by oral plus local sulphanilamide) followed a plastic repair operation carried out when this streptococcus was present in the wounds. The object of the operations was not achieved.

During the period of its activity, a number of patients had been admitted to the ward with other types of hæmolytic streptococcal infection (sulphonamide-sensitive) but frequent cultures had failed to detect any spread of these strains to other patients. He did not know how the Type 12 strain had been conveyed from patient to patient. That had occurred in spite of all measures taken to prevent it—oiling of floors, wearing of masks and gloves, segregation of infected cases and a continued effort to improve the technique of ward dressings. (The control of dust from blankets was not attempted.)

As a result of the experience with this strain it had become a matter of routine to test the sulphonamide-sensitiveness of all hæmolytic streptococci isolated from open wounds

distributed into the air during bedmaking. The method involved the soaking of bedclothes in a 30% solution of liquid paraffin in white spirit, the removal of excess of the solution by centrifugation and evaporation of last traces of white spirit. The method was costly, involved the use of special equipment and there was some degree of fire risk attached to it. The effectiveness of the oil in laying the dust was nevertheless striking. To overcome the present difficulties of supply of medicinal liquid paraffin, and the inconveniences of the method a search was made for other dust-laying oils which needed no extra equipment for their application. A mineral oil, slightly less highly refined but otherwise the same as liq. paraffin lev. B.P. namely "technical white oil" has been found very effective (van den Ende and Thomas, 1941). It has also been possible by using suitable emulsifying agents to prepare watery emulsions of the oil which are fine enough and stable enough to withstand centrifugation in a hydro-extractor.

COMPOSITION OF A SATISFACTORY EMULSIFIABLE OIL (OIL NO. 12), *Lancet*, 1941 (ii), 757.

| | Parts by weight |
|----------------------------------------------------|-----------------|
| Sulphonated castor oil (50% neutral finish) | 225 |
| Technical white oil | 175 |
| Ester salts powder (60% active material) | 70 |
| Cetyl alcohol | 70 |
| Cresylic acid | 70 |

These oils when applied from 20% aqueous emulsions have given results in laboratory and ward trials, as striking as those obtained with liquid paraffin. Most of the emulsifiable oils which we have used have the additional advantage of being bactericidal against bacteria deposited on them in the form of a coarse spray of broth culture. They may therefore prove particularly effective against streptococci in large droplets from the respiratory tract. This bactericidal action probably accounts for the fact that in two cubicles in which the oils were tested, not a single streptococcus was recovered from the air during bedmaking in spite of the fact that both occupants were suffering from acute streptococcal infections (one of these was a tonsillitis, the other a facial erysipelas). In earlier experiments with liquid paraffin applied from organic solution, streptococci, although greatly reduced in numbers, were never completely absent from the air.

If these oils were to be tried in large-scale hospital experiments, the ordinary laundry equipment would probably be found adequate. The oil could be added to the water of the last rinse in the washing machines, and would not therefore necessitate any serious modification of the usual routine.

The methods of dust-laying which have been described are the results of laboratory experiments and small hospital trials. Reliance has been placed on counts of the bacteria in the air, and the effect on the incidence of hospital infections has not been studied. The stimulus to the research has been the demonstration by many previous workers as well as ourselves that the bacteria in the air of hospital wards are mainly dust-borne, and that these dust-borne organisms are resistant to physical and chemical methods of air "sterilization". The number of instances in which hospital infection could directly be attributed to dust is small. Cruickshank (1938) reports the infection of burns from dust. White (1936) an instance of acute tonsillitis following the sweeping of a dusty floor. It seems probable, however, that many instances of dust-borne infection have not been recognized because of the many modes of transmission of infection which have existed at the same time. The dust-laying methods which have been outlined may help to simplify the tracing out of sources of infection. Should dust prove to be of importance, the methods which have been outlined are practicable and simple enough to be adopted as a routine measure in wards. Dust-laying is not advised for immediate and general adoption. In fact general surgeons will probably find it much less important than attention to the details of a carefully planned dressing technique. It may on the other hand be found necessary in ear, nose and throat and certain medical wards. Before these questions can be decided controlled field trials will have to be done. It is not sufficient to study the incidence of cross-infection in two wards, one of which has floor and bedclothes treated with dust-laying oils whereas other modes of infection, such as by contact or large droplets, are uncontrolled in both. Instead, it is important to realize

Section of Radiology

President—M. H. JUPE, D.M.R.E.

[October 17, 1941]

DISCUSSION ON THE FUNCTION OF THE RADIOLOGIST IN THE DIAGNOSIS AND TREATMENT OF CHEST INJURIES

Dr. L. G. Blair (*Thoracic Surgical Unit, Harefield*): In the Harefield Thoracic Surgical Unit the radiologist is invited to join the weekly round made by the whole group of physicians and surgeons, and to enter into the subsequent free discussion of all cases. By this team-work full value is obtained from the X-ray evidence available in any particular case. In the absence of such a combined consultation I personally do not report on a case unless I have a complete history and some indication of the clinical condition of the patient. I shall describe the technique in general and give a short account of the localization of foreign bodies, and of some conditions where the diagnosis is almost entirely radiological.

Preliminary films.—Radiology is important in the diagnosis and control of treatment of injuries of the chest. It is essential to get adequate films, postero-anterior or antero-posterior and lateral, at the earliest possible moment. These films enable the clinician to form an early and accurate assessment of the case. Failure to provide good films and consequent misinterpretation have led to delay in instituting correct treatment, and therefore the almost inevitable train of disasters. We are so convinced of this that now as a routine at Harefield, whenever possible cases on admission are taken to the ward via the X-ray department. These cases should be examined without removing them from the stretcher. It is far less strain on a patient to have good films taken with a high-powered plant in this way than to have indifferent films taken with a portable apparatus, with consequent long exposure; in the ward.

From these preliminary films a fairly accurate knowledge of the conditions in the chest can be obtained, the presence of foreign bodies noted, and some estimate of their situation made. The whole of the chest should be included in the films, otherwise foreign bodies may be missed owing to the films having been badly centred or centred on the more obviously injured side of the chest.

Localization of foreign bodies.—I have not had to make a single depth measurement for a foreign body in the chest during the past two years, and I am certain that such measurements are unnecessary and useless. It is the anatomical localization which is important and which can be made with certainty by a rapid screening examination. If, on respiration, the foreign body moves with the lung markings near it, it is *in* the lung; if it does not so move, it is *outside*. If it moves with the ribs it is *outside* the lung and in close contact with the ribs. If it does not move with either it is very superficial unless it is in the region of the shoulder girdle.

Foreign body in the lung.—Having decided that the foreign body is in the lung further localization is required. From the preliminary films and a knowledge of the position of the various septa it should be possible to say in which lobe of the lung the foreign body lies. If there is a free pleura further localization is waste of time as the foreign body drops with the lung as soon as the pleura is opened, and then, I am told,

in the plastic unit, and if a resistant strain turned up the surgeons were very reluctant to operate.

Colonel Colebrook could not agree with the suggestion made by Professor Paterson Ross that a whole-time officer as Supervisor for the Control of Infection (M.R.C. War Memo. No. 6) was not required in hospitals. There was need for a general policy in the hospitals, and for the appointment of an officer with authority to superintend theatre procedure and ward technique, &c.

In conclusion, Colonel Colebrook advocated simple demonstrations and practical work for the sisters and nurses in the laboratory—planting of infected swabs before and after two minutes boiling, shaking of blankets over blood-agar plates, &c., &c., so that their interest should be stimulated and they should get a real working knowledge of the problems presented by cross-infection.

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Localization of foreign bodies.—I have not had to make a single depth measurement for a foreign body in the chest during the past two years, and I am certain that such measurements are unnecessary and useless. It is the anatomical localization which is important and which can be made with certainty by a rapid screening examination. If, on respiration, the foreign body moves with the lung markings near it, it is *in* the lung; if it does not so move, it is *outside*. If it moves with the ribs it is *outside* the lung and in close contact with the ribs. If it does not move with either it is very superficial unless it is in the region of the shoulder girdle.

Foreign body in the lung.—Having decided that the foreign body is in the lung further localization is required. From the preliminary films and a knowledge of the position of the various septa it should be possible to say in which lobe of the lung the foreign body lies. If there is a free pleura further localization is waste of time as the foreign body drops with the lung as soon as the pleura is opened, and then, I am told,

in the plastic unit, and if a resistant strain turned up the surgeons were very reluctant to operate.

Colonel Colebrook could not agree with the suggestion made by Professor Paterson Ross that a whole-time officer as Supervisor for the Control of Infection (M.R.C. War Memo. No. 6) was not required in hospitals. There was need for a general policy in the hospitals, and for the appointment of an officer with authority to superintend theatre procedure and ward technique, &c.

In conclusion, Colonel Colebrook advocated simple demonstrations and practical work for the sisters and nurses in the laboratory—planting of infected swabs before and after two minutes boiling, shaking of blankets over blood-agar plates, &c., &c., so that their interest should be stimulated and they should get a real working knowledge of the problems presented by cross-infection.

the case of a lower lobe, which is illustrated (fig. 7), will appear as a triangular shadow at the base. Screening examination at this time will show a "mediastinal wandering" into the side of the pneumothorax and by bronchoscopy or bronchography it should be possible to demonstrate the narrowing of the bronchus. In the case illustrated here the lobe was eventually removed by Mr. Holmes Sellors.

Much has been written about the effects of blast on the lung. The typical picture as described by Zuckerman (1941) is now almost too well known to need description, with its peripheral distribution and shadows rather resembling pleural thickening (fig. 8). It is, however, interesting to note the comparative rapidity of resolution shown in these cases, where in about three days considerable clearing of the lung fields has occurred and frequently in ten days they have returned to normal, and to compare this speed of resolution with another type of shadow found in the chest following injury. This second type of shadow is much more frequently localized to one lung and complete resolution may take as long as six or eight weeks. It has, moreover, been seen in cases where there was no possibility of blast injury and these appearances, we think, are due to gross localized hæmatoma of the lung (fig. 9).

There is another type of shadow which may be found in the chest following trauma. This is a somewhat rounded opacity with well-cut, clearly defined margins. We have so far seen only one example of this and we were at a loss to account for it. Mr. Holmes Sellors explored the chest and found that the opacity was due to organizing blood-clot in a cavity in the lung, the walls of which were ragged and did not resemble the walls of a pre-existing cyst. We consider, therefore, that it is an unusual type of hæmatoma (fig. 10). The mention of a pre-existing cyst leads to a word of warning. Because a case is admitted as an air-raid casualty one should not necessarily interpret all the shadows present as being due to the accident. In one case that was admitted to our Chest Unit as an air-raid casualty with a history of injury to the right lower ribs and hæmoptysis, a rounded shadow was seen in the right lower zone which persisted unchanged for some time. Further investigation proved this to be a primary bronchial carcinoma. There is little to say from the purely radiological standpoint about hæmothorax, as in appearance it resembles an ordinary effusion with the exception of the occasional and somewhat bizarre appearances that may result from clot formation.

Though we are primarily radiologists yet are we doctors also, and if as the result of our radiological investigations we are able to make suggestions for treatment to our clinical colleagues, then I think we should do so.

It has long been known, although little attention has been paid to it, that intrapleural pressures vary with the position of the individual. It may be accepted that the intrapleural pressures are lowest when the patient is lying on the opposite side to the one being investigated; that the pressure becomes more positive when the patient is erect; that there is a further rise with the patient lying flat on the back, and a very definite increase of pressure when the individual is lying on the side in which the pleural readings are being taken. Livingstone (1928) says: "With changes in posture of the body the diaphragm alters in level making a difference in the volume of the chest. . . . In the erect position the diaphragm is low and the intrathoracic volume is large; in the supine position it is high and the volume is diminished." A. E. Barclay ("The Digestive Tract" 1936), stated that: "When a healthy subject lies on one side or the other . . . it (the diaphragm) swings about a central point so that the lower side goes up in the chest and the upper side becomes correspondingly lower."

Some years ago, by taking films in the various positions, I was able to show that the variation in pressure was due not only to the alteration in position of the diaphragm but to mediastinal displacement, the mediastinum falling into the dependant side. A possible practical application of this is seen when it is realized that the escape of air or blood into the pleural cavity is only controlled in the vast majority of cases when the pressure in the pleural cavity has risen sufficiently high as to close the opening into the pleura or to compress the vessel which is bleeding. With a view to using posture to control this pressure, with the co-operation of my colleagues, I have taken some further films in the various positions while they have read the intrapleural pressures. I will only quote one case, but the results have been consistent throughout. The pressures in this particular case, which were read in the left chest, were as follows: Lying on the right side - 12 to - 4; erect - 14 to - 2; lying on the back - 13 to 0; lying on the left side - 10 to +3. The films were taken in expiration. The expiratory pressure has been

can be easily felt. If, however, the pleura is adherent more help can be given to the surgeon. At Harefield in such cases we mark the skin overlying the foreign body, using a very small diaphragm, in the antero-posterior and lateral planes. These markings give the rough position and depth of the foreign body. A further mark is then made on the skin to show the nearest position on the surface to the foreign body. At the same time a count of the ribs is made and the rib at the site of the nearest point is noted. This counting of the ribs is important as there are two possible fallacies. First of all the skin marking in relation to the rib and foreign body will vary with the position of the arm. Movement of the arm may result in the shifting of the skin marking by as much as two interspaces. It is therefore necessary to place the arm in the position it will be in at the time of operation. The ward staff should be instructed not to remove these marks before operation.

Foreign bodies in the mediastinum, including the heart.—Again, the localization of mediastinal foreign bodies should be anatomical and depth measurements are of little use. From antero-posterior and lateral films in the majority of cases a fairly accurate anatomical localization can be made. A screening examination will also help, particularly as to the type of movement, if any, that the foreign body shows. There should be no difficulty in distinguishing between the transmitted pulsation of a foreign body close to the heart and great vessels from the dancing movements of a foreign body which is in the chambers of the heart (fig. 1).

Use of tomography in chest injuries.—During the course of two years I have not been requested to do a single tomogram for a parenchymatous chest lesion. I have attempted tomography for fractures of the sternum with slight success in the lateral plane, and I think none at all in the antero-posterior. I have, however, found it of value in the assessment of damage in fractures involving the dorsal spine. When the superadded shadows of the lungs and ribs are removed a surprisingly clear picture can be obtained of the dorsal vertebrae (fig. 2).

Some types of injury where the diagnosis depends almost entirely on radiology.—The appearances of a tension pneumothorax with an almost completely translucent hemithorax, a depressed diaphragm on the same side, and a marked mediastinal displacement away from the pneumothorax are well known (fig. 3). But there are several conditions which, from both the radiological and clinical points of view, may closely simulate the tension pneumothorax. Some of these conditions may be traumatic, others not. It is possible to get a giant cyst of the lung giving an exactly similar radiological picture. It is only when the radiological evidence is correlated with the physical condition of the patient that it is possible to get some idea of the probable diagnosis. There will be no history of an artificial pneumothorax, no very dramatic onset, and the patient will not show the distress that would have been present with a tension pneumothorax. An illustration of such a case is shown by kind permission of Mr. Tudor Edwards who removed a giant cyst (fig. 4).

There is another condition which both radiologically and clinically may be confused with a tension pneumothorax. It is traumatic in origin and follows gross compression of the thorax or abdomen with consequent diaphragmatic rupture and hernia of the stomach into the thoracic cavity. If, however, the upper limit of the air-containing space is examined carefully it will be seen to be curved with the convexity of the curve upwards. This appearance should always suggest the possibility of diaphragmatic hernia, as a spontaneous pneumothorax would not give this shadow unless there had been a previous lesion at the apex causing an adherent pleura, and then the upper limit of the pneumothorax would be much straighter and would not show this curved line with the convexity upwards (fig. 5).

A further condition which may be mistaken for localized pneumothorax is the appearance given by emphysematous bullae, but if the films are carefully examined in these cases it is possible to see very fine strands crossing the apparent pneumothorax cavity, strands which are too fine for the adhesions of a pneumothorax. It is important to recognize this as diagnostic needling is not free from danger (fig. 6).

It is important to follow up these cases of tension pneumothorax and to watch the re-expansion of the lung, as there is always the possibility of some injury to the bronchial tree having occurred at the time of accident which may lead to stenosis of a bronchus. The lobe or portion of the lobe involved will not then re-expand completely and in

the worry to the patient is no greater than if the portable plant is used and the results are infinitely more satisfactory to ourselves. However circumstances must guide procedure in this respect (figs. 13 and 14).

The necessity for full and complete radiological investigation is of course obvious; without this, serious injuries may be overlooked in the absence of adequate physical signs or in the confusion that inevitably results from multiplicity of lesions. What may ultimately turn out to be the major injury may not be recognized at the initial clinical examination or may be relegated to a minor position of importance. For example in the cases of obvious chest injuries a penetrating foreign body may end its course some distance from the thorax. I recall a case in which a bullet entered the upper chest and ultimately lodged in the thigh, but the mention of this is only to draw attention to the necessity of ensuring that complete radiological investigation be made. With multiple injuries and when individuals are close to the site of an explosion the chest comes in for its fair share of the bodily damage. An intrathoracic lesion may escape notice in the early stages if the clinical signs and symptoms are slight—as in a case of “blast” which can occur with practically no symptoms.

There are some circumstances in which radiology can give little or no help. The lateral view of the upper thorax is never particularly clear on account of the thickness of the shoulder girdles, and when there are injuries to the shoulder or clavicle the arm cannot always be moved out of the way; consequently the advantages of even an average film are discounted. A case in point was that of a man who was shot by a revolver bullet. On the antero-posterior film the bullet was absolutely central and from its suspected course its presence in the posterior mediastinum was suggested. The injury, however, had involved the clavicle and the necessary lateral film for accurate localization could not be taken. At operation it was found that the missile had passed through the upper chest drilling the upper lobe of the lung and had become lodged in the spinal muscles. The necessity for lateral as well as antero-posterior films of the chest is essential whenever accurate localization is required.

The metallic nature of bullets and bomb fragments makes their recognition a comparatively easy matter, but there are numerous other substances, such as glass, wood, brick and cloth material, which are not easily identified when deeply embedded in the chest. Some of these have been recognized, but I fear that there are many that have escaped notice by virtue of their relatively radio-translucent nature.

Radiology by itself can identify fluid collections within the pleural cavity, but it remains for the clinical history and the aspirating needle to determine whether such fluid is simple effusion, blood or pus. Similar diagnostic difficulties occur with pulmonary shadows which may be produced by hæmorrhages, consolidation and patchy collapse, and other aspects of the case must be studied to arrive at a more accurate answer.

Foreign bodies.—In considering foreign bodies in the chest Dr. Blair has indicated the method of determining their relationship to the chest wall and from the surgical aspect a more detailed consideration of intrapulmonary missiles can be made. A good deal can be learned from correctly distanced and centred films in two planes. The method of finding the lateral plane of the foreign body and then transferring this line to the antero-posterior film and making a careful rib count serves well, but from one's experience in the drainage of lung abscesses for example one knows how easy it is to be an inch or so out. If the foreign body is not unduly large we find, therefore, that it is advisable to localize it under the screen with the patient's arm held in the position that it will be in at actual operation. The dark minutes occupied in screening are amply repaid at the full light of operation.

It is difficult to decide what missiles can safely be left alone and what are best removed. It would appear that life is not compatible with an embedded fragment in the chest much over one and a half to two cubic inches—at any rate we have not seen them in survivors. Foreign bodies may be removed—shortly after their reception, or at a later date when intrathoracic conditions have returned to normal. “Immediate” removal is often practised when the missile is of large size; it is not the actual foreign body that demands attention, but coincidental intrathoracic hæmorrhage and lung damage. At thoracotomy bleeding can be arrested, the pleural cavity cleaned out and lung damage repaired in addition to removing the missile. At the same time as much primary excision of the superficial wounds and wound track as is feasible can be undertaken. These cases

chosen as this is the one that is least affected by variations in the depth of respiration. Tracings have been made from these films and superimposed. In the first one the continuous line represents the state of affairs in a patient lying on the right side, the broken line the condition inside the chest when the patient lies on the left side (fig. 11). In the second illustration the continuous line is a tracing of the supine film and the broken line the tracing of a film taken with the patient lying on the left side (fig. 12). It will be readily seen that though there is an alteration in the expiratory pressure from -4 to $+3$ with a turn from right to left side, and of 0 to $+3$ by simply turning from the supine to the left side, yet there is no extra burden thrown on the other lung by a displaced mediastinum. In fact, the mediastinum is displaced into the injured side. It was found necessary to remove 400 c.c. of air to reduce the pressure from $+3$ to atmospheric; in other words by turning the patient from the supine position into the left lateral recumbent position there had been a saving of 400 c.c. of air and the balance of mediastinal displacement is still considerably in the patient's favour. Now this is an absolutely constant finding, and should, in my opinion, mean that if, as a first-aid measure, every case showing injury to the chest is turned on to the affected side the loss of air or blood will be considerably smaller.

Three possible arguments may be raised against this theory. The first is that the dependant lung may show congestion. There is no proof of this and I think the change in appearance is due to the increased compression of the lung. Moreover, Hilton (1925) showed that after the induction of a pneumothorax there was a reduced flow of blood through the collapsed lung. Another point that may be raised is based on the work of Björkman (1934) in his studies on broncho-spirometry. He claims that in the lateral position the function of the upper lung is diminished. I feel from the radiological studies we have made that there is some other explanation of his findings, and think that possibly in his series the patients, when lying on the side, had the dorsal spine curved with the convexity downwards thus narrowing the rib spaces on the upper side. This would, of course, lead to diminished respiratory exchange in this lung. If, however, the patient is straight in the lateral position or is lying over a cushion placed under the bad side then this state of affairs does not arise. The last objection that might be raised is that when the patient is lying on the injured side it might be more difficult for him to expectorate from the lung with a consequent tendency to massive collapse. I doubt if this would occur, and as the use of this posture is only suggested as a first-aid measure the possibility need not be seriously considered. By this simple measure it may be possible to prevent the more immediate effects of marked exsanguination and the more distressing symptoms of pressure pneumothorax. The surgeons' work should also be easier, in that the resultant cavity with which they have to deal will be smaller, possibly half the size of the one that would have been left had the patient been kept in the more accepted position following injury.

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Mr. T. Holmes Sellors: The main thing that has struck me in connexion with the early stages of casualty work is the average inadequacy of the original films. Portable X-rays are obviously better than nothing, though I frankly admit that many of the films produced, particularly the lateral views, are quite beyond my interpretation. In most hospitals radiography is not considered until the patient is in bed and fully settled in the ward. Now if it were possible for the patient to be X-rayed on the full set on his way from the receiving room to the ward the results would be of incomparably greater value. It should be practicable for this to be done without moving the patient from the stretcher and without undue manipulation and distress. A severely injured individual who has survived transport to hospital is not likely to suffer much additional harm from an expeditious visit to the X-ray department on his way to bed. In support of our contention I would mention that with post-operative cases we find that the majority of them can be wheeled in their beds to the X-ray room for the taking of necessary films:

soon as the risk of active bleeding is over—within the first twenty-four to forty-eight hours. With a large hæmothorax the removal of blood will have to be associated with a certain amount of gas-replacement. After the initial aspiration, clinical examination and X-rays will show how much fluid is left and the aspiration should be repeated every day or every other day until the pleural cavity is completely dry. A clear costophrenic sinus suggests efficient removal, but this should be checked some days later in case any effusion has collected. The radiological control of the final stages of aspiration is an important one and a fluid level towards the base is more surely recognized by X-rays than by percussion, particularly on the right side.

Sometimes an irregular basal mass is found and this is probably produced by clot (fig. 17) which cannot be removed through a needle. If this is of any size the question of evacuating it through a small thoracotomy incision should be considered. The whole treatment aims at producing a rapidly expanded lung which will reach a chest wall that is not unduly thickened by organizing blood and fibrin. The disaster of a badly treated hæmothorax, even if there has been no infection, has to be seen to be believed (fig. 18). It is no good urging that conservative measures give quite good results; there is all the difference between a patient who is able to get about and one who returns to his work with a vital capacity of 80% to 90% of his normal.

The main complication and fear that attends hæmothorax is *infection*. In the diagnosis of this radiology can give little or no help unless it recognizes the presence or increase of gas above the fluid in the case of anaerobic infections. The colour, smell and bacteriological findings of the aspirated fluid must give the answer. However, when infection has developed radiology can give assistance in determining the right site for drainage when this becomes necessary. Admittedly with a free and extensive collection of infected blood trial by aspiration gives the most dependent point of the cavity with considerable accuracy; but if there is any doubt the injection of a few cubic centimetres of radio-opaque oil into the cavity followed by two-plane X-ray films gives practical visual help.

The control of an infected pleural cavity is frequently one of the most mishandled features of surgery, as witnessed by the large numbers of chronic empyemata that are allowed to occur every year. Detailed and continued observation of the healing process is essential once adequate drainage has been instituted and maintained. Radiology gives inestimable help during the whole period of closure if the cavity is filled with radio-opaque oil at regular intervals. The ability to visualize the size and shape of the empyema cavity from the antero-posterior and lateral films enables the surgeon to give proper attention to details of drainage and tube control before it is too late. These pleurograms do not only help (fig. 19) by showing the form of the cavity; they may show unsuspected pockets which communicate with the main cavity. Also bottle-neck tracks and small broncho-pleural fistulae may be detected in a manner that cannot be achieved by any other method. Pleurograms are far better than trusting blindly to nature, probes or bougies. In cases where there is pleural thickening the use of a barium impregnated or "loaded" tubing is useful since the tubing shows up clearly on X-ray.

Abdomino-thoracic injuries.—Injuries which involve both chest and abdomen are associated with perforation of the diaphragm—a feature which may not be of much actual significance on the right, but which may have dramatic consequences on the left. If the tear in the diaphragm is of any size the probability of the stomach being found in the thorax is considerable.

Closed crush injuries to the lower chest, as may be instanced by explosions, car accidents, shunting-yard misadventures, and falls from a height, may produce rupture of the diaphragm. If this occurs on the left the intra-abdominal pressure and the pull of the pleural pressure causes the stomach to enter the pleural cavity; and the big bulk of the gastric organ gives rise to many of the errors in diagnosis that Dr. Blair has already indicated. These mistakes are common in spite of good radiological pictures and in any case of doubt the thin upper curved line of the stomach wall should be looked for under the rib shadows.

Penetrating wounds which involve the diaphragm are almost certain to damage some abdominal viscus at the same time. Liver, stomach, colon and spleen may all be damaged at the same time, but in most cases the most obvious and important lesion affects the stomach. In these circumstances diagnosis is rarely helped by X-rays. Perforation of gut might be expected to give signs of free gas in the peritoneum, but in the cases

on the whole do well in spite of their apparent severity, and as a rule there is little difficulty in finding the foreign body as the wound track is identified. Large fragments tend to be single, whereas small pieces are often part of a general spattering of the patient, who consequently has multiple injuries to be considered. The presumable reason why large fragments are so commonly single is that survival after being hit by several big lumps of metal is a precarious matter, and multiple injuries of this character are rarely seen by the surgeon.

"Immediate" removal of a foreign body incidental to arrest of hæmorrhage and wound excision is the ideal of treatment, but in practice it often happens that the chest injury is treated expectantly and the question of foreign body removal does not come up until later. During this interval such conditions as hæmorthorax should be treated by aspiration and actual intervention only considered when all wounds are healed and the danger of infection has passed or truly subsided—on an average a period of three to four weeks.

It is often urged that a foreign body which has been accommodated by the lung for some time without giving trouble might well be left alone, apart from instances in which symptoms make its removal a more urgent matter. But the later history of such cases is studded by the occurrence of hæmoptyses, lung abscess formation and bronchiectasis. In a case recently under my care, the patient as a boy was stabbed by a hat-pin which broke off and after an initial hæmoptysis gave no trouble or discomfort; now, thirty-two years later (fig. 15), the man has had several hæmoptyses which can be attributed directly to the embedded foreign body. As a rough working rule we would suggest that any retained foreign body (fig. 16) bigger than the little finger-nail should be removed. The patient should be for preference youngish and fairly fit. Another factor that has considerable bearing on the decision is the position of the missile in the lung; if it is near the surface it may give rise to later pleural adhesions and pain, and in this region it is easily reached. *Per contra*, if it lies close to the pericardium or lung root it may constitute a dangerous centre for an infective lesion at a later date and be extremely difficult to approach. A young girl who had a small bomb fragment close to the pericardium did well at the start under conservative treatment. However, an effusion in the pericardium developed as a direct result of irritation; this subsided as soon as the foreign body was removed.

If removal is decided upon it is advisable to attempt induction of an artificial pneumothorax. In a proportion of cases the pleura is free and radiology will probably be able to suggest in which lobe the foreign body may be found. The procedure in this case is to perform a small formal thoracotomy and to identify the fragment in the lung by palpation and if necessary needling. On the other hand if the pleura is adherent accurate screening localization makes it possible to remove the foreign body through a small incision directly over it. It is worth noting that when adherent lung is incised its elastic character causes the wound to gape and retract, so that the surgeon is liable to push the fragment inwards when probing or feeling for it and thus to find it at a deeper level than it actually was at the start.

Hæmorthorax.—Blood in the pleural cavity is the most common finding in cases of chest injury. The blood may come from lung or chest wall, and it is important to see whether ribs are broken or not. Absence of rib injury indicates that bleeding from intercostal vessels is unlikely, but their fracture immediately suggests the possibility. Associated air in the pleura in the absence of a functional external opening means that lung has been punctured. If blood and/or air accumulate to any extent in the pleural sac the lung is collapsed and the actual pressure of the alien material may cause displacement of the mediastinum. Tension or pressure phenomena are common and should be recognized clinically more frequently than they are; it is often left to radiology to make the diagnosis. Relief of the patient's extreme distress can be obtained by putting a needle into the pleura without delay and removing a sufficient quantity of fluid or air.

The essential treatment of a hæmorthorax of any appreciable size is its early removal. It has been suggested that a small collection be left to absorb, but there is no justification for running the risk of the subsequent loss of respiratory function by following this course. Intrapleural blood tends to remain fluid unless infection supervenes, and it can easily be aspirated through a needle. The line to adopt is to empty the chest completely as

soon as the risk of active bleeding is over—within the first twenty-four to forty-eight hours. With a large hæmothorax the removal of blood will have to be associated with a certain amount of gas-replacement. After the initial aspiration, clinical examination and X-rays will show how much fluid is left and the aspiration should be repeated every day or every other day until the pleural cavity is completely dry. A clear costophrenic sinus suggests efficient removal, but this should be checked some days later in case any effusion has collected. The radiological control of the final stages of aspiration is an important one and a fluid level towards the base is more surely recognized by X-rays than by percussion, particularly on the right side.

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on the whole do well in spite of their apparent severity, and as a rule there is little difficulty in finding the foreign body as the wound track is identified. Large fragments tend to be single, whereas small pieces are often part of a general spattering of the patient, who consequently has multiple injuries to be considered. The presumable reason why large fragments are so commonly single is that survival after being hit by several big lumps of metal is a precarious matter, and multiple injuries of this character are rarely seen by the surgeon.

"Immediate" removal of a foreign body incidental to arrest of hæmorrhage and wound excision is the ideal of treatment, but in practice it often happens that the chest injury is treated expectantly and the question of foreign body removal does not come up until later. During this interval such conditions as hæmothorax should be treated by aspiration and actual intervention only considered when all wounds are healed and the danger of infection has passed or truly subsided—on an average a period of three to four weeks.

It is often urged that a foreign body which has been accommodated by the lung for some time without giving trouble might well be left alone, apart from instances in which symptoms make its removal a more urgent matter. But the later history of such cases is studded by the occurrence of hæmoptyses, lung abscess formation and bronchiectasis. In a case recently under my care, the patient as a boy was stabbed by a hat-pin which broke off and after an initial hæmoptysis gave no trouble or discomfort; now, thirty-two years later (fig. 15), the man has had several hæmoptyses which can be attributed directly to the embedded foreign body. As a rough working rule we would suggest that any retained foreign body (fig. 16) bigger than the little finger-nail should be removed. The patient should be for preference youngish and fairly fit. Another factor that has considerable bearing on the decision is the position of the missile in the lung; if it is near the surface it may give rise to later pleural adhesions and pain, and in this region it is easily reached. *Per contra*, if it lies close to the pericardium or lung root it may constitute a dangerous centre for an infective lesion at a later date and be extremely difficult to approach. A young girl who had a small bomb fragment close to the pericardium did well at the start under conservative treatment. However, an effusion in the pericardium developed as a direct result of irritation; this subsided as soon as the foreign body was removed.

If removal is decided upon it is advisable to attempt induction of an artificial pneumothorax. In a proportion of cases the pleura is free and radiology will probably be able to suggest in which lobe the foreign body may be found. The procedure in this case is to perform a small formal thoracotomy and to identify the fragment in the lung by palpation and if necessary needling. On the other hand if the pleura is adherent accurate screening localization makes it possible to remove the foreign body through a small incision directly over it. It is worth noting that when adherent lung is incised its elastic character causes the wound to gape and retract, so that the surgeon is liable to push the fragment inwards when probing or feeling for it and thus to find it at a deeper level than it actually was at the start.

Hæmothorax.—Blood in the pleural cavity is the most common finding in cases of chest injury. The blood may come from lung or chest wall, and it is important to see whether ribs are broken or not. Absence of rib injury indicates that bleeding from intercostal vessels is unlikely, but their fracture immediately suggests the possibility. Associated air in the pleura in the absence of a functional external opening means that lung has been punctured. If blood and/or air accumulate to any extent in the pleural sac the lung is collapsed and the actual pressure of the alien material may cause displacement of the mediastinum. Tension or pressure phenomena are common and should be recognized clinically more frequently than they are; it is often left to radiology to make the diagnosis. Relief of the patient's extreme distress can be obtained by putting a needle into the pleura without delay and removing a sufficient quantity of fluid or air.

The essential treatment of a hæmothorax of any appreciable size is its early removal. It has been suggested that a small collection be left to absorb, but there is no justification for running the risk of the subsequent loss of respiratory function by following this course. Intrapleural blood tends to remain fluid unless infection supervenes, and it can easily be aspirated through a needle. The line to adopt is to empty the chest completely as

soon as the risk of active bleeding is over—within the first twenty-four to forty-eight hours. With a large hæmothorax the removal of blood will have to be associated with a certain amount of gas-replacement. After the initial aspiration, clinical examination and X-rays will show how much fluid is left and the aspiration should be repeated every day or every other day until the pleural cavity is completely dry. A clear costo-phrenic sinus suggests efficient removal, but this should be checked some days later in case any effusion has collected. The radiological control of the final stages of aspiration is an important one and a fluid level towards the base is more surely recognized by X-rays than by percussion, particularly on the right side.

Sometimes an irregular basal mass is found and this is probably produced by clot (fig. 17) which cannot be removed through a needle. If this is of any size the question of evacuating it through a small thoracotomy incision should be considered. The whole treatment aims at producing a rapidly expanded lung which will reach a chest wall that is not unduly thickened by organizing blood and fibrin. The disaster of a badly treated hæmothorax, even if there has been no infection, has to be seen to be believed (fig. 18). It is no good urging that conservative measures give quite good results; there is all the difference between a patient who is able to get about and one who returns to his work with a vital capacity of 80% to 90% of his normal.

The main complication and fear that attends hæmothorax is *infection*. In the diagnosis of this radiology can give little or no help unless it recognizes the presence or increase of gas above the fluid in the case of anaerobic infections. The colour, smell and bacteriological findings of the aspirated fluid must give the answer. However, when infection has developed radiology can give assistance in determining the right site for drainage when this becomes necessary. Admittedly with a free and extensive collection of infected blood trial by aspiration gives the most dependent point of the cavity with considerable accuracy; but if there is any doubt the injection of a few cubic centimetres of radio-opaque oil into the cavity followed by two-plane X-ray films gives practical visual help.

The control of an infected pleural cavity is frequently one of the most mishandled features of surgery, as witnessed by the large numbers of chronic empyemata that are allowed to occur every year. Detailed and continued observation of the healing process is essential once adequate drainage has been instituted and maintained. Radiology gives inestimable help during the whole period of closure if the cavity is filled with radio-opaque oil at regular intervals. The ability to visualize the size and shape of the empyema cavity from the antero-posterior and lateral films enables the surgeon to give proper attention to details of drainage and tube control before it is too late. These pleurograms do not only help (fig. 19) by showing the form of the cavity; they may show unsuspected pockets which communicate with the main cavity. Also bottle-neck tracks and small broncho-pleural fistulæ may be detected in a manner that cannot be achieved by any other method. Pleurograms are far better than trusting blindly to nature, probes or bougies. In cases where there is pleural thickening the use of a barium impregnated or "loaded" tubing is useful since the tubing shows up clearly on X-ray.

Abdomino-thoracic injuries.—Injuries which involve both chest and abdomen are associated with perforation of the diaphragm—a feature which may not be of much actual significance on the right, but which may have dramatic consequences on the left. If the tear in the diaphragm is of any size the probability of the stomach being found in the thorax is considerable.

Closed crush injuries to the lower chest, as may be instanced by explosions, car accidents, shunting-yard misadventures, and falls from a height, may produce rupture of the diaphragm. If this occurs on the left the intra-abdominal pressure and the pull of the pleural pressure causes the stomach to enter the pleural cavity, and the big bulk of the gastric organ gives rise to many of the errors in diagnosis that Dr. Blair has already indicated. These mistakes are common in spite of good radiological pictures and in any case of doubt the thin upper curved line of the stomach wall should be looked for under the rib shadows.

Penetrating wounds which involve the diaphragm are almost certain to damage some abdominal viscus at the same time. Liver, stomach, colon and spleen may all be damaged at the same time, but in most cases the most obvious and important lesion affects the stomach. In these circumstances diagnosis is rarely helped by X-rays. Perforation of gut might be expected to give signs of free gas in the peritoneum, but in the cases

that I have encountered there has been no indication of this. The stomach usually remains in the abdomen in distinction to the closed type of injury and the presence of stomach contents in the pleural cavity suggests that the stomach is often blown through the diaphragm into the chest and then retires in a collapsed state to its normal position. In a recent case of bullet wound with entry close to the heart, radiological evidence pointed to extensive thoracic damage, but gave no indication of what had happened below the diaphragm. At thoracotomy it was found that the pleural cavity was full of blood and hard-boiled egg; the small puncture in the diaphragm was enlarged and it was found that both walls of the stomach had been drilled, in addition to damage to liver and spleen. The stomach was collapsed, but there was no sign of stomach contents in the peritoneum. Clearly if the tear in the diaphragm is extensive the free communication between the two serous sacs may lead to a more varied and easily recognized X-ray picture.

Rupture of bronchus.—Dr. Blair has already shown the case of a child who after a crush injury developed a tension pneumothorax and later a collapsed right lower lobe (fig. 7). Bronchoscopy revealed a soft stricture at the opening of the lower lobe and in spite of repeated dilatation the narrowing persisted. The lower and middle lobes were removed by dissection and the specimen showed that the stricture had become complete, and the bronchus was represented by a fibrous band. The collapsed lung contained dilated bronchi full of mucus. The cartilaginous wall of the bronchus had actually been torn across close to the hilum leaving temporary functional continuity through a cuff of mucous membrane. This type of injury has been reported, but its full significance has not been realized. It is a strong indication of the importance of following up all cases of crush injury for some time after their apparent cure; radiograms should be taken and watch kept for any signs of collapse in the lung tissue.

Dr. W. D. W. Brooks: In no branch of medicine or surgery can the radiologist be of greater assistance to the clinician than in the management of cases of chest injury. I am convinced that co-operation in the sense of a weekly conference at which the radiologist and the medical and surgical staff are present, is most important.

A chest injury, if it is of some severity, commonly gives rise to four major sequelae which singly or together may be fatal to the patient. These are shock, hæmorrhage, disordered respiratory and circulatory function, and finally infection. The diagnostic and therapeutic problems presented by the first of these are not, in the light of existing knowledge, matters in which radiology is of any assistance. The onset, and gravity of hæmorrhage too are assessed for the most part by other methods, although when the chest is closed, the exact location and extent of a hæmothorax can best be demonstrated by radiological means, as can the subsequent progress of this thoracic lesion when appropriate therapy has been instituted.

We need efficient yet simply applied tests of respiratory and circulatory function under abnormal conditions. The clinical symptoms and signs, and all the available simple methods of estimating function in these circumstances are frequently fallacious and always incomplete, while more elaborate procedures such as the estimation of the cardiac output or the components of the lung volume, cannot as a rule be applied to just those cases where they are most needed; moreover their determination is difficult and tedious, so that they have to-day an academic rather than a practical significance. We thus are unable to evaluate accurately the degree of impairment of respiratory and circulatory function which a chest injury has caused. Furthermore we usually tend to err the more grossly in our estimate during the early acute phases when proper and commensurate resuscitation is urgently required. Of necessity, a clinical assessment is employed, and I would emphasize that it is more likely to approach the truth if it is based upon an accurate knowledge of what may be called the pathological anatomy which the case presents. It is here that radiology and radioscopy play a most important part. Not only do they usually permit the identification of the nature of the lesion, or lesions, with far greater certainty than is at present otherwise possible, but, in addition, they often allow a limited forecast to be made of the type and degree of respiratory or circulatory disability which may in consequence arise, or which indeed may already be present yet undetected by the crude methods at our disposal. Furthermore, the progress of the case, in respect of its pathological anatomy can best be followed by these means, to our great advantage in ordering appropriate therapy.

Infection as a complication in a chest injury is for the most part recognized and its

severity assessed by non-radiological methods. For example, pus, blood, chyle or serous effusions in the pleural cavity, give the same radiological appearances, and similarly it is not always possible to distinguish blast lesions or hæmatomata from pneumonic lesions. On the other hand when the clinical and other evidence indicates the presence of intrathoracic infection, its localization and extent, at any stage in the course of the case can, as a rule, best and most accurately be judged by radiological examination.

I should like to discuss at this stage certain types of chest injury.

Pneumothorax.—It is neither necessary nor desirable to X-ray a patient with a "sucking" pneumothorax. The diagnosis is obvious, and temporary closure of the wound is an urgent necessity. When this preliminary therapy has been completed radiological investigation is of the same importance and value as it is in those cases of pneumothorax not dependent upon an open wound of the chest wall.

A small closed pneumothorax is not as a rule productive of serious symptoms, its presence can often be detected clinically and can nearly always be identified radiologically.

It is not generally realized that the dyspnoea caused by a pneumothorax bears little relation to the extent of the pulmonary collapse. A closer relationship exists between the degree of dyspnoea and the extent of the mediastinal oscillation which occurs with respiration. This phenomenon can best be recognized and its extent estimated by radioscopy.

The knowledge which X-ray examination will give of the extent of pulmonary collapse is, however, of some therapeutic importance, since the progress of the lung's re-expansion can be accurately followed in this way.

The relatively rare valvular pneumothorax which may easily and rapidly be fatal, can as a rule be recognized by clinical examination, and confirmed by measuring the intrapleural pressure over an interval of time. The radiological differential diagnosis has been discussed. In practice, in cases of chest injury, the conditions with which on clinical examination it is most likely to be confused are diaphragmatic hernia, and, when the left side is involved, atelectasis of a large proportion of the lung, which will occasion a correspondingly considerable rise of the diaphragm. Although clinical signs should often be decisive, these conditions may be distinguished from each other with certainty at X-ray examination.

A further important function of radiology when a pneumothorax is present lies in the fact that it will reveal the co-existence of other lesions, all clinical evidence of which may be obscured by the pneumothorax. Thus, for example, intrathoracic foreign bodies, atelectasis of the underlying lung, hæmatomata, and the presence of small amounts of fluid in the pleural cavity may, any or all, complicate pneumothorax and will be best demonstrated radiologically.

In cases of penetrating wounds pneumothorax will frequently be complicated by more or less extensive surgical emphysema which will mask the physical signs. The extent of both lesions can be shown radiologically.

Finally, therapeutic artificial pneumothorax may be used for the control of hæmoptysis occasioned by thoracic injury—more especially since prophylactic chemotherapy is available to diminish the risk of pyothorax the procedure might otherwise entail (Kretzschmar, 1940). The clinical management of such a pneumothorax may need frequent radiological control just as does therapy for the traumatically caused spontaneous pneumothorax.

Fractures of the thoracic cage.—The site and extent of fractures of the bony skeleton of the thorax can only be located accurately by radiological means, though clinical examination is often more informative in cases in which the sternum and costal cartilages are involved. At such an examination co-existent lesions will also be demonstrated and this becomes the more important since with a loose thoracic wall percussion loses much of its value (fig. 20). Hernia of the lung is as a rule more effectively demonstrated clinically.

Sternal fractures, and those of the costal cartilages and anterior ribs occasion gross mobility of the thoracic cage and give rise, just as does a sucking pneumothorax, to paradoxical respiration (fig. 21). Multiple rib fractures elsewhere may have the same effect. The condition is usually obvious by inspiratory recession of the chest wall, it is also revealed by screening at which examination the degree of the co-existent mediastinal oscillation will be demonstrated. At this examination the efficiency of therapeutic strapping can be gauged and so the decision as to the necessity of utilizing a Both respirator (Brooks, 1941) will be facilitated. Gunshot or other injuries to the bodies of the vertebræ, which may

give rise to continuous pain akin to that sometimes caused by pressure by an aneurysm may be localized radiologically—and the localization will have proportionately greater importance if consecutive involvement of the spinal cord has occurred.

Blast lesions and localized hæmatomata of the lungs.—Blast lesions of the lungs though quite extensive may give little or no clinical evidence of their presence, while even if such evidence of these lesions is forthcoming it is not in my experience in any way characteristic. Radiological examination is therefore of the first importance in the recognition of the disorder, moreover in this way only can its extent be demonstrated. Some concept of the gravity of the situation will thereby be obtained. In general it seems probable that the more extensive the damage the greater is the likelihood that the patient is approaching the limit of his respiratory resources, though I have encountered cases in which, in the absence of shock, respiratory embarrassment has seemed disproportionately great as compared with the radiological extent of their blast injury. Some of these patients have been shown radiologically to develop an increase in the pulmonary lesions during the next two days.

Patients with blast lesions of some severity tolerate general anaesthesia and surgical procedures poorly. Finally, the multiple hæmatomata to which blast gives rise are absorbed comparatively quickly.

From this knowledge it follows that all patients who have been exposed to the risk of blast should have a complete radiological examination of the chest at the earliest possible moment, and that the examination should be repeated until, as far as radiology can show, the lesions are absorbed. During this period, only in dire necessity will operation for the relief of such other wounds as the patient may have sustained be permitted.

Localized pulmonary hæmatomata following the forcible impact of any hard object against the chest wall may vary in size and density from a just perceptible radiological lesion to an apparent consolidation of a whole lung (figs. 22 and 23). Large or small, their clinical detection is uncertain, and I have been impressed by the infrequency with which bronchial breathing is transmitted even by peripheral hæmatomata of considerable size. Their demonstration and localization is simple and exact by radiology, and their relatively slow absorption can well be followed by repeated examinations of this kind (fig. 24).

Bronchopneumonic lesions may be confused with the effects of blast, while the pneumonias, infarct, fat embolism, and if the lesion is circumscribed, peripheral neoplasms and lung abscess may resemble hæmatomata. Taken in conjunction, the clinical and radiological findings, together with a study for a limited time of the course of the disorder are as a rule sufficiently decisive for diagnosis.

Pulmonary atelectasis.—It is as a rule possible to distinguish extensive pulmonary atelectasis from other lesions at clinical examination in an uncomplicated case. Chest injuries are, however, frequently complex, so that classical signs are given in a minority of cases. In such circumstances the value of radiology and radioscopy is great indeed, for atelectasis of the lung constitutes an emergency which needs early recognition and prompt therapy (fig. 25). The subsequent progress of the case can also best be followed radiologically. If in spite of bronchoscopy and other measures the atelectasis persists, bronchiectasis is likely ultimately to result. The site and extent of this disease can accurately be shown by bronchography—a procedure which is essential for its proper treatment.

Cardiovascular injuries.—Since the size, shape, and position of the heart can only accurately be ascertained by radioscopy and radiology, and since this knowledge is often of considerable value in many types of chest injury both cardiac and extracardiac, we are frequently for this reason in need of a radiologist's help. For example, a foreign body adjacent to the heart, particularly if it touches that organ's posterior surface, may set up marked delirium cordis although little or no cardiac damage has been sustained. The value of the localization of the foreign body, together with the ascertained increased probability of anatomical normality of the heart in such a case is manifest. Foreign bodies in the heart will likewise be revealed. Cardiac contusion on the other hand often presents no abnormal radiological appearances. Gross cardiac irregularity occurs not infrequently if sudden severe mediastinal displacement takes place, and to the irregularity is added severe impairment of circulatory (and respiratory) function if mediastinal oscillation co-exists. X-ray examination will provide the explanation for what may otherwise in these circumstances be a very confusing condition. Cardiac failure should usually be detected clinically, but if doubt exists the diagnostic help radiology will give in demonstrating an alteration in shape and size of the heart and increased pulmonary vascular congestion may justify its use.

The physical signs over the heart in the presence of a pneumothorax, especially if surgical emphysema is a complication, are sometimes bizarre in the extreme. In such a case as a rule it seems probable that pericarditis, or that a pericardium containing air or fluid or both, is present. Radiology can then do much to establish whether the heart is normal or not.

Similarly, though the peripheral and local consequences of severe pericardial lesions of the above types give rise to well-recognized physical signs, exact diagnosis and the early institution of appropriate therapy are greatly facilitated by radiological investigation. Radiology may similarly help in showing the site and size of vascular complications of chest wounds such as aneurysms of various kinds including arteriovenous aneurysm.

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The second point was that many of the films shown that afternoon appeared to have been cut in half, and the section often appeared at a point of clinical importance. This abuse of the film was often carried out by the Ministry of Pensions, and it would be helpful if they could find some other way of treating the film so that it would go into a smaller compass. Rolling the film into a cylinder took up less space and was less mutilating.

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FIG. 1.—Foreign body in mediastinum; lying just anterior to the arch of the aorta, removed at operation.

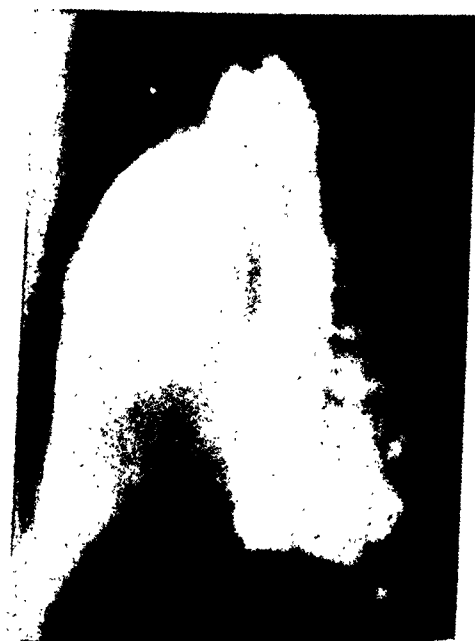


FIG. 2.—Tomogram showing three fractured vertebrae in dorsal spine. Not seen on X-ray examination with portable apparatus.

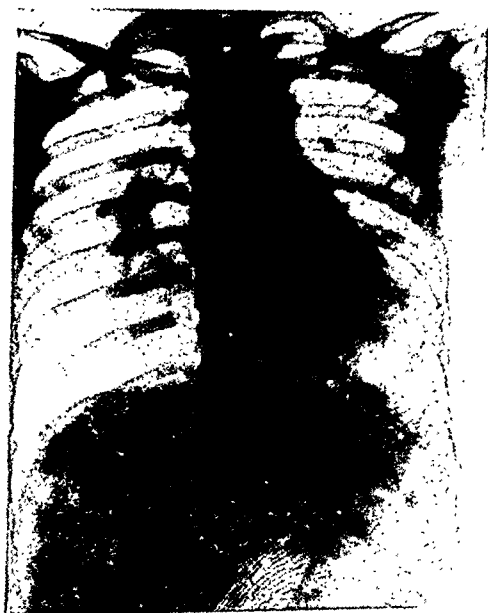


FIG. 3.—Tension pneumothorax associated with fractured ribs.

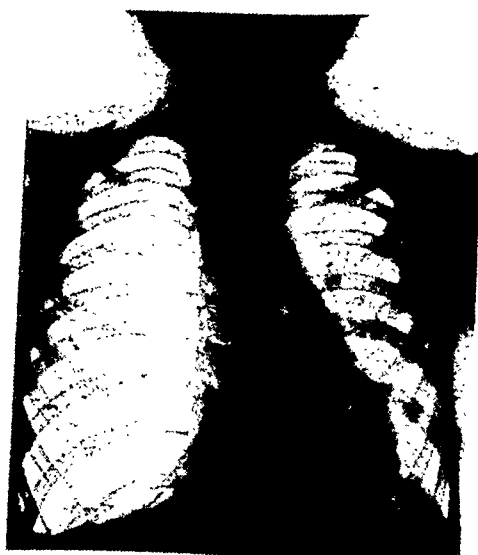


FIG. 4.—Giant cyst simulating pneumothorax.



FIG. 5.—Traumatic diaphragmatic hernia simulating pneumothorax.

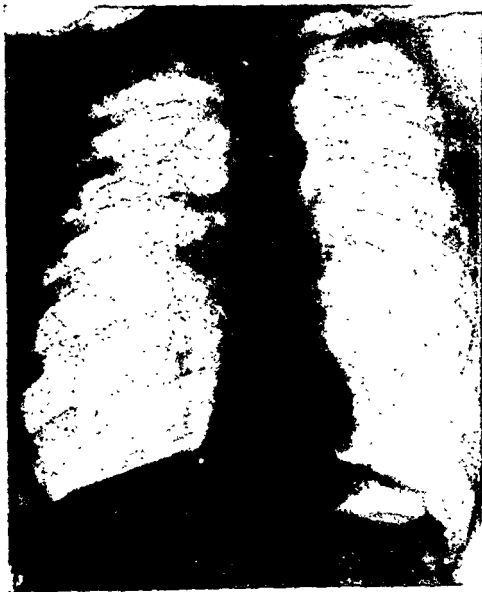


FIG. 6.—Emphysematous bullæ simulating pneumothorax.

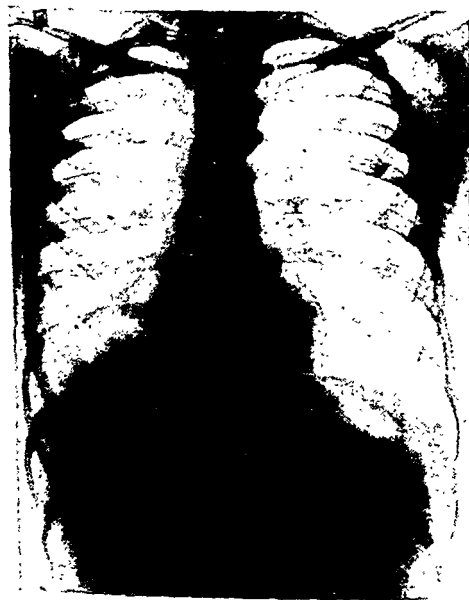


FIG. 7.—The same case as fig. 3 after re-expansion. There is a partially collapsed lower lobe.



FIG. 8.—Bilateral blast injury of lung.



FIG. 9.—Haematoma of lung with small extrathoracic foreign body.

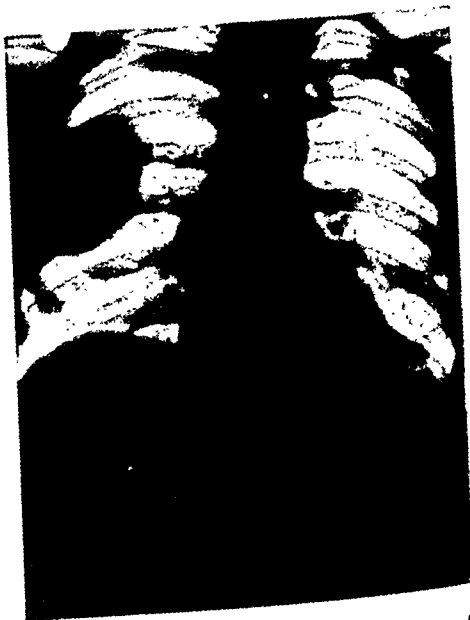


FIG. 10.—Unusual form of haematoma of lung which did not resolve.

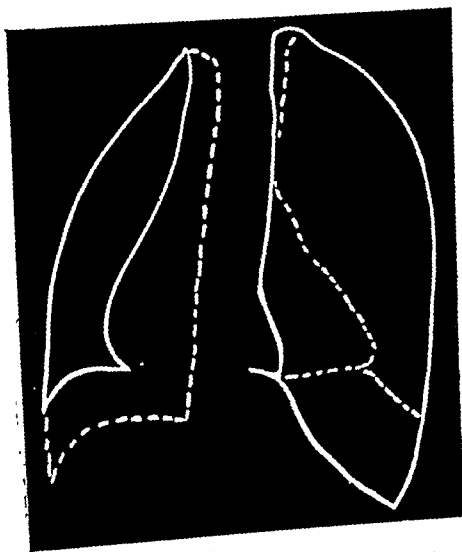


FIG. 11.—Superimposed tracings to show mediastinal displacement with patient lying on right and left sides respectively. The continuous line shows the patient on the right side, the broken line the patient on the left side.

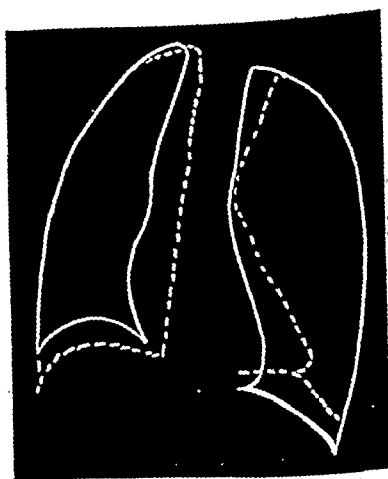


FIG. 12.—Superimposed tracings to show a mediastinal displacement with patient supine and on the left side. The continuous line shows the patient supine; the broken line shows the patient lying on the left side.



FIG. 13.—Bomb fragment close to heart with associated h matoma, h emothorax, and subcutaneous emphysema.



FIG. 14.—Lateral view of same case. Typical of films taken with portable apparatus shortly after injury.



FIG. 15.—Needle in base of left lung. This produced symptoms only after thirty-two years. It has been removed.



FIG. 16.—Foreign body in posterior part of left chest—about the smallest size suitable for removal.



FIG. 9.—Hamatoma of lung with small extrathoracic foreign body.

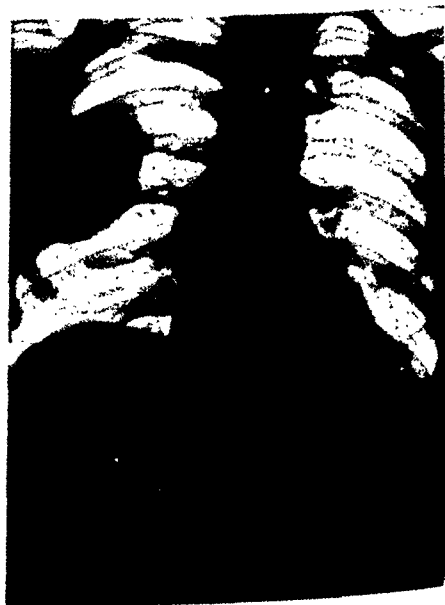


FIG. 10.—Unusual form of hematoma of lung which did not resolve.

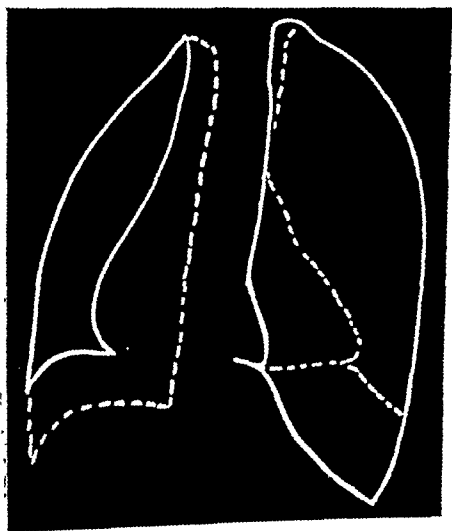


FIG. 11.—Superimposed tracings to show mediastinal displacement with patient lying on, right and left sides respectively. The continuous line shows the patient on the right side, the broken line the patient on the left side.

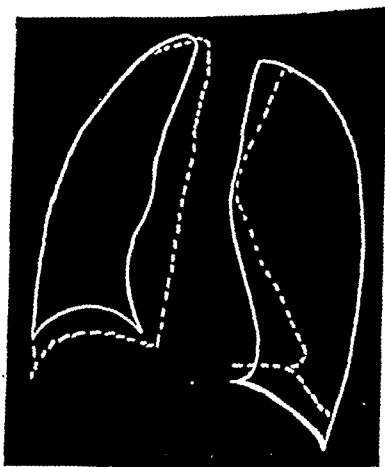


FIG. 12.—Superimposed tracings to show a mediastinal displacement with patient supine and on the left side. The continuous line shows the patient supine; the broken line shows the patient lying on the left side.



FIG. 20.—Crush injury producing fracture of upper left ribs with hemothorax and hematoma-formation in the left chest.

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FIG. 22.—Stove-in chest with opacity almost entirely due to hematoma and simulating hemothorax.



FIG. 23.—Same case some time later after partial resolution of hematoma.

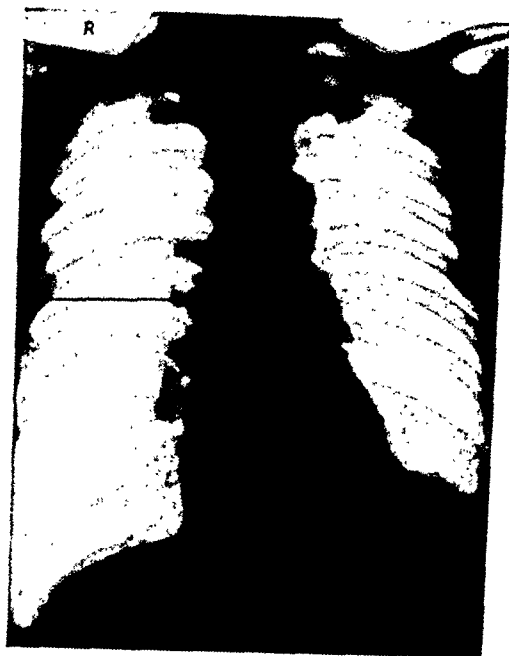


FIG. 17.—Small residue of blood-clot at the left base remaining after complete aspiration of a haemothorax which was produced by a minute foreign body overlying the region of the first interspace anteriorly.



FIG. 18.—Untreated haemothorax showing diminution in size of the right hemithorax; thickened visceral and parietal pleura.



FIG. 19.—Pleurogram to show imperfectly treated pocket at apex.

Section of Comparative Medicine

President—G. DUNLOP-MARTIN, M.R.C.V.S.

[October 21, 1941]

DISCUSSION ON THE GENERAL PRINCIPLES INVOLVED IN A CAMPAIGN TO CONTROL CERTAIN DISEASES OF CATTLE INIMICAL TO MAN

Mr. H. W. Steele-Bodger: The control of disease in the dairy cow is important in order to preserve the health of the animal itself and so extend its usefulness and to prevent the transference of infection from the animal to man. It is true, of course, that some infectious diseases from which cattle suffer are not inimical to man, but those which are, and which happen also to cause much ill-health among the animals themselves, have been chosen as subjects for discussion at future meetings. These diseases are responsible for immense financial loss to agriculture and to the country, and cause much wastage of essential foods.

Concerning clinical tuberculosis in humans, the expressed medical point of view is that a decreasing but still far too high proportion of cases is directly traceable to the cow secreting tubercle bacilli in her milk. It is estimated that 0.5% of lactating cows actually suffer from tuberculosis of the udder. Milk is also blamed as the medium by which *Brucella abortus* is conveyed to man. Though it is not possible to ascertain the incidence of undulant fever in this country, it is probable that many cases are not reported and that some go undiagnosed. Milk again is held to be responsible for not a few of the streptococcal and staphylococcal conditions which affect human beings.

The solution offered by Medicine is compulsory pasteurization; this is undoubtedly commendable and beneficial, but it does nothing to solve the problem at its source—in fact, pasteurization alone rather connives at a perpetuation of the existing condition from the point of view of the milk producer. It produces artificially a "safe" milk, if the process is carried out efficiently. A healthy disease-free cattle population would not only supply naturally safe milk, it would eliminate a loss to agriculture of millions of pounds annually and thus would make more animals available for consumption at a cheaper price—a thing greatly to be desired in the present age of malnutrition.

What is the veterinary reaction to the problem? So far attempts have been made to minimize the risk of infection of milk by tubercle or other organisms by means of clinical examinations carried out under the Milk and Dairies Acts and Orders, the Milk (Special Designations) Order, and the Tuberculosis Order (a procedure which is but a palliative measure). The other present alternative is the attempted eradication of tuberculosis by means of the much-criticized tuberculin test. When the problems associated with the standardization and specificity of tuberculin are solved, how much further forward shall we be in the control of tuberculosis? Are there still some amongst us who believe that what is possible to achieve in the rearing district of, for example, Wales,



FIG. 21.—Fractured lower sternum produced by crush injury.



FIG. 24.—Crush injury producing haematoma towards the right base with petechial haemorrhages through lung field and subcutaneous emphysema.

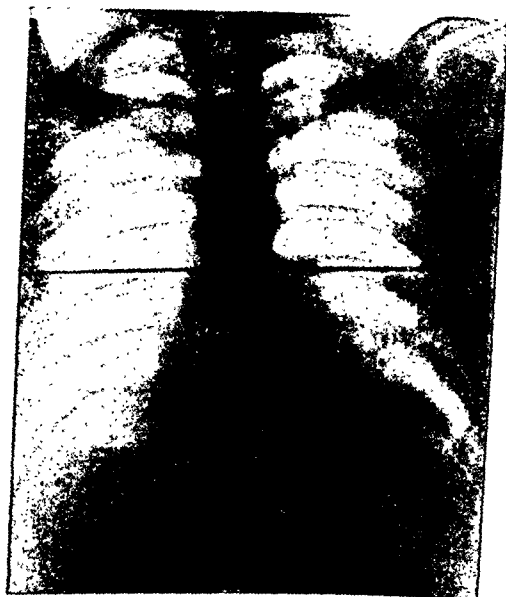


FIG. 25.—Bilateral basal atelectasis following penetrating injury by small bomb fragment which is lying in the middle of the left chest.

Perhaps the only group of diseases ripe for eradication is the acid alcohol-fast group comprising mammalian and avian tuberculosis and Johne's disease. Weybridge mammalian tuberculin seems to be a diagnostic agent for the group.

One would need more evidence than has yet been produced to believe that brucellosis can be controlled by the use of live vaccines. Temporary infertility at the outset is indistinguishable from sterility of a more permanent nature, and if remedied by the measures suggested will leave a balance of 30% of cows still awaiting further treatment. Mastitis due to *Streptococcus agalactiae* appears to be entirely absent from some countries. Clinical mastitis due to other causes is more difficult to control and it is here that the clinician will need all the assistance of our expert laboratory technicians.

Mr. G. N. Gould said the diseases enumerated by the opener were the scourge of agriculture. In regard to tuberculin testing, no one could pretend that the present position was satisfactory to the practising veterinary surgeon or to the dairy farmer, nor would it be until a tuberculin could be devised free from the present criticisms. While it was possible to appreciate that the building up of tuberculin tested reservoirs of cattle had certain advantages, he could not see that the present scheme contributed materially to the control or eradication of bovine tuberculosis, since reacting animals were passed back into circulation.

In regard to *Brucella*, the difficulties of eradication were known to all veterinary surgeons who had attempted it. The eradication policy for abortion appeared to depend upon a test which was by no means efficient. Under present farming conditions particularly, eradication of this disease had little or no place and the building up of resistance by immunization was the only common-sense policy. A standardized live vaccine of known virulence was essential.

Professor T. J. Bosworth said that he had hoped to hear what were the general principles involved in the campaign to control these diseases so that an opinion might be formed as to their soundness. Unfortunately the opener had failed to enlighten them and had seemed to suggest that the principles for dealing with each disease might be formulated in the discussions which had been arranged for subsequent meetings of the Section.

Such discussions would be valuable, but a scheme for dealing with these diseases had already been produced and it was being put into operation; it did not command unanimous approval, for many felt it could not produce the results which had been claimed for it. He was inclined to agree with a previous speaker that more progress might be made by concentrating on the control of a smaller number of diseases, but, however that might be and taking into account the urgency of doing all possible to increase meat and milk production, he was of the opinion that nothing was more likely to hamper progress than making promises to the farmers which could not be fulfilled.

Mr. S. J. Edwards said that if success in the control of contagious diseases of cattle was to be achieved, close co-operation between the farmer and the veterinary surgeon would have to be maintained. He instanced the voluntary tuberculosis eradication scheme initiated in Ayrshire in 1930 where practically complete freedom from infection was achieved in an area containing thirty dairy herds.

The incentives provided in this case were free tuberculin testing and advice on methods of eradication; in turn the farmers gradually disposed of reactors and, where necessary, renovated their premises. The fact that freedom from disease was aimed at in one group of herds ensured complete co-operation of the farmers in the area.

With the other contagious diseases of cattle the best results would also be attained by similar methods, although it might not be feasible to control more than two diseases at a time.

Dr. H. J. Parish: It has been stated (C. F. Brockington, *Brit. M. J.*, 1937 (i), 667) that probably three-quarters of the milch cows in England would at any one time be found to be excreting one or more of the following three organisms in virulent form: *Mycobacterium tuberculosis*, *Br. abortus* and *Streptococcus*. This may be an exaggeration, but it indicates the magnitude of our problem.

Milk-borne diseases fall into two categories—the infections of cattle and the conse-

is also possible in the intense milk-producing areas such as Cheshire? Are we not pursuing a shadow as many did for so long in attempting to eradicate contagious abortion from this country by blood testing and the isolation and elimination of reactors to the agglutination test? It is, I think, now generally agreed that the control of contagious abortion by vaccination with a reliable vaccine is the only practical method in a country such as ours, whilst the present system of farming obtains. I believe that the same holds good with regard to the control of tuberculosis. Research workers and clinicians will have to strive to perfect a vaccine which will confer reasonable immunity upon the inoculated animal, whilst taking every precaution to ensure that it cannot prove harmful to the human being. The effect which the control of tuberculosis in cattle would have upon the health of the human population in this country is incalculable.

Brucellosis is a major scourge of cattle and is probably indirectly responsible for more than half the cases of infertility in bovines. In the U.S.A., in an attempt to control brucellosis by means of the agglutination test alone, 2,000,000 reactors to the test were slaughtered between 1934 and 1939. The disease is now controlled by calf vaccination and the agglutination test before an animal enters the herd.

Trichomoniasis in bovines is also becoming a major problem in this country and I understand the incidence of this disease in humans is increasing, though I am not aware of definite evidence that there is any link between the infections in humans and bovines.

Mastitis, particularly that due to *Str. agalactiae*, is another major scourge of bovines, and it has recently been estimated that mastitis, contagious abortion and sterility are responsible for an annual loss of approximately 200,000,000 gallons of milk, equivalent, with milk at 1s. 3d. a gallon, to an annual loss of £12,500,000; as milk is now about 2s. a gallon, the loss is £20,000,000. If we include the loss of the calf crop due to these diseases we have a further loss of £1,500,000, whilst the loss due to replacement of dairy cows affected with these three diseases is to-day in the region of £5,000,000 per annum. Our total loss is then in the region of £27,000,000. This is not the whole picture. It is known that in milk affected with streptococci, the solids-not-fat content is markedly reduced and that the butter-fat from affected quarters is reduced by 25%. In the aggregate there is a loss to the nation annually of over 83,000 tons of a solid mixture of protein, sugar and minerals, and a loss of over 31,000 tons of butter-fat.

In considering these problems one must bear in mind the present system of farming in this country. Improved animal husbandry and better methods of hygiene should be aimed at; we should endeavour to reinforce the natural resistance of the animal and to employ such methods of immunization as are available. Control on such lines is preferable at this time to the elimination of these diseases and the isolation of healthy animals from the chances of infection. The day may come when such methods can be practised on a large scale with success, but until their incidence is lower, control, rather than elimination, must be the rule.

In collaboration with the Ministry of Agriculture and the National Farmers' Union, the National Veterinary Medical Association has elaborated a scheme for the control of mastitis, contagious abortion, and infertility; this scheme will be discussed at the other meetings during this session.

Dr. C. L. Oakley: The diseases of animals inimical to man are divisible into two classes: those directly infectious to him and those which, by reducing his food supply, may render him more susceptible to infectious disease. Apart from sterility, young animals, with few exceptions, are born free from disease; the main problem is, therefore, one of animal husbandry. The problem has a serious economic side also—farming must pay, and a method of immunization which protected every cow from disease but at the same time increased the price of meat beyond the capacity of the public to pay would obviously be a failure.

Mr. J. R. Barker: The first principle after the admission of an evil in society is the formulation of a remedy and propaganda for its acceptance. The National Veterinary Medical Association Scheme has not yet received sufficient help from the Press in its campaign for the control of certain diseases of cattle inimical to man.

Animal husbandry based upon veterinary science and bovine ecology is the next essential. Animal husbandry varies throughout the country and herd owners need advice founded upon local knowledge and given by experienced clinical workers.

culture. The relatively lower value of cattle in New Zealand compared with Great Britain certainly facilitated more drastic measures; compensation payable to owners was on a commensurate scale. Mastitis was a most formidable problem and widespread infertility constituted another serious obstacle to the dairying industry as a whole.

Apart from the general interest which the scheme under discussion would arouse, interest also centred around the important trade in pedigree livestock. It would add to the confidence of prospective importers to feel that organized control of the diseases under consideration would shortly be undertaken.

Dr. W. R. Wooldridge said the scheme referred to by Mr. Steele-Bodger arose out of the work of a special Committee of the N.V.M.A. In a report issued an estimate of the losses resulting from mastitis, contagious abortion, sterility (temporary abortion) and Johne's disease was given. This loss, considered to be about £20,000,000 annually, was sufficiently serious—particularly in time of war—to justify, in the opinion of many authoritative bodies, a scheme for the control of the four diseases concerned. Even though some of the knowledge of the particular disease did not appear to be absolutely perfect, yet it was felt to be sufficiently proven to be of service in bringing about a reasonable control which would lead to a material reduction in these losses.

In brief this scheme depended upon a contract arranged between a veterinary surgeon and his client, whereby the practitioner undertook for an agreed fee the control of the four diseases specified, it being understood that the government would grant special facilities to those entering into these contracts. These facilities included certain free laboratory assistance, the free issue of a standardized vaccine for the control of contagious bovine abortion, and the supply of sulphanilamide at a very much reduced cost.

Although this scheme was not under official government supervision, it must be regarded as a semi-official scheme and not an entirely *ad hoc* scheme, depending solely upon individual arrangements between practitioner and client.

A misunderstanding seemed to have arisen concerning the claims of those supporting the proposed scheme. This misunderstanding assumed that those advocating the scheme believed that practically the whole of the huge loss mentioned would be saved if the scheme were introduced, whereas, in fact, no such claim had ever been made. But it was felt that a useful reduction of such losses could be obtained by this scheme and that it would pave the way to more effective schemes later on.

Like other speakers, Dr. Wooldridge was disappointed that the discussion had not ranged more directly round the "Principles Governing the Methods of Control of Animal Diseases". There were two important principles which seemed worth mentioning: (1) The scientific knowledge upon which a method of control of any disease of animals is to be based must be such that it has reasonable prospects of giving beneficial results, even though it may not lead to a control sufficiently complete to make early eradication of the disease possible; and (2) as the practice of animal husbandry in its widest sense is essentially an economic problem the application of any method for the control of animal diseases must be economically sound. This was so whether one considered it from the point of view of the State or the individual owner. The limitations of these two principles were obvious and needed no elaboration at this stage.

Dr. Wooldridge said that he was not clear as to how the professions of human and veterinary medicine could intimately collaborate in the control of diseases of animals except indirectly as a result of a greater appreciation on the part of the one of the difficulties and rather different outlook of the other. Essentially the doctor must insist that food-stuffs of animal origin were safe for human consumption, whilst the veterinary surgeon's aim was to develop and maintain healthy animal stock so that wholesome products could be obtained free, so far as possible, from all disease-producing organisms.

The controversy which had for some time raged over the pasteurization of milk arose out of a lack of appreciation of these differences in outlook and was due to the fact that local authorities, in endeavouring to carry out their obligations to their public, had different points of view put to them as how best milk could be delivered safe and wholesome to the consumer.

Medical Officers of Health naturally insisted that the product must be safe and, they therefore felt that the best means of achieving this at once was pasteurization; whereas agriculturists and veterinary surgeons, appreciating that the product produced on the farm was in those days often of poor quality, wished to institute methods for the more

quent excretion of organisms in milk and the contamination of milk during and after its withdrawal from the cow. Apart from well-recognized infections, cattle may transmit to man many undiagnosed and undiagnosable fevers.

The ideal procedure for the prevention of disease would involve absolutely healthy cattle, well housed and groomed, well fed and tended by healthy men, cleanly in their milking methods; contamination should also be avoided between cow-shed and consumer. Such an ideal is impossible largely for economic reasons and because healthy human carriers of streptococci, &c., would be difficult to control.

In the control of tuberculosis, much can undoubtedly be done by efficient tuberculin testing; but if pastures and premises are heavily contaminated, eradication of the disease by this means would be extremely difficult and expensive. Vaccination against tuberculosis has been mentioned; in experiments with B.C.G. in guinea-pigs, death of the infected animals can be delayed but survival is almost unattainable. Better results have been achieved with a vaccine of Wells' vole bacillus, but much more work is necessary.

There are gaps in our knowledge of *Br. abortus* infection. If large numbers of cows are infected and excreting *Br. abortus* in their milk, why should the disease in man be so uncommon? Presumably many infections are overlooked, or possibly the majority of *Br. abortus* strains of cattle have little power of infecting man.

Many outbreaks of staphylococcus food poisoning have been associated with unpasteurized milk or cream.

There are two lines of advance: (1) We should do all we can to remove sources of infection from herds. (2) Milk should be protected by efficient pasteurization. It should be bottled by machinery and delivered in sealed containers.

Dr. C. L. Oakley said that he was not certain that the ideal was perfectly healthy animals if that meant herds entirely free from infection. Infectious disease had both an immediate and a remote effect; it might infect man directly, but more important it might not only reduce the immediate yield by the death or slaughter of infected animals, but also seriously reduce the number of breeders. The death or slaughter of an infected animal meant the loss also of its potential offspring. It was useless to carp at farmers for not using methods of eliminating disease which would lead to bankruptcy. Consequently, though some diseases might be controlled in isolated areas, the ordinary transport and marketing of animals must introduce infected animals from less favoured spots; and infection-free herds would surely prove extremely susceptible to such infection. Isolation involved economic problems of profound importance; and it seemed doubtful whether elimination of disease by slaughtering could be done without serious reduction of our food supply. More attention should be paid to immunization against disease whereby the originally uninfected animal might be kept safe from the ordinary risks of its relatively short life.

Mr. Geo. Dunlop-Martin said the most important point in his opinion was that the scheme mentioned by the opener would lead to early access to animals affected with these diseases. It must of a certainty mean that very many cases would be saved. Thus the "end product" of this early access must also mean more food for the nation—especially meat and milk—at this time and for the coming years of the utmost importance to the wartime food problem.

Lieut.-Colonel H. A. Reid said the problems now under consideration were similar to if not identical with those obtaining in New Zealand. It was estimated, however, that not more than 10% of the cattle of New Zealand were affected with tuberculosis. This was probably due to climatic conditions enabling livestock to graze throughout the year. They were never housed.

There were in Britain many slum animal habitations, and any scheme for control of bovine tuberculosis should regard this factor as vitally important in contributing to the spread of infection.

New Zealand authorities had not hitherto favoured the extensive use of either living or dead vaccines as a means of combating bovine abortion. The Dominion Veterinary Service had a free hand in animal health. It operated independently of the medical profession in the control of animal disease which might be communicable to man. Meat inspection was handled entirely by the veterinary division of the Department of Agri-

Questions had been raised as to the ripeness of the time for a nation-wide scheme on the principles proposed by the N.V.M.A. There was admittedly much yet to learn but the present need was great and a considerable body of knowledge was available which could be used at once. One outstanding principle of the scheme put forward was that of early access to the farm, and on a herd basis. That control of disease in that way was advantageous and practicable was already proven. Much more could be achieved by formal and regular access to a larger number of farms as part of a planned scheme available to all.

One real danger of a short-term scheme could be foreseen and met: that was the danger that it could become too cut and dried. But if it was regarded as only a beginning, if use was made continuously of the material and experience which would thereby become available to increase the common fund of knowledge, and if pre- and post-graduate education were kept abreast, an intensive short-term effort could be merged into a long-term policy of continually increasing efficacy and breadth.

Mr. Steele-Bodger (in reply to the discussion) said he was glad to hear the medical members declare that the most important general principle in the control of animal disease was improved animal husbandry. If all our herds and flocks could have free range, disease control would be much easier than at present, but it would appear inevitable that in certain parts of this country animals must be housed at certain times of the year. He did not agree with Mr. S. J. Edwards that pulling down old buildings and putting up new ones would solve the problem of disease in dairy herds.

The area method, so successful in the United States, was not possible in this country whilst the present system of farming obtained.

The problem of disease control or eradication was stressed by Dr. Oakley when he spoke of the susceptibility of a disease-free herd to reinfection and he was sure that the policy to adopt in this country was to endeavour to reinforce resistance to disease rather than to attempt to eliminate it.

Dr. J. T. Edwards (Pirbright) said that after some years of experience at home and abroad in the study of animal diseases, and of the history of their control, he wondered whether any general principles could be enunciated. Particular problems depended upon so many various factors—of which not the least important was the availability of suitable weapons for control—that each needed to be grappled with according to its particular circumstances.

Viewing the general problem of cattle disease, mainly as an ecologist, four or five ill-defined strata could be discerned: Firstly, the epizootic diseases, of virus, protozoan, or bacterial origin, which, when they occurred in a given territory, were so damaging that they usually demanded drastic action by the State. Secondly, the enzootic diseases, mainly of bacterial origin, which again yielded fairly readily to preventive measures of control. Thirdly, the insidious diseases, in which bacterial infection was either the primary cause or in which the virulence of a subpathogenic bacterium was greatly enhanced thanks to intensive domestication. In this stratum fell largely the bacterial diseases, such as tuberculosis, mastitis, and contagious abortion. Perhaps the helminthic diseases could also be included in this stratum. Fourthly, the diseases caused by dietary errors or deficiencies and disturbances in hormonal equilibrium; these again were largely the outcome of domestication and forcing the animal by selective breeding to yield products for the use of man far in excess of what had been intended by nature. Sterility seemed to fall mainly within this stratum, as did milk fever. So ill-defined and interlocked were the third and fourth strata in this picture that there came to mind the recent brilliant work of Shope on the multiplicity of "insults" required to give rise to a natural outbreak of influenza. A disease like bovine mastitis, which for some time had appeared to be caused solely by certain bacterial agents, would now seem to have a more complex aetiology, with, in the phenomena of evolution and involution of the mammary tissue, factors of the fourth stratum acting as contributory agencies. Likewise, the bacterial metritis associated in a preponderance of cases with sterility would possibly be found to be determined primarily by faulty involution of the genital tract conditioned by disturbance in the normal hormonal equilibrium during this phase in the reproductive cycle. Fifthly, although not so evident, but still real, especially in highly bred dairy stock, were the culminating conditions of intensive domestication represented by the neuroses; the

efficient production of sound and safe milk. In considering these two points of view local authorities all too frequently regarded them as alternative schemes whereas they should have been complementary; often medical and veterinary advisers found themselves in opposite camps and made the mistake of not collaborating to bring home to the local authority that efficient production on the farm was complementary and not an alternative to pasteurization. In his view the veterinary profession must realize that at present efficient pasteurization of milk must be insisted upon by the medical profession who should appreciate on the other hand that improved methods in the production of clean and safe milk upon the farm were equally essential in the interests of the consumer.

It was quite clear that unless the officer responsible for the quality and safety of foods of animal origin, whether he belonged to the medical or veterinary profession, realized that the points of view of the consumer and of the good producer were not in fact in conflict but were collateral, progress in this sphere would be considerably retarded.

Dr. A. D. McEwen said he agreed there was a danger in the eradication of a disease from individual herds whilst it remained enzootic throughout the country.

Unfortunately conditions in Britain were not so favourable for the control of tuberculosis as in the United States. Nevertheless the wholesale methods followed in the States had encouraged this country to concentrate a large section of its veterinary service on attempts to eradicate tuberculosis from a number of individual herds. These efforts assisted those interested in the herds but did not benefit the population as a whole, as the milk supply was made no safer, nor was the price reduced, thereby encouraging greater consumption. Furthermore, the interest devoted to eradicating the disease from a number of herds had probably prevented adequate attention being given to immunization of the cattle population against tuberculosis.

With regard to contagious abortion, the United States' policy of slaughtering female cattle that reacted to the agglutination test, fitted in with the economics of the "New Deal", which aimed at a reduction in animal produce in order to raise prices for farmers. The slaughter of the 2,000,000 cattle referred to by Mr. Steele-Bodger had not effected the control of the disease. In Britain the incidence of *Br. abortus* in cattle was probably higher than in the United States, and it appeared that at present the greatest hope of control lay in immunization. The general opinion was that animals could only develop slight immunity to *Br. abortus*, but it had been shown that animals infected with a virulent strain of *Br. abortus*, whether still infected or recovered from infection, possessed a remarkably solid immunity to superinfection or reinfection. This should encourage investigations on immunity.

He agreed with Dr. Parish that as man contracted certain contagious diseases from the ingestion of raw milk, it should be pasteurized. Pasteurization need have no adverse influence on dairy hygiene or on efforts to control disease in cattle.

Dr. J. M. Alston said he was glad to hear Dr. McEwen as a veterinarian encourage pasteurization of milk, for opposition between veterinary and medical workers on this question was unnecessary. The primary task of the medical profession in this was to protect human health and, if a safe supply was obtainable, to encourage the drinking of more milk—thus encouraging an increased supply of milk from healthy cows. *Br. abortus* did appear to be of low virulence for human beings, but pasteurization affected the question, because in London nearly all of the few people infected with *Br. abortus* whom he had met had taken unpasteurized milk shortly before their illness.

Dr. A. W. Stableforth said that earlier speakers had referred to two main points of contact between the medical and veterinary professions on this subject: the danger from animal diseases communicable to man and the importance of the increased milk supply to be expected as a result of intensive measures for the control of disease in milk herds. The danger from cattle diseases communicable to man could, as already said, be effectively dealt with by proper pasteurization, and he believed this to be fortunate for another reason; it enabled control efforts to be focused on those diseases of major agricultural importance which caused the greatest lowering of milk yield. Milk was one of the essential foods—particularly for children, pregnant mothers and invalids; figures showed that much more milk would be available if some of the more common cattle diseases could be controlled. The relative values of various methods of control varied according to the disease.

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Viewing the general problem of cattle disease, mainly as an ecologist, four or five ill-defined strata could be discerned: Firstly, the epizootic diseases, of virus, protozoan, or bacterial origin, which, when they occurred in a given territory, were so damaging that they usually demanded drastic action by the State. Secondly, the enzootic diseases, mainly of bacterial origin, which again yielded fairly readily to preventive measures of control. Thirdly, the insidious diseases, in which bacterial infection was either the primary cause or in which the virulence of a subpathogenic bacterium was greatly enhanced thanks to intensive domestication. In this stratum fell largely the bacterial diseases, such as tuberculosis, mastitis, and contagious abortion. Perhaps the helminthic diseases could also be included in this stratum. Fourthly, the diseases caused by dietary errors or deficiencies and disturbances in hormonal equilibrium; these again were largely the outcome of domestication and forcing the animal by selective breeding to yield products for the use of man far in excess of what had been intended by nature. Sterility seemed to fall mainly within this stratum, as did milk fever. So ill-defined and interlocked were the third and fourth strata in this picture that there came to mind the recent brilliant work of Shope on the multiplicity of "insults" required to give rise to a natural outbreak of influenza. A disease like bovine mastitis, which for some time had appeared to be caused solely by certain bacterial agents, would now seem to have a more complex aetiology, with, in the phenomena of evolution and involution of the mammary tissue, factors of the fourth stratum acting as contributory agencies. Likewise, the bacterial metritis associated in a preponderance of cases with sterility would possibly be found to be determined primarily by faulty involution of the genital tract conditioned by disturbance in the normal hormonal equilibrium during this phase in the reproductive cycle. Fifthly, although not so evident, but still real, especially in highly bred dairy stock, were the culminating conditions of intensive domestication represented by the neuroses: the

phenomenon was far more evident in another domesticated species—the canine—by the present wide prevalence of canine hysteria.

The chief problem in this country to-day was to combat those conditions that lay deep in the third and fourth strata. The imminence of the diseases in the uppermost strata was made to appear remote thanks to the stringent official action taken to safeguard the bovine population from them. Only as far back as the sixties of the last century rinderpest became after an interval a major disease problem of cattle, and it was only eradicated after the State, paying heed finally to veterinary, rather than medical, advice, had spent about £5,000,000 sterling in applying the drastic "stamping-out" methods which had since come to be known as "cattle plague measures", and which were to-day those currently employed, for want of a proved better system, for the control of foot-and-mouth disease.

Mr. H. T. Matthews said that he believed that we were hardly in a position to talk about *eradication* of the diseases but should think first in terms of *reduction* to controllable proportions, and, in that sense, might give the vaccination policy a chance before instituting more radical measures. That is to say we needed a fairly long-term plan aiming at reduction of incidence before the longer-term policy of complete eradication.

He agreed that the diseases under discussion fell into a class where animal husbandry became important. As most calves were born by design and were born healthy, if we sheltered them from the hazards of disease and enhanced their resistance artificially, we should be able to produce a much more healthy generation within a measurable period. It seemed quite clear, in relation to these diseases, that the work of the laboratory, of the clinician and of pure animal husbandry must be combined into one policy based on team work.

Section of Otology

President—F. W. WATKYN-THOMAS, F.R.C.S.

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Otitic Meningitis

PRESIDENT'S ADDRESS

By F. W. WATKYN-THOMAS, F.R.C.S.

WHEN Charles West left St. Bartholomew's twenty years ago, he suggested to some of us that the two outstanding problems of otology awaiting solution were the mechanism of the auditory focus, and the treatment of otitic meningitis.

Auditory focus, I fear, is still a vague blur somewhere just over the edge of the otological subconscious, but in the treatment of meningitis a great—a really great—advance has been made. How great that advance has been we may not fully realize. Statistics are convincing, but still more convincing is the simple fact that twenty years ago the most retiring surgeon felt it his duty to mention the survival of a single patient in whom otitic meningitis had progressed to the extent of organisms in the cerebrospinal fluid. Some six hundred years ago a public-spirited philosopher, one Ockham, forged a weapon which should be in the hand of every doctor. He called it his razor. It was a formula, a simple formula, and it ran thus:

“Entities must not be multiplied.”

So I shall say that by otitic meningitis, I mean the reaction of the pi-arachnoid system to an advancing infection from the middle ear and its adnexa. “Meningism,” “serous,” “bacterial” or “suppurative” meningitis, “encysted” meningitis—are all stages of a single process as modified by virulence or resistance.

Meningitis is not particularly dangerous because organisms are present in the cerebrospinal fluid; it is the other way round; the organisms are present in the fluid because the meningitis is particularly dangerous; because the resistance has been overcome, and the infection has reached its most dangerous stage. For this reason I avoid the statistical subclassifications. They only make a difficult problem more difficult.

If we accept, as we are bound to, this view of meningitis, it is clear that the principles of treatment are the principles applicable to every advancing suppuration; elimination of the focus, drainage, and the appropriate counter-medication, serum treatment or chemotherapy as the case may be. And here may I remind you of the fact, so obvious that in the past it has often been ignored, that the patient who dies of meningitis is killed by septicaemia, not by increased intracranial pressure. The principles are simple enough—they usually are; how exactly to apply them has been the difficulty—it usually is!

A short time ago a colleague of ours, realizing like Mr. Michael Finsbury that “there’s nothing like a little judicious levity” in a delightful essay, advised us to study the great

detectives of fiction. Such a study should make us sympathize deeply with their unhappy biographers, who were always being exhorted to apply "my methods" or to exercise their little grey cells without any hint as to how they were to set about it. We have been too often in their position—especially in the treatment of meningitis.

In one striking instance the application of the principles of elimination and drainage was rewarded by brilliant success. Invasion of the pi-arachnoid space by suppuration extending through the labyrinth was dreaded as the most formidable attack of all. The infection of the cisterna pontis by way of the internal auditory meatus resulted in a spread of infection so swift and so massive that it was almost invariably fatal. The fear of it was one of the greatest hindrances to any surgical procedure on the labyrinth.

Nevertheless, the condition was most suitable for resolute surgery. The infecting focus, the labyrinth, could be thoroughly and easily eliminated; adequate drainage at the site of infection was provided by a ready-made non-collapsible drainage tube, the internal auditory meatus. In 1908 West and Sydney Scott did the first translabyrinthine drainage and saved the patient; the surgical principles were vindicated. Unfortunately the method is rigorously limited; it is invaluable for labyrinthine infections, but only for labyrinthine infections, and with the advance of mastoid surgery such infections have become steadily less frequent.

Our difficulties in applying the principles have been anatomical and physiological, especially in regard to the irreproachable neutrality of the choroid plexus, which obstinately refused admission to all therapeutic remedies offered to it.

Between the anatomical and physiological problems we can draw no rigid boundary, for structure is only the machinery of function. Still, for convenience we may say that the major difficulties of elimination of the focus are anatomical; the major difficulties of meningeal drainage, physiological.

ELIMINATION OF THE FOCUS.

Apart from extirpation of an infected labyrinth, drainage of a leaking brain abscess, or clearance of an infected venous thrombus, the orthodox elimination of focus was a mastoid operation, done as thoroughly as possible, with exposure of the dura of middle and posterior fossae.

The brain abscess that leaks is usually one that has already doomed the patient; an infected sinus thrombus is usually detected before meningitis starts, and is operated on for its own sake. As Eagleton, Kopetzky, and Marx have shown, in the majority of cases the track of infection is through the petrous bone, and reaches the perivascular sheath by a retrograde venous thrombosis, and it is to Eagleton that we owe the demonstration of the method of attack. Extradural, intradural (venous sinuses) and pial veins all communicate. These last, the veins of the cortex and cerebral substance, are endothelial tubes, with no adventitia except the arachnoid mesh, save for a short collar of fibrous tissue where they enter the sinuses. At what Eagleton calls "the points of venous vulnerability" they are in close contact with bone which may become infected. If once organisms enter these endothelial tubes they can readily pass through the walls and into the arachnoid mesh, into the perivascular space, that double cuff of pi-arachnoid which surrounds and accompanies the vessels and is the channel of the cerebrospinal fluid. The most dangerous of these "points of vulnerability" are (a) a small venous plexus on the upper surface of the petrous in the angle between the superior and lateral canals. (Here we should remember that Frenckner has described a series of cases in which he followed a line of infected cells through the arch of the superior canal up to the apex; also, that all of us have at some time or another, found considerable difficulty in elevating the dura from the bone in this region.) (b) The vein of the subarcuate fossa. (c) The petrosal vein in the internal auditory meatus. (d) An inconstant plexus perforating the dura and entering the cellular bone near the apex (Pietrantonio), and (e) at the point of communication of the cortical veins and the sinus, especially around the upper edge of the petrous.

To these "vulnerable points" we must add another. Steurer states that in the infant there is direct vascular communication between the dura and the submucosa of the middle ear. Normally these vessels undergo involution during the process of pneumatization and are reduced to strands of connective tissue, but if pneumatization is arrested they persist. According to Witmaack there is one at the tegmen and another below the angle of the petrous pyramid near the dura of the sinus. This makes a diagonal link

from the antrum up to the sinus angle. It may be from 2-7 mm. long and as thick as a "knitting needle." It is most usually found when the mastoid is dense and the cells are few. I have encountered this vessel in a well-pneumatized mastoid, and had some difficulty with it.

This leads us to a brief consideration of the petrous itself.

The petrous is laid down in cartilage, which in embryonic life is largely replaced by red marrow. When pneumatization advances the red marrow is converted to yellow, but faced by infection it can revert again to red. Mass figures show complete pneumatization in 35% of adult petrous bones; marrow is present in more than 90%. It can be stated, generally, that where pneumatization is complete, the freedom of communication between blood-vessels of the dura and bone is largely abolished, so that danger of meningitis is diminished, although infection can travel more easily through the bone. When pneumatization does not occur the dural communications are more persistent, but the red marrow (which, as has been said, reforms in the presence of infection, even after it has advanced to the yellow stage) is capable of an admirable resistance. In neither case are necrosis and sequestration of the petrous surface common; in pneumatized bone there is excellent blood supply by vessels of the subendosteal layers; in the non-pneumatized bone by vascular loops in the free spaces of the marrow. These facts account for the frequency with which petrous infections clear up with the simplest adequate drainage, and the comparative rarity of meningitis as a complication of aural suppuration. For we must remember that it is, fortunately, not a common complication.

Eagleton, in 1929, advocated an operation for "unlocking" the petrous. Almour and Kopeztzky devised an operation for draining the petrous apex. Ramadier and Frenckner suggested others. These attacks on the apex are more adapted for the prevention of meningitis than for its cure, but they have all added to our knowledge of the surgical possibilities of the petrous bone, and have helped greatly to simplify the first of our tasks—the elimination of the focus.

In the establishment of drainage we failed for a long time because of our faulty tactics. We sought an anatomical approach, and ignored the physiological aspect. We tried to drain accumulations, rather than to re-establish the normal circulation of the cerebrospinal fluid.

In one instance, as I have said, we had a brilliant success; that was in translabyrinthine drainage for meningitis of labyrinthine origin. But here many factors were in our favour. The very fury of the meningeal invasion drove us to early operation: to operate before the fluid was heavy with protein. Looking back on our results, we may ask whether the real reason for our success was not the perfect elimination of focus provided by the operation; not only elimination of focus in the bone, but also prompt removal of a massive dose of highly infected fluid in the cisterna pontis. We must remember that the arachnoid mesh in the internal meatus is extraordinarily fine; so fine, that some localization of infection is likely to happen here; Politzer describes a "chronic abscess" of the internal meatus. Another valuable aid is the free flow of fluid into the labyrinth, which must help to wash out any organisms lurking in the bony recesses. It was often noticed that when there was no free rush of fluid on breaking through into the internal meatus, the operation usually failed.

George Jenkins worked out a method of intrathecal lavage which, in his hands, sometimes gave excellent results. Unfortunately, the difficulties of an elaborate technique prevented its general adoption. Some patients survived after mastoidectomy and continuous lumbar drainage. Drainage of the cisterna magna was often tried. As far as I know, no patient survived this. Incidentally, it is not conclusive proof that because a patient survives an operation, that operation has necessarily saved him.

Incisions of the membranes and the insertion of drainage tubes into the lateral cistern were useless. In the belief that the great difficulty of drainage was due to loculation by adhesions, some surgeons tried to break them down by intrathecal injections of air and of acetylene. A few good results were claimed (as I have suggested, the power of survival of some patients is remarkable), but after a short time nothing more was heard of the method; which speaks for itself.

Our tactics were all wrong. Dixon and Halliburton had shown us that the cerebrospinal fluid is formed by the choroid plexus. Weed, and his co-workers, proved that it is absorbed into the blood-stream by the arachnoid villi, and if the arachnoid villi are

blocked absorption is prevented. We know, too, that the fluid not only passes from the ventricles into the main pi-arachnoid spaces, it also percolates the whole brain substance by way of the perivascular sheaths.

As meningitis is an inflammatory reaction, the cerebrospinal fluid is quickly loaded with cells and coagulable protein, which block the exits of the arachnoid villi, and also obstruct the perivascular sheaths of the deep vessels. The physiologists know the answer. Massive intravenous injections of hypotonic saline increase the flow of cerebrospinal fluid. Kubie applied this clinically and showed that it was possible to wash out the meninges from within, removing the surplus by repeated lumbar taps. This method has the further advantage that it dilutes the protein-laden fluid; that the pi-arachnoid space is kept open by the free flow, and the risk of loculation is diminished. Further work along these lines should solve the difficulty of drainage.

COUNTER-MEDICATION

Here the difficulty was not of our making. The choroid plexus put up a barrier which was, for a long time, insuperable. The only chemical antiseptic which it would sanction was hexamine, and that was quite useless. Hexamine only acts as an antiseptic when it breaks down, and it does not break down in the cerebrospinal fluid.

Sera and antitoxins were rejected unhesitatingly by the plexus. Intrathecal injection of antitoxin succeeded often in tetanus and meningococcal meningitis, but the same method failed pretty consistently in suppurative meningitis.

Intrathecal or intracarotid injections of substances such as mercurochrome and bile-salts may possibly have destroyed the organisms; unfortunately they usually also destroyed the patient.

A more promising line of attack was again suggested by the work of the physiologists. As hypotonic saline increases the flow, so hypertonic saline intravenously, or even massive depletion by magnesium sulphate enemas, will draw off cerebrospinal fluid (probably to the extent of reversing the current in the perivascular spaces) and shrink the brain. As we know, the Monro-Kellie doctrine lays down that the contents of the cranial box must always remain the same in bulk, so the loss of cerebrospinal fluid must be compensated by an increased blood volume. Accordingly attempts were made by salt-depletion and intravenous transfusion of immune blood, to overwhelm the resistance of the choroid in a "thirsty" brain. This work was still in progress when the discovery of the sulphonamides gave us a powerful antiseptic which could traverse the barriers, and so solved the outstanding difficulty of chemotherapy.

THE MANAGEMENT OF A CASE OF OTITIC MENINGITIS

The fact that a patient has otitis and meningitis is not proof that the meningitis is due to the otitis.

There is one group of cases, usually in children, when meningeal symptoms and acute otitis media are practically simultaneous. These fulminant cases are not, in my opinion, cases of otitic meningitis. They are blood-borne invasions which strike both the meningeal spaces and the middle ear. It is probable that the organisms to a large extent reach the meninges by way of the middle-ear vessels, through the preformed paths which exist in the petrous temporal, but the meningitis is not, strictly speaking, otogenous or otitic.

If we accept this view in such cases, we will refrain from surgery in the early stage, because there is no focus in the temporal bone to eliminate, and pin our hopes on chemotherapy. If the drum bulges, we shall incise it, and if, later on, we find a definite mastoid suppuration, we shall regard it as a fixation abscess and drain it as such. But the rules of treatment laid down for otitic meningitis do not apply, because these cases are not due to middle-ear infection.

Again, it is not impossible for a patient with an otitis media to have, independently or otherwise, meningococcal or tuberculous meningitis. The cerebrospinal fluid will give us the evidence.

Further, a patient with otitis media may have, often does have, accessory sinus suppuration, and there is always the possibility that his meningitis may be due to his sinus suppuration, not to his ear.

The diagnosis of established meningitis is generally easy. The signs are those of acute and increasing intracranial pressure with severe toxæmia.

Usually the onset is sudden, after a day or two of vague and ominous discomfort. It is our ambition to detect the condition at its earliest, and not to await the classical syndrome—which, as is often the case with classical syndromes, is the prodrome of approaching death.

Headache and pyrexia are the first and the most constant warnings. We can make a clinical rule that no patient with an uncomplicated mastoid suppuration has any right to have a headache. He may have pain in the ear or radiating from it, but pain not so localized should always raise the suspicion of intracranial irritation. Pyrexia is nearly always an early sign. Slowing of the pulse is uncommon. Some stiffness of the neck is usual and early, but it is not always found even in advanced cases. Probably the severity varies with the extent of invasion of the posterior fossa.

Some congestion of the discs is common and early. My personal experience is that it is present in a considerable majority of cases. Vertigo and nystagmus are not unusual, even in cases where there is no labyrinth lesion. I have never been able to fit them into any set pattern to my own satisfaction, or to correlate them definitely with other findings, but Eagleton, whose opinion no one can afford to neglect, definitely states that:

(1) Absent response to the cold caloric test of the opposite ear in the upright position, with positive response in the horizontal position, is produced by increased pressure.

(2) Absence of response to the cold caloric test with good hearing is due to exudate in the pi-arachnoid prolongation around the 8th nerve.

(3) Irregular nystagmus with good hearing indicates posterior fossa effusion.

(4) Irregular attacks of vertical nystagmus indicate effusion around the bulbar region.

Focal signs, when present, are usually caused by a localized collection. These collections enlarge fairly rapidly, and as they enlarge they leak, for the barrier adhesions are weak and imperfect. The commonest site for them is said to be either above the tegmen, or in the posterior fossa, internal to the bend of the lateral sinus. Above the tegmen they may cause quadrant hemianopsia by pressure on the optic radiation. I have seen such a collection in the region of Broca's convolution give an excellent imitation, from the neurological aspect, of an increasing middle meningeal hæmorrhage.

The determining fact in our diagnosis is the result of the lumbar puncture, and lumbar puncture should be done in every case where there is the least suspicion of meningeal invasion.

It should be done carefully, for there can be no doubt that sudden death has followed excessive withdrawal of fluid, although we do not really know how, exactly, death in these cases was caused. Also, it is probable, that excessive withdrawal of the fluid, if bacteriæmia is present, can actually induce a meningeal invasion. The slow withdrawal of 2-3 c.c. can do no harm, and that amount is quite enough for the pathologist.

We want information, above all, about the cell content; then the bacteriology if any. The albumin and sugar content are more interesting than important. The estimation of chlorides is more valuable for prognosis than for diagnosis. The lower the chloride content, the worse are the patient's chances. We want to diagnose and treat the condition before the chlorides fall.

In special cases diagnosis is made difficult by causal or concomitant conditions.

(1) Septic thrombophlebitis of the lateral sinus seldom causes meningitis unless it is left untreated for far longer than is usual now. Even in such untreated cases, cerebellar abscess is the more usual sequel. But sinus thrombosis and meningitis do sometimes occur together, and at quite an early stage.

I have seen three such cases in the last two years. In all of them the focus of infection was in the petrous. In two of them the thrombus was at the junction of the superior petrosal and lateral sinuses. Probably in all three cases thrombosis and meningitis were both due to the petrous infection, not to each other. Cases have been reported in which sinus thrombosis accompanied suppurative labyrinthitis (I have seen two such cases), and in such cases there is a great risk that the sinus condition may be overshadowed by the more urgent and dramatic signs of meningeal invasion.

It is a possibility which we must always remember, for however efficiently we deal with the petrous or the labyrinth, however vigorously we administer sulphonamides, all will be in vain if we overlook a spreading infection in the lateral sinus.

(2) Brain abscess: It is reasonable to assume that a brain abscess starts like any other abscess, with exudation of fluid and infiltration of leucocytes. It starts, in fact, as an encephalitis and it must be accompanied by meningeal reaction. Borries has shown that

a clearing fluid and increasing symptoms are diagnostic of brain abscess. If, at the first suspicion of encephalitis we attacked the focus and treated the condition as we should treat a meningitis, we would stand a good chance of arresting the further development of an abscess.

The leaking brain abscess is quite different. It is uncommon, but it happens. Dan McKenzie and Broughton Barnes described cases in which recurrent attacks of meningitis were caused by cerebellar abscesses. A cerebral abscess which leaks into the ventricle is nearly always fatal, but Jenkins saved one by intrathecal lavage. In these cases, the abscess usually develops silently, and is only found when the onset of meningitis demands operation.

(3) Labyrinthitis and meningitis: If a patient with labyrinthine suppuration, total loss of hearing on the affected side and nystagmus of destruction, gets pyrexia and a headache, he has got meningitis. The patient with latent labyrinthitis (so-called) is more difficult. In plain English "latent labyrinthitis" means that the patient has had an attack of suppurative labyrinthitis, which has been diagnosed as a "bilious attack" or "gastric influenza", and has recovered with a dead (and infected) labyrinth. If months, or years, later the organisms invade the meninges by the internal auditory meatus, there will be no warning labyrinth storm. The first signs will be the signs of meningitis.

In this way, too, a patient who has suffered labyrinth destruction from a fractured base may get a meningitis without warning from an acute otitis media and invasion through the unclosed fracture line.

In every case of meningitis we should investigate the labyrinth by the caloric test and hearing tests (making sure for these tests that the opposite ear is efficiently masked by a noise box or other method).

(4) Petrositis: The difficulty here is that we may pay too much attention to the localized 5th nerve pain around and behind the eye, and too little attention to the fact that the patient is getting more generalized headache. Pyrexia and extension of headache are our warning signs. When we find them we should certainly do a lumbar puncture and probably we should operate.

TREATMENT.

Our diagnosis is made, and as far as possible that diagnosis has included the source of infection.

It is frequently said that "sulphonamides have revolutionized the treatment of otitic meningitis." This is an unhappy example of that chronic looseness of statement for which our profession is so justifiably notorious. Sulphonamides have done nothing of the kind. They have enormously improved our results, but the principles of treatment are the same. What has happened is that we have another, and most valuable, weapon to our hand.

The sulphonamides are most efficient in the tissue fluids and the blood; they are at a disadvantage in bony spaces, and they are almost powerless against a collection of pus. Our first duty is still to eliminate the focus. As Jansen said many years ago: "The first sign of meningitis is the last call to operate."

In meningitis of labyrinth origin I feel that the right course still is to open and drain the labyrinth, and to complete the operation by breaking through into the internal auditory meatus. With further experience of chemotherapy we may be able to modify this. We know that sulphonamides freely enter the cerebrospinal fluid, and that there is free communication between the cerebrospinal fluid and the perilymph. In fact, the labyrinth is a bony space in which, against the usual rule, sulphonamides should be able to act well. For this reason, we may hope that in the future many cases of labyrinthitis will be cured by chemotherapy, and that the need for opening the labyrinth will be exceptional. But when labyrinthitis has advanced to meningeal invasion, I do not think we can take any risks until we know far more. There is a definite focus and that focus must be eliminated. The final step—opening the meatus—is simple and rapid, and we should take it. The stakes are too high for us to omit any precaution.

The actual method which I have usually followed is that of West and Scott—opening the vestibule and the external canal, and then entering the internal meatus through the fundus of the vestibule. If this is done with a small, very sharp, gouge, held firmly without tilting, and if we are careful to avoid any pressure against the upper border of the oval window, it is usually possible to avoid the facial nerve. A few strands of silkworm gut can be put into the meatus for drainage, but if the flow of cerebrospinal fluid is encouraged by intravenous hypertonic saline, I do not think it is necessary.

In the majority of cases the only evident focus is the infected middle ear and mastoid. Here our right course is to explore the petrous by a route which will give us a good exposure of the lateral sinus and the dura of the posterior and middle fossæ. The methods of drainage suggested by Almour and Kopetzky, Frenckner and Ramadier, apply to collections localized in the petrous, and are unsuitable for the extending infection. If we have any clinical evidence as to the state of the petrous, so much the better. If an expert radiologist is available his help may be most valuable, but once meningitis has appeared we are not justified in delaying operation to get a skiagram.

Unless the labyrinth is involved, a radical mastoid is not usually needed. I start with a wide epitympano-mastoid operation and free removal of the outer attic wall, and clear the cells as far as possible before exposing the dura. The lateral sinus is exposed up to and beyond the entrance of the superior petrosal, then the tegmen is removed and the squamous temporal freely resected. Next the bone of Trautman's triangle and the posterior margin of the petrous are attacked. My greatest difficulty is always in stripping the dura over the superior canal. I have found the curved-on-flat end of a Hill's septal elevator the most comfortable weapon. Another spot which is sometimes difficult is the outer extremity of the petrous angle. A suboccipital puncture sometimes helps to make the brain more manageable. I have never done a ventricle puncture as a help to the operation, but I see no reason why one should not do so in a suitable case. There can be no risk of infecting the brain tissue thereby, as the brain is already percolated by the infected cerebrospinal fluid.

The dura is stripped up to the apex and posteriorly to the lip of the internal meatus. It can then be thoroughly examined—the dura of the cerebellum on the posterior petrous aspect should be inspected too. Unless there is an encysted collection of fluid or evidence of an abscess, the dura should not be incised. It is quite useless to do it for drainage, as the brain sits down firmly in the hole and blocks it. The upper and posterior surfaces of the petrous are carefully examined, and any softened or inflamed bone is cut away. Any abscess in the bone can be opened with a curved curette. If pus is found a rubber dam drain should be put in and the wound kept widely open with rubber packing.

During the operation pieces of bone should be put into broth for the pathologist. As far as possible these pieces should be blood free, because blood will hinder the growth of pneumococci. A swab from the nasopharynx will often reveal the causal organism.

Also, during the operation, an assistant should give an intravenous injection of half-strength normal saline, about 500 c.c. for an adult; a lumbar or suboccipital tap should be done and as much cerebrospinal fluid as possible drawn off.

Chemotherapy should be started as soon as the vomiting from the anæsthetic has passed. If the organism is known the appropriate sulphonamide should be given. If it is not known, pending the pathologist's report, it is probably well to give sulphapyridine, which has wider activities than sulphanilamide. It should, of course, always be given for pneumococcal infections; for streptococcal infections sulphanilamide seems better.

To get the best results from sulphonamide treatment we should have the help of the physician and the pathologist. We must know what the organism is; what is the concentration of the drug in the blood serum and in the cerebrospinal fluid; and we must watch the blood-count and the urine. Too often, as far as chemotherapy goes, we fight with one hand tied behind our backs because, before he comes to operation, the patient has endured a long, futile and dangerous medication with too small doses, perhaps of the wrong drug, spread over too long a period. His symptoms have been masked, his white cells already lowered, and he has already had so much of the drug that we cannot risk the heavy loading dose necessary for saturation.

After-treatment consists of full sulphonamide dosage, lumbar puncture as long as headache persists, and hypotonic saline intravenously until the cerebrospinal fluid has cleared.

Complications in our field are unusual, but there is one, and a new one, which occasionally occurs. That is, recurrence of meningitis after the patient is apparently cured. I have seen one case; in this the attack was more spinal than cerebral, and was probably due to the rupture of an encysted collection in the spinal theca. The patient recovered on sulhapyridine. The original lesion was a pneumococcal petrous, plus superior petrosal sinus invasion.

To sum up: The cardinal principles of the treatment of otitic meningitis are surgical intervention; the establishment of drainage and the application of chemotherapy. By

the combination of these three, our results have been enormously improved, and we can hope for still greater improvement.

Discussion.—Mr. E. D. DAVIS said that in his Address the President stated that patients died of septicaemia and not of the local condition of the brain; but many patients died from a disturbance of the medullary centres, hence the Cheyne-Stokes breathing. In the early stage of septicaemia before there were actual signs of meningitis, organisms might be found in the blood, but at a later stage these organisms disappeared and that fact made the diagnosis of a blood-borne infection uncertain. The earliest possible diagnosis of threatened meningitis was most important. Severe headache following an operation for acute mastoiditis with a rise of temperature was one of the early signs. Infection spread from unopened retro-facial air-cells to the cancellous bone surrounding the bony labyrinth then through the posterior wall of the petrous bone to the posterior fossa. This route was more common than petro-itis which, in his experience, was very rare.

The speaker was satisfied that rigidity was due to the spread of suppuration to the interpeduncular or basal cistern. There were cases of meningitis on the cortex of the brain with no rigidity at all and if one waited for rigidity the chance of saving the patient would have gone. Rigidity in his experience was a late sign and he had not seen a case of really marked rigidity recover. He was sure it was not an early sign. An early diagnosis can be obtained by repeated examination of the cerebrospinal fluid. If after eradicating the septic focus by a complete mastoid operation exposing the dura of both fossae the cerebrospinal fluid showed that the meningitis was progressive then he would perform a translabyrinthine drainage operation.

Mr. T. B. LAYTON said that he was quite in agreement with Mr. Davis about rigidity in the meningitis of chronic ear disease. In that condition one should not wait for rigidity to appear. But in meningitis of the acute ear disease one should not wait for anything else. That was the difference between the two.

Mr. MUSGRAVE WOODMAN said that he was interested in the President's opinion that these cases, when they died, died from septicaemia. He wondered whether that applied also to pneumococcal meningitis. Some of the cases of pneumococcal meningitis, if not all of them, started from the ear, and they gave the otologist the greatest trouble because of the loculation within the meninges.

He had never had occasion to do the translabyrinthine operation, although he once had a case which was clearly labyrinthine in origin—a simple mastoid which ten days after operation suddenly lighted up with violent vomiting and acute nystagmus. He sent the case to Sir William Milligan, who operated with some slight help from himself. The patient recovered, but with a total and complete facial paralysis.

He thought that the regimen laid down by the President was likely to eliminate the operation of translabyrinthine drainage. Provided the same result could be obtained without that operation, he was of opinion that it should not be done. So far as chemotherapy was concerned they were badly handicapped by the very small doses administered before the surgeon had a chance of seeing the patient.

Major SCOTT STEVENSON said that it was quite wrong to state that chemotherapy properly administered had not revolutionized the treatment. The ineffective amount of chemotherapy that so many patients received was quite a different story. Chemotherapy should come first and surgery second, but the chemotherapy ought to be under the direction of the otologist. The chief point was early diagnosis. He wondered what was the criterion in the diagnosis of meningitis. When he looked up the cases recorded by Neumann, for example, he found that he had 22 recoveries out of 59 cases, but of these 22, 16 showed a sterile cerebrospinal fluid, and the question was whether they were really cases of true meningitis or not. The first essential was early diagnosis, the second was chemotherapy, and the third was removal of the septic focus after chemotherapy had been started in adequate doses.

Mr. TERENCE CAWTHORNE agreed that chemotherapy should be instituted before the operation and he added that if circumstances permitted, the drug should be given the chance of reaching the optimum level in the blood before operating. They had all seen cases in which the complications of the otitis media had been cloaked by insufficient doses of the drug, and it had been shown that in some cases small doses increased the resistance of the organism to the drug. Thus it was important to institute chemotherapy rigorously and thoroughly, and he endorsed the President's suggestion that cases should always have the serum and cerebrospinal content of the drug estimated. He felt that the concentration to be aimed at in meningitis was 10 mg. per 100 c.c., and this meant large doses. The possible danger of large doses should be no deterrent when dealing with a disease from which the patient would probably die unless large doses were given.

From the surgical angle there were three types of otitic meningitis: (1) Meningitis which occurred in association with the virgin ear; (2) meningitis which occurred with the chronic ear; and (3) meningitis which occurred in the previously operated ear.

With meningitis and the virgin ear, he felt very strongly that no surgical procedure, apart from paracentesis should be undertaken. If more than this were done, fresh avenues of infection were likely to be opened up. In the case of meningitis in an ear that had been suppurating for two or more weeks there was the probability of diseased bone keeping up the infection, and it would be necessary to undertake an extensive operation on the lines suggested by the President. Drainage of the internal auditory meatus he thought should be only undertaken for those cases in which the infection had clearly spread from a previously dead labyrinth. He had occasion to treat a patient some few months ago who had meningitis and a history of long-standing ear discharge and a dead labyrinth. At operation an obviously long-established fistula in the external canal leading to a pus-filled cavity was found. The history suggested an attack of labyrinthitis some eighteen months previously. This case was drained through the internal auditory meatus and made a complete recovery. Two days after the operation he gradually developed a facial paralysis which passed off in six weeks.

Of the third class of case, namely, patients who had recovered from previous meningitis and got a second attack he had seen two examples. Both had been treated in the original attack by a combination of chemotherapy and surgery, and they both recovered from the second attack on chemotherapy alone.

It is likely that this class of case may become increasingly frequent.

The decision to withhold surgery in otitic meningitis was often difficult to make and only an otologist was qualified to make it.

Major C. A. HUTCHINSON put in a plea for early diagnosis with bacteriological control of the infection, because he, too, was certain that the appropriate form of sulphonamide was much more effectual to the particular infection present than one chosen at random. Sulphonamide was often used irrespective of whether the infection was streptococcal or pneumococcal and in useless minimal doses.

At his hospital a bacteriologist was brought in at the earliest possible moment to decide which sulphonamide should be used, and this was started at once if possible. Meanwhile appropriate surgical treatment was adopted. As soon as the patient came round from the anæsthetic, the proper sulphonamide treatment as predetermined was at once recommenced and massive doses were given. Two signs of intolerance had been noticed—more so in some types of sulphonamides than in others—i.e. certain blood dyscrasias and gastritis.

Major T. A. CLARKE felt that the earlier part of the discussion might lead to some unnecessary surgery, particularly in early infections. Surely, he said, the purpose of surgery was to deal with localized suppuration and to drain it; unless the case was one of chronic aural suppuration or of acute otitis media with established suppuration, surgery was not indicated. They had all had cases with meningeal reaction in which they had widely opened the mastoid at an early stage of otitis media, the patient getting better; but it was possible that the patient recovered in spite of and not because of the operative interference.

Dr. HUGO FREY said he did not believe that there was any essential difference between acute and chronic cases from the point of view of their meningitic consequences. In the chronic cases—and this held good not only for meningitis but for other complications, as, for example, sinus thrombosis—the complication which appeared was a consequence not of the old infection but of an acute exacerbation, due to a new superadded infection, or, if they preferred, the sudden development of a new virulence in the existing bacteria, the reason for which was not known. If operation were carried out on a case of chronic suppuration in which there were some evidences of complication there would be found almost always in the operation signs like acute changes in the mastoid bone. Therefore he was of the opinion that from the ætiological aspect as well as the therapeutic outlook, there was no real difference between the acute case and the chronic case which, as soon as it produced a complication, came under the same heading as the acute.

The PRESIDENT said he was not convinced that the rigidity of the neck had anything to do with the bacteriological aspect. He felt that this, as Mr. Davis had pointed out, was a question of the anatomical distribution of the main fluid mass and of the site of greatest pressure, but confessed that he had not thought of its relation to the interpeduncular space. Mr. Woodman had spoken of the possibility of loculation. He himself had a case of recurrent meningitis from a pneumococcal petrositis, but the recurrence appeared during an air-raid over the week-end and a full examination of

the cerebrospinal fluid was not made. The organisms looked like pneumococci, but they were not cultured or grouped on the second occasion. The patient recovered on sulphapyridine. Major Scott Stevenson believed in chemotherapy first and surgery later. The great objection to that was that the pathologists and the chemists told them that chemotherapy was at its very worst in a bony cavity, and in the bony spaces where they were at work the sulphonamides were practically powerless against a collection of pus. He was all in favour of using chemotherapy and of sticking to chemotherapy in cases where there was no focus to eliminate; but where there was a focus they could only give chemotherapy a fair chance by first eliminating that focus. From that point of view the question raised by Mr. Cawthorne about starting with chemotherapy before attacking the focus did not arise. Otitic meningitis was a surgical emergency, and there was no time to start the sulphonamides. He preferred to get rid of the focus straight away—it made a difference only of a few hours—and then proceed to the sulphonamide administration.

He too had seen a recurrence in previously operated cases. He had seen only one case of labyrinthine invasion in an old mastoid cavity apparently healed; all the other cases of recurrent mastoiditis with meningitis had been petrous infections. One of the first petrous infections that he recognized and treated as such was a case of this kind. It was a boy who had been operated on successfully four years previously and then had a recurrence of discharge and meningitis: an abscess was found in the petrous far back on the posterior margin. A similar thing happened in a recent case. A patient who had undergone a successful Schwartze mastoid operation turned up about five years later with recurrence of discharge, and violent headache, which rapidly passed into meningitis; he developed a lateral sinus thrombosis as well, but made a good recovery.

The President agreed with what Major Hutchinson had said about the need of identifying the organism they had to attack if the sulphonamides were to be given a fair chance. One speaker (Major Scott Stevenson) had questioned whether Neumann's cases were really meningitis, because a large number of them had a sterile cerebrospinal fluid. If a patient had got an increase of pressure, an increase of cells, and a headache, he had got meningitis, and he did not see why the surgeon should wait until he had got organisms as well before making a diagnosis. Meningitis was a clinical and a pathological entity; a group of symptoms associated with a reaction of the meninges against an infection. If that infection could be treated while the meninges were still able to put up a good resistance so much the better for both the otologist and his patient.

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Section of the History of Medicine

President—J. F. HALLS DALLY, M.D.

[November 5, 1941]

The Foundation of the Polish Medical Faculty within the University of Edinburgh, Scotland

By Professor A. T. JURASZ

Dean of the Polish Medical Faculty

THE PRESIDENT, in introducing Professor Jurasz, said the Section was celebrating a historic occasion. Professor Antoni Jurasz was born in Heidelberg. He was a son of Professor Jurasz who held the Chairs of Laryngology in the Universities of Heidelberg, Lwow and Poznan. His mother was Caroline Gaspey, a daughter of Dr. Thomas Gaspey, lecturer in English in the University of Heidelberg. He studied at Heidelberg, London, Königsberg, and Leipzig so that he had had a large experience. Before the war Professor Jurasz was President of the Society of Polish Surgeons, President of the Society of Surgeons and Orthopedists of Western Poland and President of the Red Cross of Poland. He would tell them of the foundation of the Polish Medical Faculty at Edinburgh University, the first Polish medical school to be established outside its own country, which, it had been hinted, would be the precursor of a good many more. The occasion was unique and historic because of this fact.

STRESZCZENIE REFERATU, WYGŁOSZONEGO PRZEZ PROF. DR. A. JURASZA w dniu 6.XI.1941 r. w Royal Society of Medicine, w Londynie

Diękując na wstępie za zaszczyt zaproszenia go do Royal Society of Medicine dla wygłoszenia referatu w Sekcji Historii "Royal Society of Medicine", prof. dr. A. Jurasz zaznacza, iż mniejwięcej przed 12 laty miał już zaszczyt przemawiać w tym Towarzystwie na temat, zaczerpnięty z jego specjalności, a więc chirurgii, a właściwie—urologii. Obecnie tematem referatu jest historia powstania Polskiego Wydziału Lekarskiego w Edynburgu.

Po naszkicowaniu okoliczności, w których we wrześniu 1939 r., Polska, napadnięta przez wojska Hitlera, po bohaterskiej obronie, uległa przeważającej militarnej sile niemieckiej, prof. Jurasz przechodzi do bardziej szczegółowego omówienia tych systematycznych i celowych przesładowań, którym ulegają od dwóch zgorą lat, nauka i kultura polska na ziemiach okupowanych przez najeźdźców.

Zamknięcie wszystkich szkół wyższych i średnich, obrabowanie wszystkich zakładów naukowych i bibliotek w Polsce, uwięzienie setek profesorów uniwersytetów w Krakowie, Warszawie, Lwowie, Wilnie i Poznaniu—wszystko to w oświadczeniu Generalnego Gubernatora ziem okupowanych, Franka, znalazło następującą morywację: "Naród Polski nie potrzebuje wykształcenia. Elementarne szkoły są najzupełniej wystarczające. Wyższe zakłady naukowe w Polsce nigdy nie zostaną ponownie otwarte. Polska natomiast będzie "pustynią intelektualną". Postępując w myśl tej barbarzyńskiej zasady Niemcy usiłują wypłenić w Polsce to wszystko, coby reprezentuje jej wiedzę, jej kulturę duchową, jej sztukę i literaturę. O systematyczności, z jaką Niemcy przeprowadzają rabunek polskich zakładów naukowych, bibliotek i pracowni, świadczyć, iż na czele

specjalnych komisji przebyłych do miast uniwersyteckich, celem wywiezienia do Niemiec bądź całych działów bibliotecznych, bądź też całkowitych urządzeń zakładów naukowych. Instytut Fizyczny przy Uniwersytecie Warszawskim stali profesorowie niemieccy, posilkujący się dawnymi rachunkami firm niemieckich, które dostarczały do Polski aparaty i dzieła naukowe. Działając więc przy pomocy terroru, obozów koncentracyjnych, grabieży i rzekomych "konfiskat", Niemcy dążą świadomie do obrócenia Polski w "pustynię intelektualną".

Przechodząc do historii powstania Polskiego Wydziału Lekarskiego w Edynburgu, prof. dr. A. Jurasz szczegółowo omawia okoliczności, w jakich zrodziła się ta myśl.

W czerwcu 1940 r., po katastrofie Francji, część wojsk polskich znalazła się w Wielkiej Brytanii, a ściślej mówiąc, w Szkocji. W jednym z obozów znajdowała się znaczna ilość polskich lekarzy wojskowych. Pułkownik Irvin Fortescu, który był wówczas oficerem łącznikowym pomiędzy władzami brytejskimi a dowództwem wojsk polskich w Szkocji, stwierdziwszy, iż w okresie reorganizacji wojsk armii polskiej nie wszyscy lekarze polscy znajdą zatrudnienie, wystąpił z propozycją by lekarze specjaliści udali się na pewien czas do wojskowego szpitala w Edynburgu na staż oraz dla nawiązania pierwszych kontaktów z miejscowym światem lekarskim. Komendantem szpitala wojskowego (Castle Military Hospital) w Edynburgu był ppłk. prof. Crew, który nie tylko zaakceptował ten projekt, lecz następnie, dowiedziawszy się iż pomiędzy przebywającymi w Szkocji polskimi lekarzami wojskowymi znajduje się, z jednej strony, znaczna ilość profesorów i wykładowców polskich wydziałów medycznych—z drugiej strony—znaczna ilość absolwentów i studentów polskich wydziałów lekarskich, wystąpił z projektem, by w porozumieniu z fakultetem medycznym Uniwersytetu w Edynburgu, utworzyć Polski Wydział Lekarski przy tymże Uniwersytecie, dając tą drogą możliwość profesorom i wykładowcom polskim kontynuacji ich pracy naukowej, zaś absolwentom i studentom ukończenia ich studiów lekarskich.

Projekt ten znalazł aprobatę polskich władz wojskowych, a Szef Służby Zdrowia i Korpusu, stacjonującego w Szkocji, płk. dr. Kurtz, popierany przez generała Kukiela, dowódcę Wojsk polskich w Szkocji, wystosował raport do Naczelnego Wodza w Londynie z przedstawieniem całej sprawy. Szef Służby Zdrowia Sztabu Naczelnego Wodza, płk. dr. Gergovich przedstawił sprawę Generalowi Sikorskiemu i uzyskał jego aprobatę. Minister Spraw Wewnętrznych, prof. Stanisław Kot, uznając wielką doniosłość dla nauki polskiej utworzenia Wydziału Lekarskiego w Edynburgu, udzielił entuzjastycznego poparcia całej sprawie i, na podstawie uchwały Rady Ministrów 22 października 1940 r. upoważnił prof. dr. A. Jurasza do prowadzenia pertraktacji w imieniu Rządu w sprawie utworzenia Polskiego Wydziału Lekarskiego przy Uniwersytecie w Edynburgu.

Przedstawiona przez ppłk. prof. Crew Wydziałowi Lekarskiemu w Edynburgu sprawa utworzenia Polskiego Wydziału Lekarskiego przy tym Uniwersytecie, została przez Dziekana tego Wydziału prof. Sydney Smith i cały Wydział jednomyślnie zaakceptowana. Rektor Uniwersytetu Sir Thomas Holland, również i Senat Uniwersytecki wyrazili swą aprobatę.

Rozpoczęły się pertraktacje, w których ze strony polskiej uczestniczył Komitet Organizacyjny w osobach: prof. Jurasz—przewodniczący, członkowie: prof. J. Fegler (Kraków), prof. W. Koskowski (Lwów), prof. Lakner (Poznań), prof. Rogalski (Kraków), prof. Rostowski (Lwów), oraz Szef Służby Zdrowia Armii Polskiej płk. dr. Gergovich.

W dniu 24 lutego 1940 r. umowa, dotycząca utworzenia Polskiego Wydziału Lekarskiego przy Uniwersytecie w Edynburgu, została podpisana pomiędzy Rządem Polskim a Uniwersytetem Edynburskim.

W dniu 22 marca 1941 r. w obecności Prezydenta Rzplitej i przedstawicieli Rządu Brytyjskiego, Miasta Edynburga, władz uniwersyteckich, odbyła się uroczysta inauguracja Polskiego Wydziału Lekarskiego w Edynburgu.

Spis profesorów i wykładowców Polskiego Wydziału Lekarskiego

| | | |
|-------------------------------|-------------------------|-----------------|
| prof. dr. A. Jurasz (Dziekan, | chirurgia | Uniwers. Poznań |
| prof. dr. J. Fegler | fizjologia | .. Kraków |
| prof. dr. W. Koskowski | farmakologia | .. Lwów |
| prof. dr. L. Lakner | stomatologia | .. Poznań |
| prof. dr. T. Rogalski | anatomia | .. Kraków |
| prof. dr. J. Rostowski | neurologia i psychiatra | .. Lwów |
| prof. dr. B. Nowakowski | higiena | .. Wilno |

| | | | |
|--------------------------|-----------------------|----------|----------|
| doc. dr. A. Elektorowicz | radiologia | Uniwers. | Warszawa |
| doc. dr. A. Fidler | choroby wewnętrzne | " | Warszawa |
| doc. dr. M. Kostowiecki | histologia | " | Lwów |
| doc. dr. E. Mystkowski | chemia | " | Warszawa |
| doc. dr. H. Reiss | dermatologia | " | Kraków |
| doc. dr. B. Sliżyński | biologia | " | Kraków |
| doc. dr. W. Tomaszewski | choroby wewnętrzne | " | Poznań |
| dr. B. Czemplik | fizyka | " | Poznań |
| dr. J. Dekański | toksykologia | " | Warszawa |
| dr. J. Iwaszkiewicz | otolaryngologia | " | Poznań |
| dr. J. Kochanowski | radiologia | " | Warszawa |
| dr. Z. Malkiewicz | pediatria | " | Kraków |
| dr. R. Rejthar | metody znieczulania | " | Poznań |
| dr. J. Ruskowski | okulistyka | " | Warszawa |
| dr. T. Sokolowski | chirurgia urazowa | " | Warszawa |
| dr. W. Stocki | anatomia patologiczna | " | Poznań |
| dr. C. Uhma | ginekologia | " | Kraków |

Prof. dr. Jurasz przedstawia warunki pracy w ciągu minionego roku akademickiego zaznaczając, iż odbywała się ona w atmosferze najserdeczniejszej współpracy z władzami uniwersyteckimi, władzami Royal Infirmary, oraz z władzami municypalnemi.

W ubiegłym roku akademickim Wydział miał 77 słuchaczy, w tym 47 wojskowych, 30 cywilnych. Absolwentów 30. Ilość odbytych godzin wykładów i ćwiczeń dla I roku wyniosła ogółem 640, dla IV, V i Absolwentów wykłady oraz ćwiczenia praktyczne w klinikach zajęły 1114 godzin. Ilość egzaminów złożonych z wynikiem dodatnim dla IV. V roku i dla absolwentów wyraża się w liczbie 90. Pierwszy dyplom lekarski otrzymał por. pilot, który powrócił do swego oddziału jako pilot. Nowy rok akademicki rozpoczęto mając zgórą 100 słuchaczy.

Kończąc swój referat prof. dr. Jurasz stwierdza, iż powstanie Polskiego Wydziału Lekarskiego w Edynburgu jest realnym potwierdzeniem tego, że nauka jest własnością wszystkich narodów, wszystkich społeczeństw w ich dążeniu do tego nowego porządku świata, który będzie oparty nie na interesach poszczególnych jednostek i narodów, lecz całej ludzkości. Jednocześnie, służąc, jako dowód niezniszczalnej wytrwałości i żywotności polskiej nauki, Polski Wydział Lekarski w Edynburgu jest czynnym odcinkiem walki o Wolność Polski i polskiej kultury.

To-day my task is to speak on the Foundation of the Polish Medical Faculty within the University of Edinburgh.

Attacked in September 1939 by an enemy who, by his constant reassurances of peaceful aims, had lulled the watchfulness of the world, the Poles were the first in Europe to take up arms and go forth into an unequal struggle. The formidable war machine of Hitler has broken the possibility of organized armed resistance in Poland. Since our military collapse Hitler has declared his second and no less atrocious war on Polish culture, science and spirit. He announced through General Governor Franck that: "The Polish slave people have no need for education, the elementary school is more than enough. There will never again be a higher institution of learning in Poland, instead there shall be an intellectual desert."

This announcement was the formal pretext for closing all universities and higher schools of every kind; learned societies have been banned; Polish periodicals and books have been forbidden.

To ensure that this time Polish science should not rise again, the greater part of the universities' teaching staffs have been arrested. The example of Cracow has shown us how one hundred and eighty distinguished men of science and professors were deported to a concentration camp in November 1939. Many died as a result of privation, and their deaths have been announced to their families simply by sending them their ashes. The professors of Poznan University have met a similar fate, and during the last few weeks we have heard of the same crime being perpetrated in the case of the professors of Lwow and Warsaw.

The Germans have shown great ingenuity in finding justification for killing prominent members of the Polish scientific world—one of which is, that the community is responsible for the action of the individual member. By adopting this principle they have not hesitated to shoot eminent scientists, although they were completely innocent. An outstanding example is the death of the eminent zoologist, Kopeć, known for his studies of insect life.

Very exact reports of what is happening have been received by our former Minister of the Interior—Professor Kot—now our Ambassador in Soviet Russia. In one of his lectures he has revealed how all libraries have been closed, the contents of many have been confiscated, while others have been reopened as German institutions in order to serve the cause of the Germanization of Poland. Everything of special value has been taken from the museums, galleries and collections and sent to Germany. Even cathedral and church treasures have not escaped. It is shameful to have to say that in this criminal plunder of our cultural institutions German professors of distinction, who were formerly our guests at Scientific Congresses, have taken a very active part. It is significant that in some cases these men had with them copies of the receipted bills given to them by the German firms from whom the equipment had been purchased originally. This explains the exact knowledge these Germans had of what there was to be taken away. Names like those of Dr. Augsburg, Professor Mühlmann, Professor Pullhammer, Dr. Richter will never be forgotten in this connexion! This systematic and well-prepared looting, after hostilities had ceased, has brought no smaller losses on the Polish nation than all the aerial and artillery bombardments of Warsaw.

Another sign of the German hatred of Polish culture is the requisitioning by the invaders of all buildings connected with education. The Ministry of Public Instruction is occupied by the Gestapo; Warsaw University now houses the Security Police; the secondary schools are occupied by the Army and it is not without interest to learn that the Jewish Students' Hostel in Cracow has been transformed into a brothel.

People who have been reared under Western rule cannot comprehend the endless vulgar atrocities which are being committed with such mastery and delight in the endeavour to exterminate Polish culture.

In June 1940 the Polish Army was brought over from France to the British Isles. Amongst them were Polish doctors who intended to fight for the common cause, and here I am touching more closely the point connected with the foundation of the Polish Faculty of Medicine in Edinburgh.

When the Poles were stationed in Scotland, Colonel Irvine Fortescue was D.D.M.S. Scottish Command. Finding considerable numbers of Polish doctors, and among them members of Polish Medical Faculties, in the army, he looked around for something that might occupy and refresh their minds. He consulted Lieut.-Colonel Professor F. A. E. Crew, who at that time was commanding the Military Castle Hospital in Edinburgh, and it was arranged that Colonel Fortescue should suggest to the Polish military authorities that batches of twenty Polish medical officers should be attached to the Military Hospital, Edinburgh, for periods of a fortnight, and that Lieut.-Colonel Crew would make further arrangements with the Medical Faculty of the University and with the Managing Board of the Royal Infirmary, whereby they might be spread among the different university medical departments and infirmary clinics.

This suggestion was eagerly accepted by the Polish military authorities, and batch after batch of officers came to renew their interest in professional activities. But it was soon recognized that the linguistic difficulties were robbing these good intentions of much of their potential value.

Lieut.-Colonel Crew, being assured that, among the Polish medical officers then in Scotland, there were many who had held academic positions in Polish Universities and also that among the troops there were many students of medicine whose studies had been interrupted by the war, made the further suggestion that these Polish professors should be allowed to come to Edinburgh and teach their own people in their own language, and that the medical undergraduates should be allowed to come and finish their curriculum and proceed to graduation.

These suggestions finding favour with Lieut.-Colonel Dr. Kurtz, the Director of Medical Services of the Polish Forces in Scotland, Professor Crew thereupon presented them to the Faculty of Medicine of the University of Edinburgh. Lieut.-Colonel Dr. Kurtz, with the support and approval of General Kukiel, G.O.C. Polish Forces in Scotland, sent in a report on the proposition to the Polish General Staff in London, asking the military authorities to permit the medical officers of academic standing to settle in Edinburgh, in order that they might participate in the activities of the different University departments to which they were to be attached, and also to allow those medical students who had finished all their classes in Poland but had not graduated to come to Edinburgh for a period of from three to six months, to be given refresher courses, and thereafter graduate and return to the army as reinforcements for the Medical Service. The Chief Medical Officer of the Polish Army, Lieut.-Colonel Gergovitch, approved this proposition and obtained the consent of General Sikorski, Commander in Chief of the Polish Army in Great Britain. The Polish Minister for the Interior, Professor Kot, enthusiastically took up the idea, recognizing its great value not only from the point of view of the individual and general advantages for the army, and for the future medical service in the depopulated and devastated Poland, but also as forming a closer cultural link between the Polish and British nations.

In October 1940 Professor Kot instructed me to proceed to Edinburgh and get into contact with the University authorities there.

The reception I encountered in Edinburgh surpassed all expectations. Lt.-Colonel Crew proved a wise and friendly adviser of immense value. The Dean of the Medical Faculty, Professor Sidney Smith, placed before his Faculty the suggestion that we should form a medical school of our own: they gave it their unanimous approval and forwarded to the Senatus Academicus for consideration the resolution that a Polish Medical Faculty should straightway be created. The principal, Sir Thomas Holland, gave his powerful support. On October 7 I was able to report to the Polish Minister for the Interior that, in my considered opinion, a Polish Medical Faculty should be formed. The Polish Government in consequence appointed me their representative at the University of Edinburgh with full powers to carry out the whole plan.

The University issued an official invitation to the Polish Prime Minister, General Sikorski, which was readily accepted. The secretary of the University, and the legal adviser to the Polish Government worked out the constitution of the new school. The heads of all departments of the Edinburgh Medical Faculty placed the resources of these departments at the services of their Polish confrères. The Board of Management of the Royal Infirmary agreed to open their lecture theatres and wards to Polish professors, lecturers and students and recently accommodation in some municipal hospitals has, by the courtesy and generosity of the Municipal Authorities, been placed at the disposal of the Polish Medical Faculty.

To design the internal structure of the Polish Medical Faculty, Professor Kot formed, under his direction, an organizing committee which consisted of: Professor Dr. Jerzy Fegler (Physiology), Cracow University; Professor Antoni Jurasz (Surgery), Poznan University; Professor Dr. Włodzimierz Koskowski (Pharmacology), Lwow University; Professor Dr. Leon Lakner (Stomatology), Poznan University; Professor Dr. Tadeusz Rogalski (Anatomy), Cracow University; Professor Dr. Jacob Rostowski (Neurology), Lwow University; and the Chief Medical Officer of the Polish Army, Colonel Gergovitch. This organizing committee, of which I was the convener, next called into being the teaching staff of the new school. The members of it were the six professors already mentioned, seven docents at Polish Universities, and ten eminent specialists.

The names of the seven docents are: Docent Dr. Adam Elektorowicz (Radiology), Warsaw University; Docent Dr. Antoni Fidler (Medicine), Warsaw University; Docent Dr. Marjan Kostowiecki (Histology), Lwow University; Docent Dr. Edmund Mystkowski (Chemistry), Warsaw University; Docent Dr. Henryk Reiss (Dermatology and Venereal Diseases), Cracow University; Docent Dr. Bronisław Słizynski (Biology), Cracow University; Docent Dr. Wiktor Tomaszewski (Medicine), Poznan University.

The specialists are: Dr. Bernard Czemplik (Physicist), Poznan; Dr. Jerzy Dekanski (Toxicology), Warsaw; Dr. Jarosław Iwaszkiewicz (Ear, Nose and Throat), Poznan; Dr. Jan Kochanowski (Radiology), Warsaw; Dr. Zdzisław Malkiewicz (Pædiatrics), Cracow; Dr. Roman Rejthar (Surgery), Poznan; Dr. Jan Ruszkowski (Ophthalmology), Warsaw; Dr.

Tadeus Sokolowski (Traumatology), Warsaw; Dr. Wacław Stocki (Pathological Histology and Pathology), Poznań; Dr. Czesław Uhma (Gynaecology and Obstetrics), Cracow.

The majority of these men had been called to the army service for the duration of the war.

Before the beginning of the academic year all members of the teaching staff had settled in their new environment. They were received in a very friendly and hospitable manner in the different departments and clinics of the University. The Central Medical Library has been put at their disposal and all necessary arrangements for starting instruction and research work have been completed.

It was on February 24, 1941, that the Agreement was signed between the Polish Government and the University of Edinburgh on the basis of which the Polish School of Medicine was created.

The chief points in the constitution of the Polish Medical School may be summed up as follows:

(1) The Head of the Polish School of Medicine is the Dean who has the power of a Rector of Polish University.

(2) The Faculty consists of Polish professors who have held Chairs in Poland, and of professors of the Faculty of Medicine of Edinburgh University representing only such subjects for which Polish docents or Polish lecturers are not available.

(3) A member of the Faculty is also, according to Polish law, one delegate of the Polish Docents.

(4) The Dean shall be one of the Polish professors elected by the Faculty and approved by the competent Polish Authority after consultation with the University of Edinburgh.

(5) The curriculum and the standards of teaching and the examination in the school shall be the same as that required in the medical faculties in Polish universities, and as far as possible the Medical Faculty of the University of Edinburgh.

(6) The programme of teaching is somewhat different from the British programme; according to Polish law, it includes, as subjects of examination, Ophthalmology, Otolaryngology, Neurology, Stomatology and Radiology.

(7) The examinations are practical, theoretical and oral.

(8) The Chairman of the Examination Board is the Dean.

According to this constitution the Faculty was established as follows:

THE DEAN

Professor A. T. Jurasz

MEMBERS

Polish

Professor Dr. Fegler
 Professor Dr. Koskowski
 Professor Dr. Lakner
 Professor Dr. Rogalski
 Professor Dr. Rostowski
 Dr. Elektorowicz (delegate of the Docents)
 Professor Nowakowski, Professor of Hygiene and Public Health at Wilno University, joined the Faculty at the end of the first academic year.

British

Professor W. G. Clark, M.O.H.
 Professor F. A. E. Crew, M.D., D.Sc., D.P.H.
 Professor L. S. P. Davidson, B.A., M.D., F.R.C.P.E.
 Professor A. M. Drennan, M.D., F.R.C.P.E.
 Professor R. W. Johnstone, C.B.E., M.A., M.D.
 Professor Charles McNeil, M.A., M.D., F.R.C.P.
 Professor T. J. Mackie, M.D., D.P.H., F.R.S.
 Professor G. F. Marrian, D.Sc.
 Professor Sidney Smith, M.D., D.P.H., F.R.C.P.E.

The solemn inauguration of the Polish Medical Faculty took place on March 22, 1941, in the presence of the President of the Republic of Poland, Władysław Raczkiewicz, and the Representative of the British Government, the Lord President of the Council, the Right Honorable Sir John Anderson, the Sheriff of the Lothians, the Lord Provost of Edinburgh, the Earl of Rosebery and Professor Kot, the Polish Minister for the Interior.

This unique and dignified ceremony has left an indelible impression on the minds of all who participated in it. It was one of the brightest moments of our lives since we left

Poland. We felt that a great responsibility had been entrusted to us by our leaders, that we had the confidence of the University Authorities, and not least, that we had the faith of the Polish youth. A more detailed report of the opening of the Polish Medical Faculty in Edinburgh, together with the full text of all the speeches on this historic occasion is given in the *University of Edinburgh Journal*, July 1941, II, No. 2.

Because there was not enough space available in the corresponding departments of Edinburgh we have organized our own departments of Anatomy, Physiology and Histology in the Old Anatomy Department in Bristol Street which had been placed at our disposal. These departments serve only for teaching purposes.

As a complement of the Faculty we have organized a Polish hospital of our own, called the Paderewski Hospital, in a separate building in the grounds of the Western General Hospital. We were able to equip this hospital through magnanimous and generous financial help from America through the "Refugees of England" Anglo-American Committee, and the Paderewski Testimonial Fund incorporated. The Paderewski Hospital serves for the treatment of civilian Poles as well as for military cases and it is staffed by the specialists, professors and docents of the teaching staff of the Medical Faculty. It contains 80 beds with different wards and out-patients' departments for stomatology, ophthalmology, oto-laryngology, gynecology and obstetrics, paediatrics and medicine. A supplement of 60 surgical beds has been put at our disposal in the wards of the Western General Hospital, with the use of the operating theatres and the X-ray department. The direction of this hospital remains in the hands of the Dean of the Polish Medical Faculty.

A new medical library of our own has been started and already contains 340 volumes. It has been created partly by our own purchases and partly by gifts, as for instance that of 63 excellent manuals by the Associated Graduates of Edinburgh, 62 volumes given by the British Council and 24 through Major Douglas Guthrie, &c.

Just a few days ago the first academical year ended and the second started with hardly a break. Last year the number of students was 77; amongst them were 47 serving in the army who received leave for the purpose of their studies, and 30 civilians. Some 30 had finished all their studies in Poland but had not passed their examinations and it was therefore arranged that they should go through a refresher course. The remainder, including newcomers, were distributed to the first, fourth and fifth year classes.

In bacteriology and legal medicine in which we have no Polish lecturers, Professor Sidney Smith and Professor Mackie of the Edinburgh Medical Faculty have kindly undertaken the instruction and examination of our students.

The first student to receive his diploma from the hands of the Dean of the Polish Medical Faculty in Edinburgh was a pilot in the Air Force who, despite his medical profession, returned to his fighter squadron after receiving his degree.

For the second academic year which has just started we have 120 students on our Roll, of whom five are Czechs.

The teaching and faculty body has been augmented by the arrival of Professor Bruno Nowakowski, Professor of Hygiene and Public Health, of Wilno University.

Our task was not an easy one, particularly as our aim from the very beginning was to keep the same high standard on foreign soil as we have had in the Faculties in Poland. We were determined about this and we are carrying it through. But I would like to say here that our task would have been absolutely impossible if we had not had the full understanding and the great friendship which has been shown to us by the University authorities and by our Scottish colleagues at every turn. We have received every possible support and every possible facility.

The University of Edinburgh has not only given us hospitality, it has given us its heart.

Now, why is it that in Edinburgh we have created the only existing Polish academical school at the present moment in the whole world? Was this mere chance? Perhaps not quite so because the old University of Edinburgh was not unknown to Poles. In his Inaugural Address in March 1941, Professor Kot told how among the Scottish emigrants who, centuries ago, came to live in Poland there was one who founded a scholarship for Protestant ministers in Poland to study at the University of Edinburgh. Owing to this foundation, Polish students made their appearance here in the seventeenth and eighteenth centuries. As the seat of Common Sense Philosophy the University of Edinburgh attracted the attention of John Sniadecki, mathematician and astronomer, the rector of Cracow University, at the end of the eighteenth century." Dugald Stewart was Sniadecki's master in the school of thought which he propagated in Poland. Sniadecki sent his younger

brother Andrew here, who studied under Andrew Duncan, Alexander Monroe and James Gregory, and on his return to Poland organized a Department of Modern Medical Science at the University of Wilno, basing it on his experience gained in Edinburgh. This Department served as a model for the reform of the medical faculty at the University of Cracow. It was also from Edinburgh that Andrew Sniadecki brought home the inspiration to prepare the first Polish fundamental manual of organic chemistry.

The many-sided genius of Andrew Duncan also encouraged Sniadecki to many a stroke of initiative in the arrangement of university clinics, in the propagation of the idea of physical education and professional medical publications.

The University of Edinburgh became the centre of attraction for the Poles after the year 1820, when young sons of the Polish aristocratic families arrived to study there. One of them, Konstanty Zamoyski, offered the University a set of books giving information about Poland, her history, art and culture, which has been preserved until to-day in the Advocates' Library. Owing to the encouragement of Professor John Wilson, the protector of those Poles and the most popular figure at the University of that day, one Polish student, Krystyn Lach-Szyrma, published in Edinburgh "Literary and Political Letters on Poland" (1823). The same author on his return to Poland, where he became a professor in the University of Warsaw, published a work in three volumes entitled "England and Scotland—Reminiscences from my Journal", in which he gave a striking picture of Scotland of those days, of her life, her economic and cultural peculiarities, her national character and customs, of her great men—in the first place Sir Walter Scott, and a number of the professors of Edinburgh University.

Such are some of the old cultural links between Scotland and Poland.

Several weeks ago there was a meeting in London of Scientists from allied countries. A *Magna Charta Scientiae* has been published which, in my opinion, is an act of some political importance; it has set out the reasons for which we are fighting in this, the most terrible war the world has ever known. The resolution passed at this meeting states that liberty of thought is the principal condition of human evolution. Men of science have been fighting through the ages against the oppression of free thought and tyranny, and they have suffered in the name of Liberty of Science. Further we read in this declaration that the Liberty of Learning and of Teaching are indispensable for the evolution of science and that digression from this principle would be degrading to human ideals.

Therefore the story of the creation of the Polish Medical Faculty, so dear to us Poles and particularly to us professors and representatives of medicine, surely deserves to find a place not only in the history of Medicine, but in the history of world culture, as one of the fulfilled postulates of this *Magna Charta Scientiae*. The Polish Medical Faculty in Edinburgh is an effective post in the front line of our battle against the destructive power of barbarism which seeks to set up its perverted "culture" on the ruins of Europe.

It is an affirmation that science can be international, that its tendency is towards a real and a better order of the world which must be based, not only on the interest of certain individuals and only a few nations, but on the whole of humanity.

I consider the foundation of the Polish Faculty of Medicine in Edinburgh to be an important and symbolic contribution to that culture for which we are all striving to assure long duration, safety and free development!

Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

[November 28, 1941]

Some Practical Considerations in the Control of Louse-borne Typhus Fever in Great Britain in the Light of Experience in Russia, Poland, Rumania and China

By MELVILLE D. MACKENZIE, M.D.

ABSTRACT.—This paper deals with some aspects of the control of louse-borne typhus fever. The epidemic form is associated with famine and overcrowding. In producing an epidemic in a hitherto endemic area malnutrition is of greater importance than overcrowding; another factor which brings this about is widespread movement of civil or military population thus bringing non-immunes into a district where typhus is endemic.

Epidemic typhus usually occurs in the early months of the year, whilst the epidemic form may appear at any time. Conditions under which epidemic typhus occurs favour the outbreak of other diseases so that an uncomplicated case is rarely seen.

Louse-borne typhus fever lasts from twelve to sixteen days. The incubation period is usually twelve to fourteen days, though it may be from five to twenty-one days. The onset is sudden, but is often preceded by malaise and a rise of temperature. Two common initial symptoms are acute frontal or occipital headache and bronchitis. The first sign may be mental confusion or delirium. Acute delirium is usually present after the first week.

The diagnosis at the end of the first week may be determined by the Weil-Felix reaction. The author discusses the practical value of this and the question of immunity to second attacks of the disease. The most important diagnostic signs are the absence of the rash from the face and the fact that "cropping" does not occur in the appearance of the exanthem. A description follows of the details of the administrative control of typhus with which the writer was associated in Mesopotamia, Poland, Russia, Rumania and China and the methods of disinfestation employed in these countries.

Attention is drawn to the great danger to which personnel are exposed; the special clothing that must be worn and other methods of protection are described in detail. Vaccines should not be relied upon in the present state of our knowledge. In the control of the disease even greater importance is laid on reducing lice in the population generally than in attempting to trace the remoter contacts of cases. The methods by which lice are transferred are then discussed and the predominant importance of mechanical transference is stressed. The importance of dealing with malnutrition in typhus outbreaks is referred to and the administrative and practical details for dealing with a case of typhus are given. In his conclusions, the writer emphasizes the following points: The rapidity of the spread of the disease; the difficulty of the diagnosis; the necessity for complete thoroughness in disinfestation and for detailed supervision of the protection of personnel, who should be under 30 years of age, as the mortality rate increases greatly after this age.

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vacunas en el estado actual de nos conocimientos. En el control del mal aun más rta el reducir piojos en la población general que los esfuerzos para trazar los ictos algo apartados de casos. Luego se pasa en revista los métodos por que trans- nese los piojos, y se hace notar la importancia suma de transferencias mecánicas. Se ciona la importancia de entenderse con la malnutrición en los brotes del tifo, v se ne los pormenores administrativos y prácticos del tratamiento de un caso de tifo cluyendo, el autor insiste sobre los puntos siguientes: diseminación rápida de la rmedad; dificultad del diagnóstico; necesidad de una desinfección acabadísima v una supervisión circunstancial de la protección del personal, quien debiera tenir menos reinte años, edad por encima de la que aumenta mucho la mortalidad.

ESUMO.—Esta dissertação trata de alguns aspectos do control da febre tífica transmitida piolhos. A forma epidémica aparece quando ha fome e grandes aglomerações de te. A alimentação deficiente é de maior importancia no aparecimento da epidemia n meio endêmico do que as grandes aglomerações de gente: um outro factor que a orece é o movimento em grande escala da população civil ou de grandes contingentes tropas, trazendo assim individuos não imunes para localidades onde o tifo é endêmico. O tifo endêmico aparece geralmente nos primeiros meses do ano, ao passo que a forma demica pode aparecer em qualquer época. As condições em que aparece o tifo demico favorecem o aparecimento de outras doenças, dando isto o resultado de rara- nte se encontrar um caso sem complicações. O tifo transmitido por piolhos dura de 12 a 16 dias. O periodo de incubação é geralmente 12 a 14 dias, e nalguns casos de 5 a 21 dias. O ataque é subito, mas é precedido muitas zes de mal estar e de aumento de temperatura. Dois symptomas iniciais muito vulgares o fortes dores de cabeça; na região frontal e occipital e bronquite. O primeiro sinal pode ser perturbação mental ou delirio. O delirio agudo manifesta- geralmente depois da primeira semana. O diagnostico no fim da primeira semana pode ser decidido pela reacção Weil-Felix. O autor discute o seu valor pratico e a questão de imunidade de ataques consecutivos da oença. Os sinais mais importantes para o diagnostico são a ausencia de erupção na face o facto da mesma não se manifestar em dias successivos, quando aparece o exantema. egue a descrição dos pormenores do control administrativo do tifo com que o autor steve associado na Mesopotamia, Polonia, Russia, Roménia e China, e os meios le desinfeção adotados nestes paizes. Chama-se a atenção para o grande perigo a que está exposto o pessoal hospitalario: o vestuario especial que deve usar e os outros meios de protecção são descritos minuciosa- mente. Não se deve ter muita confiança nas vacinas, no estado presente dos nossos conhecimentos. No control da doença muito maior importancia tem a despiolhacão da população em geral, do que tentar descobrir os contactos mais remotos dos casos existentes. Os meios pelos quaes se faz a transferencia dos piolhos são enumerados em seguida e a importancia predominante da transferencia mecanica deve ser bem marcada. A impor- tancia da má alimentação nas epidemias de tifo e os pormenores das medidas administra- tivas e praticas a tomar em casos de tifo tambem foram mencionados. O autor nas suas conclusões acentuou os pontos seguintes: A rapidez do alastramento da doença; a dificuldade do diagnostico; a necessidade da completa desinfeção e de minuciosa fiscalisação de protecção do pessoal, que deve ser de menos de 30 anos de idade, por ser notorio que o coeficiente da mortalidade aumenta muito em individuos de mais idade.

INTRODUCTION

EVERY year brings more evidence of how widespread rickettsial diseases are, particu- larly amongst animals, and there are now few areas of the world where rickettsial con- ditions have not been found. In addition to the new knowledge of the widespread dis- tribution of the Rickettsia themselves, a steadily increasing number of species of arthro- pods (ticks, fleas, mites) is being incriminated. Louse-borne rickettsial infection is, however, unique, in the fact that, as far as is known, it is the only type that is primarily a disease of man and consequently the only one which occurs in epidemic form. All other rickettsial conditions are, as far as man is concerned, endemic and localized, being really accidental infections. At first sight it may perhaps seem strange that flea-borne typhus does not spread in epidemic form like plague. This is probably due to two factors. In the first place the rat only carries the virus of the disease for a very short

RÉSUMÉ.—Cet article concerne certains aspects du contrôle du typhus transmis par les poux. La forme épidémique est associée à la famine et au surpeuplement. La famine est plus importante que le surpeuplement dans la production d'une épidémie dans une région de typhus endémique. Les grands mouvements de la population civile ou militaire peuvent produire le même effet, en amenant des individus non immuns dans une région de typhus endémique.

Le typhus endémique apparaît le plus souvent vers le commencement de l'année, tandis qu'une épidémie peut se déclarer à n'importe quel moment. Les conditions qui amènent le typhus épidémique favorisent aussi d'autres maladies, de sorte qu'on voit rarement un cas de typhus sans complications.

Le typhus transmis par les poux dure de 12 à 16 jours. La période d'incubation dure le plus souvent de 12 à 14 jours, mais peut durer de 5 à 21 jours. Le début est brusque, mais souvent précédé de malaise et d'une élévation de la température. Deux symptômes initiaux fréquents sont une céphalée frontale ou occipitale et une bronchite. Le premier signe peut être une confusion mentale ou un délire. Le délire aigu est généralement présent après la première semaine.

La réaction de Weil-Felix permet de poser le diagnostic à la fin de la première semaine. L'auteur discute sa valeur pratique, ainsi que la question de l'immunité contre une seconde attaque de la maladie. Les signes diagnostiques les plus importants sont l'absence de l'éruption sur la figure et l'absence de poussées successives de l'exanthème.

Il suit une description des mesures administratives contre le typhus dans lesquelles l'auteur a pris part en Mésopotamie, en Pologne, en Russie, en Roumanie et en Chine, et des méthodes de désinfection adoptées dans ces pays. Il attire l'attention sur le grand danger couru par le personnel, et décrit le costume spécial nécessaire et d'autres mesures protectrices. Dans l'état actuel de nos connaissances il ne faut pas se fier aux vaccins. Pour le contrôle de la maladie l'auteur attache encore plus d'importance à la réduction des poux qu'à la découverte des contacts éloignés du malade. Ensuite l'auteur discute les modes de transmission des poux, et insiste sur la prédominance du transport mécanique, puis il parle de l'importance de la lutte contre la malnutrition pendant une épidémie de typhus, et décrit les détails pratiques et administratifs du traitement des cas individuels.

En conclusion, l'auteur appuie sur les faits suivants: 1° la rapidité de la propagation de la maladie, 2° la difficulté du diagnostic; 3° la nécessité de la désinfection absolument complète et de la surveillance soignée de la protection du personnel, qui doit être âgé de moins de 30 ans, car la mortalité est beaucoup plus élevée au-dessus de cet âge.

RESUMEN.—Este artículo considera unos aspectos del control del tifo transmitido por piojos. La forma epidémica se asocia con hambre y apiñadura. Para la producción de una epidemia en una región hasta aquí endémica, tiene más importancia la malnutrición que la apiñadura; otro factor que efectúa ésta es el movimiento extensivo de la población civil o militar, llevando personas no inmunes a una región donde el tifo está endémica.

El tifo endémico ocurre generalmente temprano en el año, mientras que la forma epidémica puede encontrarse a cualquier hora. Las condiciones en que ocurre el tifo epidémico favorecen el brote de otras enfermedades, de suerte que rara vez se ve un caso incomplicado.

El tifo transmitido por piojos dura de 12 a 16 días. El período de incubación es generalmente de 12 a 14 días, aunque puede ser de 5 a 21 días. El ataque es súbito, pero se precede muchas veces de malestar y alza de temperatura. Dos comunes síntomas iniciales son cefalalgia aguda frontal u occipital, y bronquitis. Puede ser la perturbación mental o el delirio el primer señal. El delirio agudo se encuentra por lo general tras la primera semana.

Acabada la primera semana, el diagnóstico puede hacerse por medio de la reacción de Weil-Felix. Háblase del valor práctico de ésta y del problema de inmunidad a ataques subsiguientes del mal. Las señas diagnósticas más importantes son la ausencia de erupción en la cara y lo de que no ocurren retoños sucesivos en el aspecto de la exantema. Sigue una descripción de los detalles de control administrativo del tifo a los que estaba asociado el autor en Mesopotamia, Polonia, Rusia, Rumania y China, y de los métodos de desinfección empleados en estos país.

LLámase la atención al gran peligro que incurre el personal; descríbese detalladamente la ropa especial que hay que llevar, y otros métodos de protección. No se debiera confiar

en vacunas en el estado actual de nos conocimientos. En el control del mal aun más importa el reducir piojos en la población general que los esfuerzos para trazar los contactos algo apartados de casos. Luego se pasa en revista los métodos por que transmitense los piojos, y se hace notar la importancia suma de transferencias mecánicas. Se menciona la importancia de entenderse con la malnutrición en los brotes del tifo, y se expone los pormenores administrativos y prácticos del tratamiento de un caso de tifo. Concluyendo, el autor insiste sobre los puntos siguientes: diseminación rápida de la enfermedad; dificultad del diagnóstico; necesidad de una desinfección acabadísima y de una supervisión circunstancial de la protección del personal, quien debiera tener menos de treinta años, edad por encima de la que aumenta mucho la mortalidad.

RESUMO.—Esta dissertação trata de alguns aspectos do control da febre tífica transmitida por piolhos. A forma epidémica aparece quando ha fome e grandes aglomerações de gente. A alimentação deficiente é de maior importancia no aparecimento da epidemia num meio endémico do que as grandes aglomerações de gente; um outro factor que a favorece é o movimento em grande escala da população civil ou de grandes contingentes de tropas, trazendo assim individuos não imunes para localidades onde o tifo é endémico.

O tifo endémico aparece geralmente nos primeiros meses do anno, ao passo que a forma epidémica pode aparecer em qualquer epoca. As condições em que aparece o tifo epidémico favorecem o aparecimento de outras doenças, dando isto o resultado de raramente se encontrar um caso sem complicações.

O tifo transmitido por piolhos dura de 12 a 16 dias. O periodo de incubação é geralmente de 12 a 14 dias, e nalguns casos de 5 a 21 dias. O ataque é subito, mas é precedido muitas vezes de mal estar e de aumento de temperatura. Dois symptomas iniciaes muito vulgares são fortes dores de cabeça; na região frontal e occipital e bronquite.

O primeiro sinal pode ser perturbação mental ou delirio. O delirio agudo manifesta-se geralmente depois da primeira semana.

O diagnostico no fim da primeira semana pode ser decidido pela reacção Weil-Felix. O autor discute o seu valor pratico e a questão de imunidade de ataques consecutivos da doença. Os sinais mais importantes para o diagnostico são a ausencia de erupção na face e o facto da mesma não se manifestar em dias successivos, quando aparece o exantema. Segue a descrição dos pormenores do control administrativo do tifo com que o autor esteve associado na Mesopotamia, Polonia, Russia, Roménia e China, e os meios de desinfeção adotados nestes paizes.

Chama-se a atenção para o grande perigo a que está exposto o pessoal hospitalario: o vestuario especial que deve usar e os outros meios de protecção são descritos minuciosamente. Não se deve ter muita confiança nas vacinas, no estado presente dos nossos conhecimentos. No control da doença muito maior importancia tem a despiolhação da população em geral, do que tentar descobrir os contactos mais remotos dos casos existentes. Os meios pelos quaes se faz a transferencia dos piolhos são enumerados em seguida e a importancia predominante da transferencia mecanica deve ser bem marcada. A importancia da má alimentação nas epidemias de tifo e os pormenores das medidas administrativas e praticas a tomar em casos de tifo tambem foram mencionados. O autor nas suas conclusões acentuou os pontos seguintes: A rapidez do alastramento da doença; a dificuldade do diagnostico; a necessidade da completa desinfeção e de minuciosa fiscalisação de protecção do pessoal, que deve ser de menos de 30 anos de idade, por ser notorio que o coeficiente da mortalidade aumenta muito em individuos de mais idade.

INTRODUCTION

EVERY year brings more evidence of how widespread rickettsial diseases are, particularly amongst animals, and there are now few areas of the world where rickettsial conditions have not been found. In addition to the new knowledge of the widespread distribution of the Rickettsia themselves, a steadily increasing number of species of arthropods (ticks, fleas, mites) is being incriminated. Louse-borne rickettsial infection is, however, unique, in the fact that, as far as is known, it is the only type that is primarily a disease of man and consequently the only one which occurs in epidemic form. All other rickettsial conditions are, as far as man is concerned, endemic and localized, being really accidental infections. At first sight it may perhaps seem strange that flea-borne typhus does not spread in epidemic form like plague. This is probably due to two factors. In the first place the rat only carries the virus of the disease for a very short

time and therefore relatively few fleas are infected; secondly the disease is not fatal to the rat so that the fleas have no reason to drop off and seek other hosts.

The present paper deals only with louse-borne typhus fever, but before leaving the other forms of *Rickettsia* I should like to mention the transmutation of the mild flea-borne murine strain to the deadly louse-borne strain. In some parts of the world, particularly Mexico and Manchuria, both the flea-borne murine type and the louse-borne types exist side by side and appear largely to remain separate pathological entities. A comparable occurrence of tick-borne and louse-borne typhus is found in Rumania. On the other hand there is evidence that the ordinary flea-borne murine typhus, if transmitted through lice, will produce a disease both clinically and with immunological reactions indistinguishable from classical louse-borne typhus. From an epidemiological point of view this fact may be of very great importance and particularly so in Great Britain where both the flea-borne and tick-borne forms of the disease have occurred within recent years. Such an introduction amongst a louse-infested section of the population might cause, according to our latest evidence, an outbreak of classical typhus in this country. Moreover at present we know very little about the amount of endemic murine typhus amongst rats and other mammals in this country, though it is known that the rats of Paris, for example, are infested with murine typhus. As the object of this paper is essentially practical, it is unfortunately not possible to enter further into the extremely interesting facts now known regarding the transmutation of the *Rickettsia* viruses, their varying virulence according to the insect vector, and their relation to one another as shown by cross-immunization and agglutination.

PREDISPOSING CONDITIONS

Louse-borne typhus fever is an acute infectious disease lasting from twelve to sixteen days and characterized by a continued temperature, a generalized maculopapular rash which may become hemorrhagic, severe toxæmia, and marked nervous manifestations. The disease is carried by lice and spreads with extreme rapidity especially through a badly nourished population. Thus in Russia during the period 1919 to 1922 the estimated number of cases was 10,000,000, with 3,000,000 deaths, in a population of 120,000,000. These are stupendous figures. Their scale can be realized to some extent by recalling that in the much-described typhus epidemic in London in 1856 only 1,062 cases were recorded as treated in the London Fever Hospital out of a population of 3,000,000 whereas in Russia in the year 1921 alone there were 4,000,000 cases in a population of 120,000,000. These figures can, of course, only be approximate, as many cases diagnosed as typhus were in reality instances of relapsing fever; on the other hand a vast number of cases of typhus were never admitted to hospital and so remained unrecorded. Of the cases admitted to hospital very many were never notified by the Russian medical officers owing to pressure of work. So uncertain were the statements that when we went into a new district to survey the amount of typhus present we found it more useful to base our estimate on the number of women with recently shaved heads seen in the streets, than to rely upon notification figures. All cases on admission to hospital for typhus were closely shaved and consequently it was possible to sit in a café and determine the proportion of women with closely cropped heads to the general population and so to estimate roughly the amount of typhus in the region.

Epidemic typhus fever, is, classically, associated with famine and overcrowding, but there is a third factor which, to my mind, is perhaps of even greater importance, namely, widespread movements of military or civilian populations bringing non-immunes into a district where the disease is endemic or carrying the disease into a typhus-free region. A third possibility is that such movements may introduce into an endemic region either a new strain of the disease or one of enhanced virulence. The first mode of infection I saw well demonstrated in the epidemic in North China two years ago which was due to the introduction of masses of non-immunes with the Army into areas in North China where the disease was endemic. The second method occurred on the return of Polish prisoners of war to Poland from Siberia in 1919-1922. These men, women and children had been heavily infected with typhus in Russia, and passed into Poland at the rate of tens of thousands a day, going to regions in which the disease either was already endemic or did not exist previously; in both cases widespread epidemics resulted.

Apart from mass movements of the kinds instanced above, a striking feature of epidemics is the amount of local movements of the population that they initiate. Once

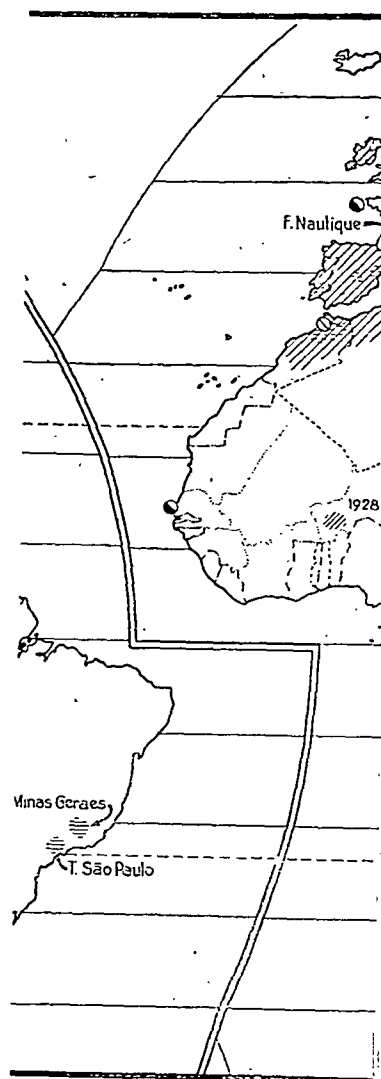
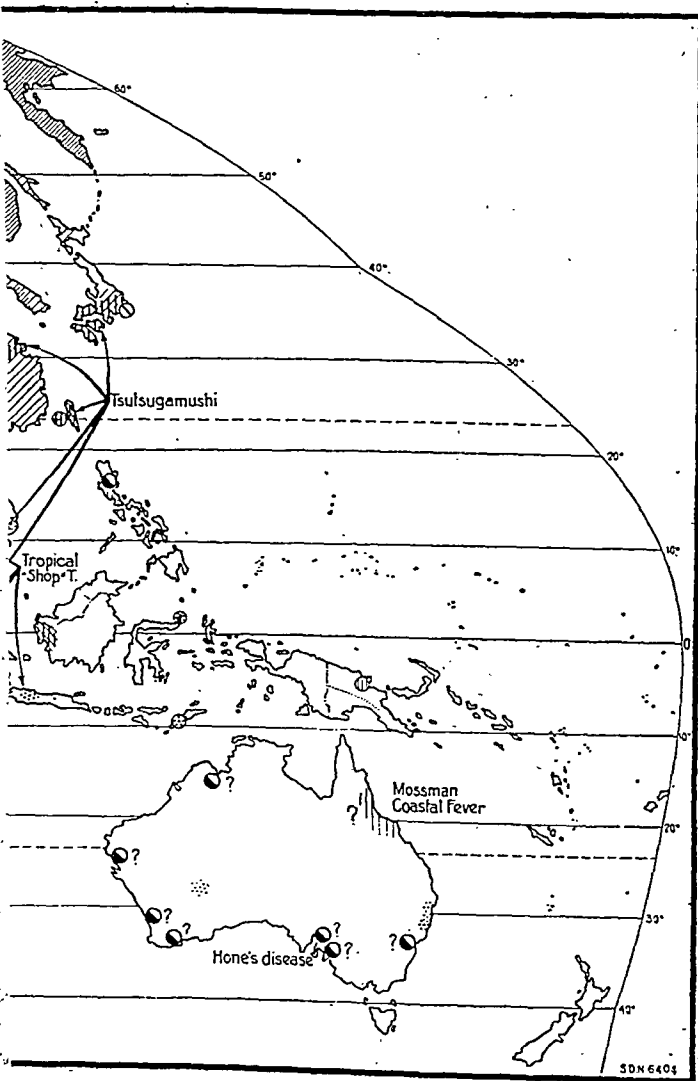


CHART 5.—Recent distrib

CHART 1.—Typhus cases reported in Poland by four-week periods from 1930-1936.
(From *League of Nations Epidemiological Report*, 1936.)

DEC.—EPID. 2

typhus is really established in a district, fear of contracting the disease, combined with terror of the appearance and acts of delirious patients, is soon widespread. Transport of food and fuel quickly breaks down, starvation threatens, the sick are abandoned, often in the roads, the houses are deserted and the terrified population flees from the infected area into a neighbouring village or another part of the town as the case may be, carrying the disease with them. Too often the hospital staffs may flee with the others.

Passing to other predisposing causes, of Murchison's two classical factors—famine and overcrowding—I should certainly lay the greater stress on the former. Typhus will spread rapidly through a population where there is no overcrowding. On the other hand as far as my experience goes, overcrowding alone is not sufficient to cause an epidemic in an endemic area, whereas famine certainly is. In many of the most heavily infected areas of Poland and Russia there was no overcrowding—in fact the reverse—as thousands of famine-stricken persons had left the villages to seek food in the town. I saw a striking example of the relatively greater importance of semi-starvation as compared with overcrowding in the Kirghiz villages north of the Caspian Sea. These villages, very remote in summer, are in winter completely cut off by 50 miles of snow from the nearest neighbour, and communications with the outside cease after the snow falls in late October. Typhus is ordinarily mildly endemic in this region, but during years of famine—the only new factor—the disease becomes widely epidemic. I had occasion to visit a number of these villages in the middle of winter, and I then realized how important a single factor, under-nourishment, may be in producing an outbreak of the disease in an isolated endemic area. The importance of semi-starvation in spreading the disease is convincingly shown by the rapidity with which epidemic typhus disappears in a district once a supply of food becomes available, and agricultural and economic reconstruction are effected. Overcrowding, inasmuch as it increases the number of lice and the facilities for the transfer of the insects, obviously must play an important part, but for the enhanced virulence required to maintain an epidemic in an endemic area, I believe that a severe degree of under-nourishment is necessary. Despite the fact that typhus is so closely related to malnutrition, it must not be forgotten that individuals who are strong and well nourished readily become infected, and very frequently die.

INFLUENCE OF CLIMATIC CONDITIONS

So far as climatic conditions are concerned, louse-borne typhus fever is a disease of cold countries and is unknown in tropical regions, though it may occur in the mountainous sectors. I have for example seen it practically on the Equator in Bolivia at a height of 15,000 feet in winter, and in Uganda the disease has occurred at an elevation of 5,400 feet. The distribution of louse-borne typhus broadly speaking may be said to cover all parts of Europe, North and Central Asia, the line of the Andes in South America and localized sections of Africa, particularly in the North. In considering the relations of louse-borne typhus to climatic conditions it must be remembered that the behaviour of endemic typhus is different from that of the disease in epidemic form. In countries where the disease is endemic it shows a constant rhythm in its visitations. Year after year the curve starts in late November or December, reaching an annual maximum in March or April, and continues until the end of June or July (Chart 1). It does not,

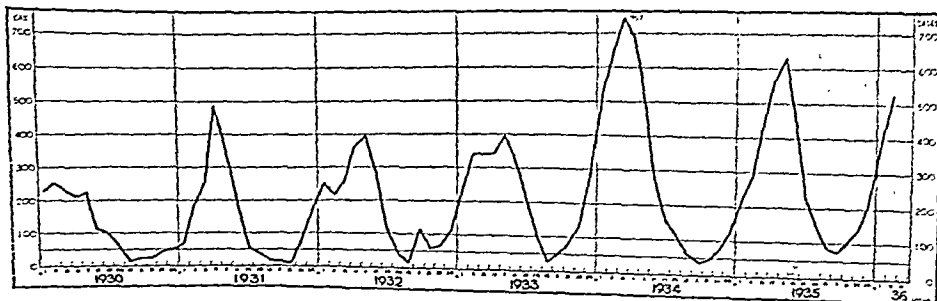


CHART 1.—Typhus cases reported in Poland by four-week periods from 1930-1936.
(From League of Nations Epidemiological Report, 1936.)

therefore, as is often thought, actually coincide with the cold period of the year but is still widespread in the heat of May and June. Epidemics of typhus on the other hand, whether in an endemic area or amongst non-immunes, can occur at any time of the year. We have had an illustration of this fact in the present epidemic in Spain, which started in April and reached its maximum during some of the hottest months of the year (Chart 2). A further example was the occurrence of typhus in Poland in 1919-1920 (Chart 3). This was superimposed on an endemic focus, but continued in epidemic

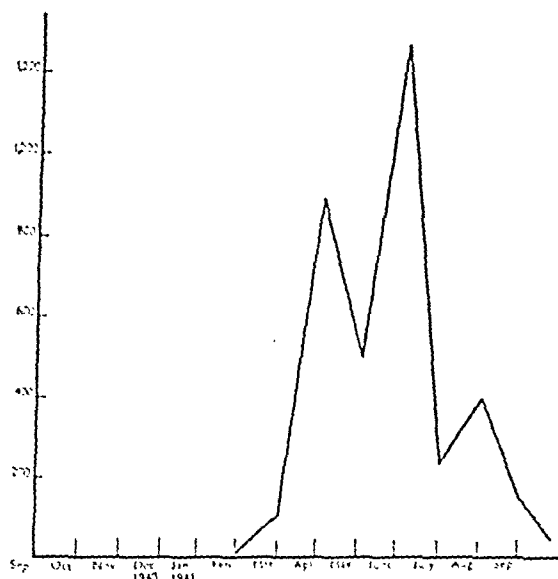


CHART 2.—Typhus in Spain, September 1940-41.

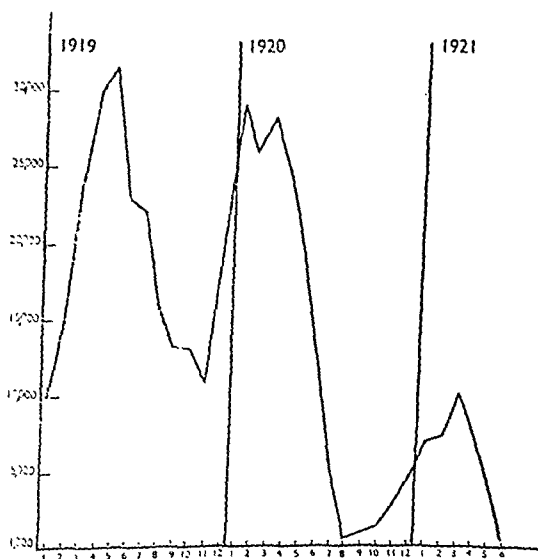


CHART 3.—Poland: Typhus cases in 1919, 1920 and to July 1, 1921.

form throughout the summer, autumn, and winter of 1919, returning to the normal endemic curve in 1920, viz. a maximum in the first six months of the year and an almost complete absence in the summer from the beginning of August onwards.

The question of the method by which the disease in endemic areas is continued between outbreaks has not been definitely answered. In an endemic typhus area one of the most striking features is the complete cessation of all cases after June or July. Repeated and careful searching during the following few months fails to reveal any cases of the disease. Though undoubtedly "missed" cases may occur during the period between gross manifestations of the disease, I believe from such investigations as I have been able to make that it is very doubtful whether these are sufficiently constant to maintain the virus from July to November or December each year. There is, of course, the possibility that a proportion of the patients may remain as *Rickettsia* "carriers". A further suggestion has been made by Cuica, Balteanu and Constantinesco (1935), who have pointed out, in the light of recent work on murine typhus: (1) the difficulty of tracing infection in non-epidemic periods from one case to another; (2) the failure to eradicate the disease from endemic areas by the isolation and delousing of definite cases; (3) the peculiar distribution of the disease and its comparative benignity in individuals born in endemic regions as compared with others. All these suggest the existence of mild, if not wholly inapparent, infections, yet ones capable of inducing immunity. An interesting point of difference between endemic and epidemic typhus relates to the virulence of the disease. In the endemic form the death-rate is generally low and fairly constant. In epidemics, although it is generally impossible to obtain accurate figures the death-rate is very variable, probably between 20% and 72%. In stating any figures, however, it must be remembered that much depends on such factors as the age of the population at risk, the hospital and treatment facilities, &c., existing, as well as the fact that figures are almost always based on hospital admissions, which do not include the mild cases amongst the children and younger members of the community. A further indeterminate factor affecting the death-rate figure is the number of non-immunes who have entered the endemic area in cases in which there has been movement of population.

The question of immunity in populations in endemic areas is of great interest. It is probably very rare for an individual to have a second attack of typhus. On the other hand individuals coming into an endemic area as adults invariably appear to get the disease more severely than adults of similar age in the endemic region. For instance in one of the endemic areas of China the death-rate amongst all Chinese adults admitted to a hospital under our supervision was 7.6% whereas that for Japanese who were newcomers to the region was 20.6%. There were probably factors which rendered these figures not directly comparable, but they bear out one's constant experience with the personnel of foreign relief units who develop the disease in endemic areas. One explanation referred to above is that population in an endemic area may be partially immunized by a milder form of typhus, possibly murine, which may even pass unnoticed by the individual.

The severity of the disease varies greatly with the age of the patient. From extreme childhood the death-rate progressively rises, until after the age of 50 the disease is practically always fatal. I cannot recollect seeing anyone over 55 who recovered, whether a native or foreigner, in an epidemic area (Chart 4, p. 8).

There is one other point in connexion with the striking variations in the virulence of the disease. It is probable that the virulence of epidemic typhus may be increased by passage through human beings, as, I think, there is no doubt the highest death-rates appear in the last stages of an epidemic. In this connexion it is of interest to note that the virus of murine typhus appears to gain in virulence in white mice on passage through animals (Giroud and Panthier, 1939, *Trop. Dis. Bull.*, 36, 984, abstracted from *Bull. Soc. path. exot.*, 32, 14).

The commonest length for the incubation period is twelve to fourteen days, though periods of five to twenty days have been recorded. In practice, it is difficult in epidemics to determine the time of the incubation as the patient has often been exposed daily to the possibility of infection. The estimation is further complicated by the fact that the patient often feels out of sorts for some days prior to the rise of temperature. Space does not permit me here to enter into the bionomics of lice, and I would only point out in this connexion that infectivity is not established in the louse until the sixth or seventh day after it has fed on a case of typhus.

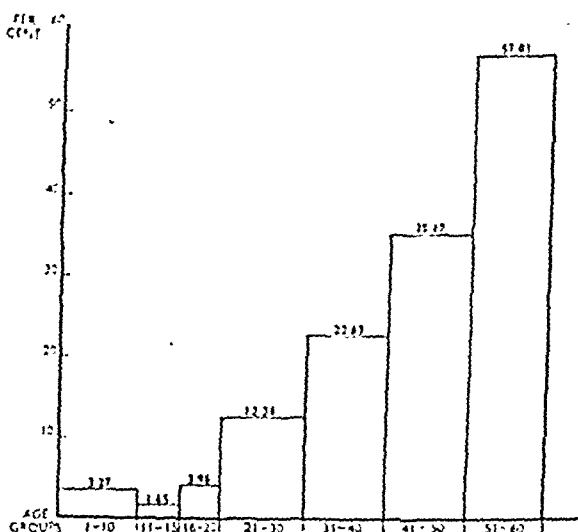


CHART 4.—Typhus mortality rate at different age-groups amongst cases admitted to the London Fever Hospital between 1862-1870. (Murchison, "Continued Fevers", page 238.)

CLINICAL ASPECTS

Turning now to the clinical aspects of typhus, the disease has been incomparably described by Murchison in his "Continued Fevers" (1873), and one has only to work amongst typhus to realize the greatness of the writing of this physician. In the very great number of cases I have seen, the symptoms manifested by the patients only serve to emphasize in my mind the accuracy of Murchison's observations. Here is his summary of the clinical characters of the disease to give a general idea of a typical case.

"Occasionally it is preceded by one or more days of slight indisposition, characterized by lassitude, vertigo, slight headache and loss of appetite, but not such as to incapacitate the patient from following his ordinary employment. With, or oftener without, these premonitory symptoms the patient is seized with slight rigors or chilliness, followed by lassitude and disinclination for exertion, frontal headache, pain in the back, pains like those from bruises in the limbs, especially in the thighs, loss of appetite and often, for a day or two, irregular chills and slight perspirations. For two or three days although the temperature may be 5° or more above the normal standard, the patient complains of chilliness, and sits close to the fire. . . . Occasionally there is nausea but rarely vomiting; the abdomen is free from pain, but there may be tenderness in the hepatic region; the bowels are constipated. . . . The respirations are somewhat accelerated and sometimes there is a slight cough. The face is flushed and dusky; the edges of the eyelids are tumefied, the conjunctivæ are injected; and the eyes water. The expression at first betokens languor and weariness but soon becomes dull, heavy, and stupid. . . . The sleep is disturbed by painful dreams and sudden starts, and after three or four nights there is talking in the sleep with slight delirium between sleeping and waking. When awake the patient is still conscious though perhaps somewhat confused in memory and intellect. With all this there is early and rapidly increasing muscular prostration; the gait is tottering, the hand shakes, and there may be tremors of the tongue; soon there is an intolerable sensation of complete exhaustion so that about the third day the patient is compelled to keep his bed. Between the fourth and seventh days, usually on the fourth or fifth, an eruption appears on the skin. It is composed of numerous spots of irregular form, varying in diameter from three or four lines to a mere speck, which are either isolated, or grouped together in patches presenting a very irregular outline, and often closely resembling the eruption of measles. At first, these spots are of a dirty pink or florid colour, and very slightly elevated above the skin and they disappear upon pressure; but after the first or second day they usually become darker and more dingy, they resemble reddish-brown stains, are no longer elevated above the skin, and do not disappear on pressure. They have

no defined margin, but merge insensibly into the colour of the surrounding skin. These spots usually come out first on the anterior fold of the axillæ and on the sides of the abdomen, and thence they spread to the chest, back, shoulders, thighs, and arms; in some cases they are first seen on the backs of the hands, they are most common on the trunk and arms and are very rarely observed on the neck or face. Along with these spots there are others which are paler and less distinct and which, from their apparent situation beneath the cuticle, have been designated 'subcuticular'. When abundant, this subcuticular rash imparts to the skin a mottled or marbled aspect, which contrasts with the darker more defined spots before described, although sometimes the two appear to merge into one another. About the end of the first week, the headache ceases, and delirium supervenes. The delirium varies in character. Occasionally it is at first acute followed by great collapse, or the noisy condition passes into low, muttering delirium. More commonly the delirium is never acute even at first. . . . The countenance becomes more dusky . . . while the prostration hourly increases. The tongue becomes dry, brown and rough along the centre and is tremulous; sordes collect upon the teeth and lips. Gradually the eruption assumes a darker shade, and about the eighth or tenth day true petechiæ of a purple or bluish tint appear in the centre of many of these spots. After three or four days, the symptoms of nervous excitement are succeeded by more or less nervous depression and stupor. At first the stupor and delirium alternate, the latter being most marked in the night time. The prostration is extreme; the patient lies on his back, moaning, muttering incoherently, or still and motionless. The expression is stupid and vacant. . . . If spoken to loudly the patient opens his eyes and stares vacantly at those about him, and when told to put out his tongue, he opens his mouth and leaves it open until desired to close it. But all this time the mind is far from inactive; the imagination conjures up the most frightful fancies, to which implicit belief is attached, and of which a distinct recollection may remain after recovery. . . . They who have passed through these mental sufferings can alone imagine their intensity. The teeth and lips are now covered with sordes; the tongue is hard and dry, dark brown or black, contracted into a ball, tremulous, and protruded with difficulty or not at all. . . . The pulse is frequent, small, weak, and undulating and not frequently intermittent or scarcely perceptible. In this state the patient may continue for many hours or several days, with life trembling in the balance, until at last the stupor passes into profound and fatal coma. But on or about the fourteenth day there is often a more or less sudden amendment. The patient falls into a quiet sleep which lasts for several hours, and from which he awakes another man. At first he is bewildered and confused and wonders where he is: but he recognizes his attendants and friends and he is now conscious of his extreme debility. The pulse and temperature have fallen; the tongue is clean and moist at the edges; there is a desire for food. . . . After two or three days the tongue becomes clean and moist all over, the appetite is ravenous, the pulse has fallen to its normal standard or is unusually slow, and the strength is rapidly regained."

It is impossible for me to add to this description, and if I refer briefly to my own clinical experience it is because I saw my cases from a different point of view to Murchison's—that of prevention rather than cure—and consequently noted more particularly the symptoms and signs from a diagnostic point of view. Moreover, in general, while Murchison was dealing practically entirely with uncomplicated cases of typhus, in the vast majority of the cases I saw there was a double or treble infection.

In the strenuous conditions under which extensive typhus epidemics occur the individual who is already weakened from malnutrition and exposure to cold often fails to notice the initial symptoms of the disease, so absorbed is he in the universal struggle for food, clothing, and warmth. Thus, though the ordinary history is that the patient had felt slightly indisposed one or two days previously to the onset of the temperature, the first sign noticed may be sudden mental confusion or a delusion in an apparently healthy individual. For instance, I recollect travelling on a locomotive from Kuibyshev to Moscow with a British colleague, a healthy man of 40. It was winter, and we crouched in front of the engine furnace with the biting wind of the Steppe on our backs. My colleague remarked suddenly that he was an apple, green on one side and red on the other. This was the onset of a severe attack of typhus. Again, it was by no means uncommon for sudden homicidal mania or suicidal attempts to be the first symptom of illness noticed, and I have known tragedies occur through this cause. An interesting fact in connexion with these sudden onsets is the long distance patients delirious with typhus will walk, in spite of the acute toxæmia of the disease, frequently under the impression that they are running away from lice. I have known delirious cases walk as much as 10 miles over the open snow of the Steppe in the depths of a Siberian winter, clad only in a shirt,

before they collapse and are found to be suffering from typhus fever. In the great majority of cases, however, acute delirium is only established at the end of the first week, and a striking feature of the patient's condition then may be his clarity and normal mental state during the day at a time when he is wildly delirious at night. Two very constant symptoms at the onset upon which I would lay stress are headache and bronchitis. The former may be frontal or occipital and its striking intensity may be a useful secondary diagnostic sign.

The clinical diagnosis of typhus fever is frequently exceedingly difficult. The conditions which favour its spread permit the occurrence of other diseases in epidemic form, and except in sporadic outbreaks, in the vast majority of the cases I have seen the diagnosis was complicated by a double infection. Thus, in Rumania, relapsing fever and malignant malaria were widely epidemic at the same time as typhus; in Russia, relapsing fever, malaria, and smallpox; in Poland, relapsing fever and typhoid fever; in China, measles and a malignant form of scarlet fever; in Bolivia, influenza and pneumonia. The clinical picture presented by one, two, or often three of these diseases in one being is obviously extremely complicated, and I have found it, therefore, of fundamental importance to be guided by the two clinical signs of the disease which are, in my experience, the most reliable. These are the absence of the rash from the face, and the fact that the rash does not appear in crops. It is said that very occasionally the typhus exanthem may occur on the face, but for all practical purposes the complete absence from the face is the most important diagnostic sign we know. The absence of "cropping" has been repeatedly stressed by those experienced in typhus since it was first noted by Stewart in 1840.

The date of appearance of the rash and its distribution on the trunk and limbs are, in my opinion, too variable to be of great value in difficult cases. Actually the exact day of the first appearance of the rash is difficult to determine, partly owing to the difficulty of saying exactly when the illness started, and partly because the rash may be extremely faint in its early stages. The commonest time is round about the fifth and sixth days, but this is by no means constant. Peacock, for instance, in a series of 28 cases stated that the exanthem appeared in two cases on the third day, three on the fourth, five on the fifth, seven on the sixth, six on the seventh, two on the eighth, two on the ninth and one on the ninth or tenth. The presence of a rash in some form is however very constant and in Murchison's cases an exanthem was noticed in 93.2% of admissions to the London Fever Hospital. The rash, however, is often faint and transient and may be absent in mild cases in children. With regard to the quantity and appearance of the rash my experience certainly bears out the fact noted by Dr. Browne Langrish as early as 1735. He writes: "Petechial spots and red efflorescence in large areas sometimes appear upon the skin . . . the brighter red they are of, so much the better sign: but when they appear of a purple brown or dusky or black colour they manifest a greater degree of putrefaction." In other words the deeper the colour of the rash, the greater its abundance, and the earlier it appears the more serious is the prognosis.

Apart from the rash, I lay some stress on the character of delirium in cases where this is marked at the onset. The delusions tend particularly to be of a terrifying type, with nightmare dreams, and may be associated with a fear of lice, which the patient imagines are consuming him, covering his pillow, dropping from the ceiling, &c. Occupational delusions are common, and what one may describe as "dissociation" delusions are often characteristic in the later stages of the illness. Thus, the patient asks for his chin to be taken off for shaving, for his legs to be hung up at the foot of the bed or in the wardrobe, or he imagines he has left a leg lying about downstairs. I have been so much struck by this type of delusion in typhus, that I feel it may be of suggestive value in diagnosis of cases in the second week of the disease.

The temperature is so often altered from the classical form by a second disease that, except in sporadic cases in non-endemic areas, it is of little guidance, at any rate in the earlier stages of the disease. On the other hand, errors of diagnosis have occurred through overlooking the fact that a temperature is invariably present in typhus fever, and cases of patients with mental illness and flea-bites have been thought to be typhus.

In sporadic cases, where the disease is commonly uncomplicated, in addition to the three classical symptoms of temperature, nervous manifestations and rash, perhaps the most suggestive symptom, in my experience, is the general appearance of the patient. The dark and heavy flush of the face, the injection of the eyes, often with petechial

hæmorrhages, and the dull stupefied look closely resemble the condition found in individuals in the later stages of an alcoholic debauch. Indeed, cases of alcoholism with vermin bites have been mistaken for typhus when the absence of temperature has been overlooked.

DIFFERENTIAL DIAGNOSIS

Of the differential diagnosis it is difficult to speak briefly, and in practice it is generally impossible definitely to diagnose the sporadic case of typhus before the rash appears on the fourth or fifth day. Until this time there are many febrile conditions which it is impossible to distinguish from typhus fever, as a Weil-Felix reaction is, of course, only present towards the end of the first week. I might, however, mention two common conditions which readily cause error in diagnosis in the earliest stage of the illness, viz. diseases of the enteric group, and the pyo-coccal infections of the central nervous system. A gradual rise of temperature, absence of the typhus facies, and the mental condition of the patient may be of assistance in excluding typhus in cases of the enteric group, but frequently it is impossible to reach a diagnosis until the results of cultures from the faeces and urine and agglutination tests are available. In the case of the pyo-coccal infections early diagnosis on purely clinical ground is often impossible, and reliance has to be placed on the results of the examination of the cerebrospinal fluid and later on the rash and the Weil-Felix reaction. The existence of paralysis or a gradual rise of temperature would be against a diagnosis of typhus. The absence of the rash from the face excludes measles and smallpox, though the prodromal rashes of the latter may closely resemble those of typhus in appearance and distribution. The fact that "cropping" does not occur in the exanthem of typhus helps in excluding the enteric group. Cases which have given rise to mistakes in diagnosis are acute febrile conditions such as pneumonia or influenza, associated with vermin bites on the trunk. In the case of vermin bites, the central puncture can readily be detected with a lens, and the lesion disappears on pressure except for the central dot. Malaria and relapsing fever can, of course, be excluded by blood films taken during the pyrexial stage. Moreover, simple infections with malaria at once react to quinine, and in the case of relapsing fever to the exhibition of salvarsan. On the other hand double infections of these diseases with typhus frequently offer extreme difficulty in diagnosis.

The diagnosis of typhus, at the end of the first week, can be determined by the Weil-Felix reaction. Briefly this depends on the fact that at the end of the first week, or earlier, the blood of cases of louse-borne typhus develops the power of agglutinating the so-called *Proteus X* strains. This reaction is specific for the typhus group of fevers and agglutination of OX19 appears to be specific for louse-borne and flea-borne typhus. The OX19 suspension issued by the Oxford Standards Laboratory should be used and the test carried out macroscopically. A strong reaction in a dilution of 1:80 or 1:100 may be regarded as positive when read by the naked eye, but in the later stages of the disease the titre usually rises to 1:1,000 or more. On the other hand, an increase in the agglutinating power of the serum, established by repeated examinations at intervals of two days, is significant, even at a titre of 1:50. From a purely epidemiological point of view the two difficulties about the use of this reaction for diagnosis are first that it occurs only relatively late in the disease—towards the end of the first week—and second, under conditions of work in an epidemic the necessary OX19 suspensions are not always available.

So far I have dealt only with the clinical aspects of the disease from the point of diagnosis during the febrile period. It is, however, often important from an epidemiological point of view to detect cases which have recently had the disease but have recovered. I should like therefore to refer to certain symptoms in convalescence which occur frequently and may persist for weeks or even months after recovery. At the beginning of convalescence the patient's mental condition is often far from normal. His mental processes are slowed down; he speaks very deliberately and cannot immediately reply to simple questions. This state may continue for some weeks and during this time an attack of mental confusion or even acute delirium may recur. A disconcerting symptom may be the sudden occurrence of transient delusions in a convalescent who is apparently absolutely normal mentally. I was much struck with the frequency in which these occurred in members of our own units. A symptom which persists long during convalescence is a slight tremor of the hands, and localized paralysis, particularly of a limb, occurs during convalescence in a number of cases. Sudden syncope may supervene

before they collapse and are found to be suffering from typhus fever. In the great majority of cases, however, acute delirium is only established at the end of the first week, and a striking feature of the patient's condition then may be his clarity and normal mental state during the day at a time when he is wildly delirious at night. Two very constant symptoms at the onset upon which I would lay stress are headache and bronchitis. The former may be frontal or occipital and its striking intensity may be a useful secondary diagnostic sign.

The clinical diagnosis of typhus fever is frequently exceedingly difficult. The conditions which favour its spread permit the occurrence of other diseases in epidemic form, and except in sporadic outbreaks, in the vast majority of the cases I have seen the diagnosis was complicated by a double infection. Thus, in Rumania, relapsing fever and malignant malaria were widely epidemic at the same time as typhus; in Russia, relapsing fever, malaria, and smallpox; in Poland, relapsing fever and typhoid fever; in China, measles and a malignant form of scarlet fever; in Bolivia, influenza and pneumonia. The clinical picture presented by one, two, or often three of these diseases in one being is obviously extremely complicated, and I have found it, therefore, of fundamental importance to be guided by the two clinical signs of the disease which are, in my experience, the most reliable. These are the absence of the rash from the face, and the fact that the rash does not appear in crops. It is said that very occasionally the typhus exanthem may occur on the face, but for all practical purposes the complete absence from the face is the most important diagnostic sign we know. The absence of "cropping" has been repeatedly stressed by those experienced in typhus since it was first noted by Stewart in 1840.

The date of appearance of the rash and its distribution on the trunk and limbs are, in my opinion, too variable to be of great value in difficult cases. Actually the exact day of the first appearance of the rash is difficult to determine, partly owing to the difficulty of saying exactly when the illness started, and partly because the rash may be extremely faint in its early stages. The commonest time is round about the fifth and sixth days, but this is by no means constant. Peacock, for instance, in a series of 28 cases stated that the exanthem appeared in two cases on the third day, three on the fourth, five on the fifth, seven on the sixth, six on the seventh, two on the eighth, two on the ninth and one on the ninth or tenth. The presence of a rash in some form is however very constant and in Murchison's cases an exanthem was noticed in 93.2% of admissions to the London Fever Hospital. The rash, however, is often faint and transient and may be absent in mild cases in children. With regard to the quantity and appearance of the rash my experience certainly bears out the fact noted by Dr. Browne Langrish as early as 1735. He writes: "Petechial spots and red efflorescence in large areas sometimes appear upon the skin . . . the brighter red they are of, so much the better sign: but when they appear of a purple brown or dusky or black colour they manifest a greater degree of putrefaction." In other words the deeper the colour of the rash, the greater its abundance, and the earlier it appears the more serious is the prognosis.

Apart from the rash, I lay some stress on the character of delirium in cases where this is marked at the onset. The delusions tend particularly to be of a terrifying type, with nightmare dreams, and may be associated with a fear of lice, which the patient imagines are consuming him, covering his pillow, dropping from the ceiling, &c. Occupational delusions are common, and what one may describe as "dissociation" delusions are often characteristic in the later stages of the illness. Thus, the patient asks for his chin to be taken off for shaving, for his legs to be hung up at the foot of the bed or in the wardrobe, or he imagines he has left a leg lying about downstairs. I have been so much struck by this type of delusion in typhus, that I feel it may be of suggestive value in diagnosis of cases in the second week of the disease.

The temperature is so often altered from the classical form by a second disease that, except in sporadic cases in non-endemic areas, it is of little guidance, at any rate in the earlier stages of the disease. On the other hand, errors of diagnosis have occurred through overlooking the fact that a temperature is invariably present in typhus fever, and cases of patients with mental illness and flea-bites have been thought to be typhus.

In sporadic cases, where the disease is commonly uncomplicated, in addition to the three classical symptoms of temperature, nervous manifestations and rash, perhaps the most suggestive symptom, in my experience, is the general appearance of the patient. The dark and heavy flush of the face, the injection of the eyes, often with petechial

the bath, and then the amount of heat and steam were increased so as to deal with the bedding and clothing. Subsequently, no further water was thrown on the stones, and the heat of the hut was allowed to dry out the material.

For furs, which are very readily infested with lice and which do not lend themselves to the ordinary methods of disinfection, crude naphthalene was used. A large box or chest was constructed at the entrance to the house and half-filled with crude naphthalene. Into this all furs and outer garments were dropped on entry to the house and left there until the following morning. I should mention that in winter in a cold country it is, of course, sufficient to hang one's garments in the open for the night for every louse to be destroyed. Whether the nits survive or not depends on the degree of cold, but there is in any case no evidence that these can transmit the disease.

In China, where padded garments have to a great extent superseded furs, brick ovens were used. These could be built rapidly in large numbers of local unburnt brick, and were heated with wood fuel or oil. The material for disinfection was introduced and left for half an hour, care being taken to hang the bedding and clothing so that complete circulation of the hot air was provided for. Ironing was found very useful on a wide scale in Russia, households being supplied with a flat iron and instructions how to use it for the destruction of lice. Other improvised methods of dealing with lice were also employed; e.g. the Serbian barrel, sack disinfector, &c., but these have two great drawbacks; firstly it is difficult to employ them on a really big scale, and secondly the fact that the garments emerge wet is a very great disadvantage, especially in a country where all moisture at once freezes hard. A further immediate measure was the acquisition and equipping of houses as hospitals for the sick, including the provision of medical stores, food, &c., and ambulance facilities.

On arrival in an area, we at once took steps to improve the nutritional conditions existing amongst the population. With typhus, especially if combined with malaria, a vicious circle is established. In the spring, the existence of these two diseases interferes with the proper sowing of the crops, with the result that the harvest is correspondingly decreased and malnutrition is increased, resulting in more typhus in the following winter and spring. This results in still less sowing during the spring and consequently more malnutrition in the following winter. To break this circle the first step was to import and issue food, and at one time over 5,000,000 were being fed daily. Concurrently, we imported large numbers of tractors, which, in lines of 30 each ploughing furrows 3 miles long, worked throughout the twenty-four hours, running at night by the aid of headlights. Seedcorn from abroad and thousands of horses from Central Asia were imported at the same time, and within two years the nutritional condition of the population was completely altered and typhus rapidly fell to its endemic normal. This was a good example of the rapidity with which epidemic typhus in an endemic area reacts to improved conditions of life.

CONTROL OF TYPHUS IN GREAT BRITAIN

The lessons to be learnt from these notes regarding control of typhus in Great Britain may be conveniently summarized as follows:

(1) All experience tragically demonstrates the great danger to which personnel working amongst typhus patients are exposed, and consequently the need for the most careful personal supervision of their activities by the medical officer in charge. The most dangerous part of the work is the search for cases, the work in the admission block of a typhus hospital, the removal and disinfection of the bedding, and also, unless careful precautions are taken, the collection of blood for the Weil-Felix reaction. In view of the lower mortality at earlier ages, only young personnel should be utilized. In the various anti-typhus units with which I have worked I would never include anyone over 40, and, where possible, only personnel under 30 were employed. No vaccine has yet been shown to protect workers under field conditions, so that in the meantime reliance should not be placed on immunization, as this may lead to relaxation of established methods of protection. The hair of all personnel working with typhus cases should be cut short and if possible shaved, including hair on the body. Protective clothing must invariably be worn, and a suitable garment for this purpose is described in the recent memorandum of the Ministry of Health on louse-borne typhus fever (Memo 252/MED). I would emphasize that protective clothing should not be worn for more than two or three hours without changing. In practice it is remarkable how rapidly a louse deposited

at any time in convalescence and in almost all cases great cardiac exhaustion is very manifest for some weeks after the illness. Localized gangrene, including cases of cancrum oris, is, of course, common in cases of typhus, and, though it often develops in the course of the illness, I have seen a number of cases where it only developed some time after the temperature fell. One or more of the above symptoms occurring in a typhus-infected area in an individual apparently weak, either from under-nourishment or as the result of indefinite illness, may suggest that the person has actually had an attack of typhus. When it is possible to carry it out, the Weil-Felix reaction is, of course, of value during convalescence in determining missed cases. The duration of the agglutinating properties of the blood is, however, variable.

With regard to second attacks, though these certainly do occur, they are rare, and, generally speaking, a single attack is considered to confer life-long immunity. Nevertheless, the great difference in the virulence of the strains, with the consequent variation of mortality in successive epidemics in the same country, and the frequency with which patients in an endemic area state that they have previously had the disease, perhaps as a child, suggest that second attacks may possibly be commoner than is generally supposed. I should add, however, that the element of doubt which may arise in diagnosis, together with the fact that there is generally only the patient's statement as to the previous diagnosis, makes any accurate conclusions impossible under epidemic conditions.

ADMINISTRATIVE MEASURES OF CONTROL OF WIDESPREAD EPIDEMICS

Though the measures taken are not likely to be applicable to Great Britain it may be of interest to outline the broader administrative steps we took when dealing with widespread epidemics of typhus fever.

The personnel of a number of units was established, including doctors, nurses, and subordinate medical auxiliaries. All were young and all were protected by the use of special clothing. Arrangements were made for the regular disinfection of the garments and for bathing the personnel. The stores required included portable baths and showers, fuel for heating water, soap, hair clippers and scissors, nail brushes, towels, &c., in addition to as good rations as it was possible to obtain. Units were sent into the various regions and were administered centrally in Poland from Warsaw, in Russia from Moscow and Kuibyshev, and, two years ago, in China from Chungking and Sian.

The next step was to put a cordon round healthy areas, with the aid of the military and barbed wire, to prevent the ingress of infected refugees. This was in many cases done locally, though eventually a cordon had to be established right across Europe, from North Poland to Rumania. Refugees were only allowed to enter this "clean" zone at certain points established on the roads and railways. Patrols watched the open country and brought stragglers into the disinfecting points. At each such point were arrangements for bathing and disinfection, and all persons passing the cordon were thoroughly "de-loused" with their belongings. The size of the work may be gathered from the fact that at one centre alone—Baranowice, on the Polish-Russian frontier in 1921—we were for a long time disinfecting each day 10,000 refugees returning to Poland from Russia. The method of disinfection varied according to the country and the apparatus available. In Poland, steam and cyanide were both used, the latter being employed on an extensive scale on the frontiers. At Baranowice, where the refugees arrived chiefly by train, a tunnel was built, into which hydrocyanic gas could be introduced. On the arrival of each train, all the passengers were given a blanket and told to strip, leaving their garments and all their belongings on the train. Each person was then bathed in hot water with soft soap and paraffin, while the train was backed into the tunnel, the engine uncoupled, and cyanide gas liberated in the tunnel. When the bathing of the refugees was completed, the train was pulled out of the tunnel by means of a rope attached to a locomotive and was allowed to air. In due course the passengers dressed, gave up their blankets, and continued on their journey. In Mesopotamia, we used a locomotive with waggons attached, into which steam, first saturated and superheated, could be passed. The train included accommodation for personnel and thus constituted a unit which could be moved to any point where typhus broke out.

In Russia, we utilized the Russian baths, with which every village is equipped. These are log huts in which fires are made under heaps of stones, which are thus heated to a high temperature. Buckets of water are thrown on the stones, the water immediately evaporating into clouds of steam. The population was first bathed and de-loused in

the bath, and then the amount of heat and steam were increased so as to deal with the bedding and clothing. Subsequently, no further water was thrown on the stones, and the heat of the hut was allowed to dry out the material.

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on the uncongenial surface of cloth crawls about until it finds an aperture at the face or wrists, through which it can gain access to the warmth and sustenance provided by the naked flesh.

Opinion is divided as to the necessity for using masks, but they should certainly be worn in any work in which the disturbance of dust may be involved, in view of the known high infectivity of the dried faeces of lice. If masks are worn, they should either cover the eyes, or be supplemented by goggles, as infection can occur through dried faecal material falling on the conjunctiva. Gloves, coming well up the forearm, must, of course, be worn. Rubber gloves are the best, as they not only fit more closely on the forearm but they also allow of the finer manipulation required in the examination of the patient and the shaving, &c. during his disinfection. While rubber gloves are the most suitable from many points of view, the ease with which they are torn constitutes in the field a grave drawback. For ambulance drivers, sanitary inspectors, disinfectors, &c., closely woven cloth gloves with a snugly fitting gauntlet are suitable. With cloth gloves, however, it is in practice difficult to avoid the formation of folds between the gauntlet and the sleeve of the garment, down which lice can crawl if the outfit is worn for any considerable length of time. Gumboots or goloshes are the most useful footwear. It is worth mentioning for the benefit of any who may be called upon to actually work and live in a heavily infected typhus area after the war, that when it is necessary to sleep in squalid peasants' houses, infested railway trains, &c., a special type of sleeping bag will be found to greatly lessen chance of infection. Such a bag, which I designed for use in Poland and Russia, was made of closely woven cotton material, about 10-12 ft. long, drawn up at the end with a circular tape which could be fastened up from the inside. Over the face were two thicknesses of gauze supported by a light metal frame. The whole bag could be boiled, and forming, as it did, complete protection from the inroads of all insects—bugs, fleas and lice—enabled one to enjoy a quiet night in comparative security.

Secondly, I would stress the rapidity with which the disease spreads, and the difficulty of tracing "contacts" in practice. In connexion with the word "contacts" there is no analogy among the diseases occurring in Great Britain to the period of infectivity of an individual who subsequently develops typhus fever. A "contact" is generally taken to cover any person exposed to infection from a patient during the time he is ill. In the case of typhus, however, the individual harbouring infected lice is capable of spreading infection from the day on which he acquires the disease to the time when he is diagnosed and disinfested, i.e. during the whole of the incubation period. The number of infected lice on an individual is, of course, far greater during the time he is actually suffering from the disease, but the incubation period cannot be overlooked from an epidemiological point of view. In this paper, therefore, the word "contacts" is used in a much wider sense than ordinarily. The incubation period may be taken as twelve to fourteen days, and after this there is a period of four to five days before the rash develops so that, even if the disease is diagnosed at the earliest possible moment (which is exceptional in sporadic cases), there is a period of sixteen to seventeen days during which the patient has been making "contacts". If, as is common in sporadic outbreaks, the disease is first recognized when the secondary cases occur, it can well be pictured how impossible it is to identify all the "contacts". We are, therefore, often driven to the possibility of dealing only with the very immediate "contacts"—the family, fellow workers, &c., whereas the most remote "contacts" are much the more numerous and are largely unknown. It is for this reason that I am strongly of opinion that the reduction of lice amongst the population generally is as important as the tracing and disinfection of more remote "contacts".

A further point that should be mentioned is that the mechanical transference of the louse is, I believe, much the commonest method of infection. A louse probably rarely voluntarily leaves an individual in cold weather except when he has a high temperature or is dead. Mechanically, however, lice are transferred in a number of ways. They may, of course, be shaken from the individual on to his pillow and sheets or, when he is undressing, on to the carpet and chairs of the bedroom. They can also be shed as their host walks or sits down, so that shop floors, railway compartments, &c., and particularly the walls and flooring of public markets become sources of infection. Perhaps one of the commonest methods of transference is rubbing shoulders in a crowd. This was certainly the case in Poland and Russia, where men visiting the market to purchase

food for our Units almost invariably returned with one or more lice upon them. In a typhus area, it is remarkable how chary people are of entering a crowd and how everyone in walking takes the greatest care not to touch any passer-by. This is a very important precaution, as a number of our personnel, not connected with typhus work, almost certainly acquired their attack of the disease from individual lice brushed off from passers-by in walking along a crowded street. Single lice can, of course, transmit the disease and an interesting fact is that many individuals in our Units, including myself, who developed typhus or relapsing fever never knew that they had had a louse upon them. It is doubtful whether infection occurs by regurgitation as in the case of the plague flea, and the ordinary method of infection is by self-inoculation—the individual scratching the bite and so crushing the louse and rubbing in the contents of its intestinal canal.

I have emphasized earlier how directly epidemics of typhus, even in endemic areas, are associated with malnutrition, and a point of great practical importance is the rapidity with which a widespread outbreak wanes in the face of improved nutritional and economic standards in the population generally. Once the disease is established in a community, even on a small scale, steps should at once be taken to combat this important epidemiological factor.

It is clear that, as the disease can readily be carried by a single louse, disinfection of the patient, the "contacts", the premises, the ambulance, and the personnel employed must be absolutely complete, and must therefore be done under the direct supervision of a medical officer. Not only must this officer assure himself beforehand that the disinfecting machine to be used is effective, but during disinfection he must make certain that the apparatus is being correctly used. We all know the temptation, for instance, there is to use little or no saturated steam and an abundance of superheated steam, or to bundle blankets together for hot-air disinfection in order to save time.

It is essential to reserve an ambulance for typhus cases and the vehicle which is chosen should be one which lends itself to complete cleansing of the interior and which affords no harbourage to insects. The patient, prior to removal, should be completely enveloped in an extra long sheet and lifted on to the stretcher so that any lice shaken off in the process are caught in the wrapping.

I need not enter into all the details of the disinfection of cases on arrival at hospital prior to admission to a ward, as these will suggest themselves in the light of my remarks with reference to the danger of the transference of the disease to personnel. There are, however, three important points: First, every hospital intended for typhus cases should be provided beforehand with a properly equipped admission block and a disinfecting apparatus and should be staffed by young personnel trained in thorough disinfection. Second, in addition to the destruction of the lice, the greatest care should be taken to ensure that all nits are also dealt with. The blood of a typhus patient is highly infectious for the two weeks of illness, and a nit which has been overlooked may hatch out, feed on the patient, and eventually cause the disease in the nurse. For this reason it has been recommended that all patients should undergo a second disinfection a week after they have been admitted to hospital. Third, it is useless to place any reliance on disinfectants in the bath given to the patient, as it is impossible to utilize a sufficiently strong solution of an ordinary disinfectant to ensure the death of the insect in the relatively short time in which it is exposed. The removal of the lice by bathing in mechanical and ordinary soap, possibly soft soap mixed with paraffin, is sufficient if conscientiously applied with a nail brush after the patient has been shaved.

The identification of the "contacts" of a case of typhus offers very considerable difficulty and indeed, as mentioned above, it is difficult to define what actually constitutes a "contact" from the point of view of disinfection. In the case of immediate "contacts"—members of the same family, co-workers, &c.—the most thorough disinfection must of course be carried out. With regard to more remote "contacts" the decision must, in my opinion, be made in the light of individual circumstances. Any individual who carries lice in an area where typhus exists is a danger to himself and to other people, and as such must be disinfested. The position is much more difficult in the case of persons normally clean who may, possibly only for a few moments, have been in contact with an individual who subsequently developed typhus. Probably the best method of dealing with these cases is to explain the danger to the individual concerned and advise him to undergo disinfection.

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Opinion is divided as to the necessity for using masks, but they should certainly be worn in any work in which the disturbance of dust may be involved, in view of the known high infectivity of the dried faces of lice. If masks are worn, they should either cover the eyes, or be supplemented by goggles, as infection can occur through dried fecal material falling on the conjunctiva. Gloves, coming well up the forearm, must, of course, be worn. Rubber gloves are the best, as they not only fit more closely on the forearm but they also allow of the finer manipulation required in the examination of the patient and the shaving, &c. during his disinfestation. While rubber gloves are the most suitable from many points of view, the ease with which they are torn constitutes in the field a grave drawback. For ambulance drivers, sanitary inspectors, disinfestors, &c., closely woven cloth gloves with a snugly fitting gauntlet are suitable. With cloth gloves, however, it is in practice difficult to avoid the formation of folds between the gauntlet and the sleeve of the garment, down which lice can crawl if the outfit is worn for any considerable length of time. Gumboots or goloshes are the most useful footwear. It is worth mentioning for the benefit of any who may be called upon to actually work and live in a heavily infected typhus area after the war, that when it is necessary to sleep in squalid peasants' houses, infested railway trains, &c., a special type of sleeping bag will be found to greatly lessen chance of infection. Such a bag, which I designed for use in Poland and Russia, was made of closely woven cotton material, about 10-12 ft. long, drawn up at the end with a circular tape which could be fastened up from the inside. Over the face were two thicknesses of gauze supported by a light metal frame. The whole bag could be boiled, and forming, as it did, complete protection from the inroads of all insects—bugs, fleas and lice—enabled one to enjoy a quiet night in comparative security.

Secondly, I would stress the rapidity with which the disease spreads, and the difficulty of tracing "contacts" in practice. In connexion with the word "contacts" there is no analogy among the diseases occurring in Great Britain to the period of infectivity of an individual who subsequently develops typhus fever. A "contact" is generally taken to cover any person exposed to infection from a patient during the time he is ill. In the case of typhus, however, the individual harbouring infected lice is capable of spreading infection from the day on which he acquires the disease to the time when he is diagnosed and disinfested, i.e. during the whole of the incubation period. The number of infected lice on an individual is, of course, far greater during the time he is actually suffering from the disease, but the incubation period cannot be overlooked from an epidemiological point of view. In this paper, therefore, the word "contacts" is used in a much wider sense than ordinarily. The incubation period may be taken as twelve to fourteen days, and after this there is a period of four to five days before the rash develops so that, even if the disease is diagnosed at the earliest possible moment (which is exceptional in sporadic cases), there is a period of sixteen to seventeen days during which the patient has been making "contacts". If, as is common in sporadic outbreaks, the disease is first recognized when the secondary cases occur, it can well be pictured how impossible it is to identify all the "contacts". We are, therefore, often driven to the possibility of dealing only with the very immediate "contacts"—the family, fellow workers, &c., whereas the most remote "contacts" are much the more numerous and are largely unknown. It is for this reason that I am strongly of opinion that the reduction of lice amongst the population generally is as important as the tracing and disinfestation of more remote "contacts".

A further point that should be mentioned is that the mechanical transference of the louse is, I believe, much the commonest method of infection. A louse probably rarely voluntarily leaves an individual in cold weather except when he has a high temperature or is dead. Mechanically, however, lice are transferred in a number of ways. They may, of course, be shaken from the individual on to his pillow and sheets or, when he is undressing, on to the carpet and chairs of the bedroom. They can also be shed as their host walks or sits down, so that shop floors, railway compartments, &c., and particularly the walls and flooring of public markets become sources of infection. Perhaps one of the commonest methods of transference is rubbing shoulders in a crowd. This was certainly the case in Poland and Russia, where men visiting the market to purchase

Laboratory Investigations on Typhus

By G. M. FINDLAY, M.D.

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THE following points are dealt with: (1) Diagnosis. (2) Specific treatment. (3) Prophylactic immunization.

(1) *Diagnosis*.—Unequivocal evidence that the disease is typhus can be obtained only by isolation and passage of the Rickettsiae in susceptible animals. In suspected cases of typhus blood should be removed and inoculated intraperitoneally into guinea-pigs and rats. When whole blood is employed it is rare to obtain Rickettsiae after the seventh day. The blood should be allowed to clot in the refrigerator, the serum removed and the clot ground up in physiological saline; if injected intraperitoneally in doses of 2 to 5 c.c. a positive reaction can often be obtained during the whole febrile period and sometimes during convalescence. If the Rickettsiae of exanthematic typhus are present in the blood of the patient a febrile reaction about 40° C. (104° F.) can be expected in the guinea-pig after an interval of about seven to twelve days: if the Rickettsiae of murine or endemic typhus are present the guinea-pigs will exhibit an orchitis in addition to fever. In guinea-pigs infected with exanthematic typhus there are no macroscopic lesions post mortem, but histologically in the brain are to be seen small collections of round cells, not necessarily related to the perivascular spaces. In guinea-pigs infected with murine typhus the tunica vaginalis is highly inflamed. Rats inoculated intraperitoneally with blood containing murine typhus Rickettsiae show a febrile reaction and some degree of inflammation of the tunica vaginalis: when exanthematic typhus Rickettsiae are present there is either no or only a transient febrile reaction in rats. In Mexico and China especially, strains of Rickettsiae have been isolated which appear to be intermediate between murine and exanthematic typhus since they cause only slight scrotal reactions in the guinea-pig.

Passage of exanthematic typhus in guinea-pigs is best carried out by intraperitoneal injection of 1 c.c. of equal parts of 10% suspensions of infected guinea-pig spleen and brain in physiological saline. The murine strain can also be passaged in series by intraperitoneal injection of scrapings of the tunica vaginalis in guinea-pigs or in rats by intraperitoneal injection of a 10% suspension of brain.

Until recently it was thought that the "nodules" found histologically in the brains of guinea-pigs were specific for typhus. Recently, however, Pinkerton and Henderson (1941) have described cases of toxoplasmosis in adults in which injection of the blood intraperitoneally into guinea-pigs caused a febrile reaction closely resembling that seen in experimental typhus and also small collections of round cells in the brain. Toxoplasms may be found in the exudate from the peritoneal cavity of the guinea-pigs. Human toxoplasmosis has not been described in this country although toxoplasms have been isolated from field voles (*Microtus agrestis*), ferrets, wombats at the Whipsnade Zoo and domestic rabbits.

The most commonly used test for typhus is the agglutination by the sera of typhus patients of the O strain of *Proteus* X19. A positive agglutination reaction should be obtained macroscopically by the end of the first week and often by the fourth or fifth day. Romero Escacena (1941), in an account of the typhus epidemic that occurred in Spain in the early months of 1941, mentions one case, however, in which the Weil-Felix reaction did not become positive till the nineteenth day of the disease, while in others the reaction was negative on the sixth, seventh or eighth day of illness, though subsequently it became positive. The reaction persists for some months after convalescence has been reached. The maximum titre against *Proteus* OX19 varies very much, not infrequently the serum may agglutinate the organism in a dilution of 1:5,000 to 1:10,000, sometimes even up to 1:100,000. There is often a relationship between the maximum titre and the severity of the disease, the titres being highest when the disease is grave. OX2, another variety of *Proteus*, is agglutinated to a lower titre by typhus sera while with the OXK strain of *Proteus* which agglutinates with tsutsugamushi sera there is usually no agglutination or only a faint trace.

In murine typhus the Weil-Felix reaction presents the same characteristics as in the exanthematic form but both OX19 and OX2 are agglutinated in lower titres, the maximum being rarely above 1:10,000 and usually between 1:200 to 1:2,000 (Spencer and Maxcy, 1930). In carrying out the Weil-Felix test it is important to include a known negative serum in order to make certain that the strain of *Proteus* used is not spontaneously agglutinable.

There are certain other diseases in which a positive Weil-Felix reaction may be obtained.

tion. It is clear that it is impossible either to trace or disinfest any but a small proportion of the more remote "contacts" of a man who for twelve days and possibly sixteen has been living an ordinary life, travelling by bus or train, at the cinema, in shops, &c., and who has subsequently developed typhus. It is for this reason that it is essential to insist on the general disinfestation of the population in addition to measures against all known "contacts". "Contacts" should be kept under surveillance for three weeks.

With regard to the disinfestation of the premises, cyanide gas is the most satisfactory. Indeed we have no evidence that sulphur or formalin as applied in ordinary disinfection has any certain lethal effect on lice or Rickettsia bodies. The fact that armchairs, sofas, wardrobes, &c., are all likely to be infective in the bedroom of a typhus case, and that none of these can readily be disinfested in an ordinary disinfector, apart from the danger of handling and transporting them, makes disinfestation *in situ* essential. Bedding and clothing of the patient and the immediate "contacts" should be particularly thoroughly disinfested. It is important to remember that lice may drop from such material and the disinfesting station should, therefore, be reserved entirely for typhus bedding and clothing until this has been dealt with. Subsequently, and before any other material is introduced for disinfection, the station must be thoroughly cleansed. One point that may be overlooked is the fact that clothing may have been sent to a laundry from a case who later developed typhus. The method to be employed for disinfestation will generally be determined by the disinfesting apparatus available. Saturated steam is, of course, thoroughly effective as is hot air, provided the garments, &c., are so suspended as to allow of free circulation of air about them. Rickettsia and lice are both killed by exposure to dry heat for five minutes at a temperature of 140° F., and instantly by exposure to a temperature of 212° F. when moist heat is utilized. Finally, in this connexion, when disinfestation is completed the cleansing of the ambulance, bedding van, disinfesting station, and the personnel engaged must not be overlooked.

Immediately typhus occurs, all practitioners should be kept informed of the existence and extent of the disease in their locality, and if necessary a brief description of some of the important diagnostic signs should be circulated. The ease with which cases of typhus are missed is well shown in figures given by Davidson and Cruickshank. In the period 1901 to 1926, 87 groups of cases occurred in Glasgow, and in 36 of these the diagnosis of the primary case was missed. The analysis of the diagnosis of the first case in each group was: Typhus 51; enteric fever 22; pneumonia 5; other diseases 9.

As typhus may masquerade under such varying clinical forms, all death certificates should be scrutinized by the Medical Officer of Health for suspicious deaths and he should keep in constant touch with all doctors in his area engaged in general practice. In addition a close search for cases must be maintained by the Medical Officer of Health and his staff, particular regard being paid to cases of illness where a doctor is not in attendance. In view of the mildness of the disease amongst children, absentees from school must be closely investigated. Propaganda informing the public how the disease is carried, how lice can be killed and outlining the disinfesting facilities available is of the greatest importance.

CONCLUSION

Though under-nourishment and fatigue render individuals more liable to typhus, the disease will readily attack individuals in perfect health and living under good conditions, and if typhus is imported into this country at the present time, the first case may well not be associated with conditions of poverty, dirt or malnutrition. The present constant air-traffic between the Continent and this country is limited to a type of individual who is unlikely to harbour lice, but who may easily be infected abroad and develop the disease after arrival. Refugees and prisoners of war arriving in this country, are, of course, in another category. The great variation in the virulence of the strains constitutes a further complication, and it must not be forgotten that an initial case in Great Britain occurring in a well-nourished subject may be either of the grave, classical type, or very mild, and thus readily overlooked. I would emphasize the rapidity of the spread of the disease in a louse-infested population, the difficulty of the diagnosis, the necessity for complete thoroughness in disinfestation and the importance of detailed supervision of the protection of personnel, in view of the great danger to which they are exposed.

Although these vaccines have been used on a large scale there are no accurate figures by which to assess their values. In Europeans severe reactions indistinguishable from murine typhus have been recorded. In Spain a living murine typhus virus is reported by Romero Escacena (1941) to have been used on about 500 persons in Seville. Although there was a complete absence of reaction after the injection there was likewise a complete absence of evidence that it in any way influenced the course of the epidemic among the asylum inmates on whom it was used.

Apart from the danger of severe reactions it seems inadvisable to use living murine Rickettsiae in a country where the population is louse-infested, in view of the possibility that after passage through the louse, the murine typhus Rickettsia may be converted into the exanthematic typhus Rickettsiae.

Ruiz Castaneda (1941) has proposed the use of a killed vaccine prepared by intranasal instillation of murine typhus Rickettsiae into rats and mice. Since the exact antigenic relationship of murine and exanthematic typhus Rickettsiae is at present unknown, it seems inadvisable to employ murine Rickettsiae when a similar vaccine can be prepared from killed exanthematic typhus Rickettsiae.

The use of killed exanthematic Rickettsiae appears to have been initiated by Da Rocha Lima (1918), who found that repeated injections of phenolized emulsions of lice infected with classical typhus conferred some degree of immunity.

This work was continued and extended by Weigl (1930) who used the phenolized intestinal contents of lice infected *per rectum* with *Rickettsia prowazekii*. The method has been used in Poland, Slovakia and Ethiopia and appears to have been of considerable value (Radlo, 1937; Drbohlav, 1938; Mariani, 1939). The serological response of individuals after vaccination by this method has been studied by Liu, Zia and Wang (1938). Both the Weil-Felix reactions and specific typhus rickettsial agglutinins were positive in the majority of sera from persons recently vaccinated, but no parallelism or constancy between the titres of the respective antibodies was noted. The rickettsial agglutinins, however, waned more rapidly than the *Proteus* agglutinins. It seems probable that immunization by this method should be re-undertaken at the beginning of each typhus season. This conclusion has also been reached by Parker (1941) after fifteen years' experience of a Rocky Mountain spotted fever vaccine prepared from the tissues of injected ticks. Apart, however, from the delicate technique of injecting Rickettsiae into the rectums of lice, Weigl's method of immunization implies a large staff of immune persons on whom the lice can be fed while the Rickettsiae are multiplying in the intestines of the lice.

Two other methods of vaccinating by killed exanthematic typhus Rickettsiae have been introduced. Durand and Giroud (1940) prepared a formalinized vaccine from the lungs of mice injected intranasally with *Rickettsia prowazekii*, while Cox and Bell (1939) prepared vaccines from the yolk sacs of developing chick embryos. Neither of these vaccines gives rise to any immediate or remote reaction on injection. Although it is easy to induce immunity in guinea-pigs by the injection of either of these vaccines their application to human immunization has not been exhaustively studied. Investigations are now proceeding on a number of volunteers who have been injected with the egg yolk vaccine prepared in various laboratories; of 37 persons who have received injections with one egg yolk vaccine only one has shown any rise in the agglutinins for *Proteus* OX19 and in specific rickettsial agglutinins, while one serum has shown rickettsicidal bodies when tested intradermally in rabbits by Giroud's method. These poor results combined with the fact that laboratory workers who have been immunized by the egg yolk vaccine have subsequently contracted typhus suggest that further research must be undertaken to determine a satisfactory method of immunization against exanthematic typhus. At present at least three injections of the vaccine must be given, probably at least once a year; a method which will confer immunity after a single injection would be far preferable.

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It is as well to recall that typhus may cause non-specific increase in the agglutination reactions for typhoid organisms: in these anamnestic reactions the increase is almost entirely in the flagellar or H agglutinins.

Apart from the agglutination of *Proteus* OX19 by the sera of typhus patients it is possible to elicit a specific agglutination of typhus Rickettsiae, as first shown by Otto and Dietrich (1917) and Weigl (1923). Fairly consistent results have been obtained with the sera of patients recovered from exanthematic typhus in Libya by using suspensions of exanthematic typhus Rickettsiae obtained from the yolk sac of developing chick embryos. The conditions for optimal agglutination, the times at which the agglutinins appear or disappear and the maximum titres obtained require further investigation. The same applies to the complement-fixation test. Positive results have been obtained with endemic typhus antigens from the yolk sac of chick embryos by Bengtson (1941) and similar results have been obtained with exanthematic typhus Rickettsiae obtained from the same tissues. Further studies are in progress.

Attention must also be drawn to another test by which it is possible to demonstrate the existence of rickettsicidal bodies in the sera of persons who have recovered from typhus. Giroud (1938) has shown that when typhus Rickettsiae are injected intradermally into rabbits there occurs a specific nodular reaction in the skin. By mixing dilutions of the serum to be tested with suspensions of Rickettsiae before intradermal injection it is possible to show the presence in sera from convalescent cases of typhus of rickettsicidal immune bodies as well as the titre of these immune bodies.

(2) *Specific treatment.*—No chemotherapeutic compound capable of influencing the course of typhus in man or experimental animals has yet been found. The use of convalescent serum was first suggested by Nicolle and Conseil (1920) who found that 20 c.c. of convalescent serum given during the incubation period might produce some attenuation of the disease but was very little good in the treatment of acute infections, a result recently confirmed by Romero Escacena (1941) in Spain. Later Nicolle and Conseil (1925) suggested the use of sera from donkeys inoculated with infected guinea-pig brain, while still more recently Durand and Balozet (1940) have used the sera of horses hyperimmunized by repeated injections of mouse lung infected with exanthematic typhus Rickettsiae. A small amount of hyperimmune horse serum is available in this country. Kuratichin, van der Scheer and Wyckoff (1940) have also prepared a serum in rabbits by injection with egg yolk sac material infected with exanthematic typhus. The serum, which can be purified by the same means used in purifying antipneumococcal rabbit serum, has a high neutralizing power when tested in guinea-pigs, but its use in the treatment of human beings has not yet been attempted.

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In North Africa living murine typhus Rickettsiae have been used, either after passage through mouse brain and envelopment with egg yolk (Lairgret *et al.*, 1937; Lairgret and Durand, 1939), or after attenuation with bile (Blanc, 1938). Rickettsiae from the faeces of rat fleas have also been used by Blanc and Baltazard (1940) in Morocco.

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Proceedings of the Royal Society of Medicine

Chairman—A. M. H. GRAY, C.B.E., M.D., President of the Society

OCCASIONAL LECTURE

[Delivered on December 12, 1941]

On the Need for Abandoning Much in Immunology that has been Regarded as Assured

By Sir ALMIROTH E. WRIGHT, M.D., F.R.S.

PART I

TECHNIQUE

It will be well to deal first with the methods which have been employed in these researches.

(1) *An Improved Technique for Making a Glass Pricker*

Three forms of glass prickers can be used for drawing blood from the finger.

The *first* is the ordinary glass pricker which is made by drawing out a piece of glass capillary tube, without giving the matter any special attention, in the peep flame of a Bunsen burner. The conical pricker so obtained has every possible demerit. It opens up a minimum of capillaries, and so furnishes very little blood; and it takes toll for what little blood it gives in the form of a good deal of pain—for the rapidly expanding cone, which follows upon the sharp point, forcibly prises the tissues asunder.

The *second* form of glass pricker is the *gouge pricker*, which was suggested by the late Dr. Clemenger, one of my fellow-workers. This is made by inserting a pin into the open end of a capillary tube, and breaking away its walls, first on one side and then on another, until the capillary tube, which was originally cylindrical, has been converted into a sharp-pointed gouge.

The *third* form is the *sliver pricker* which is illustrated in figs. 1 and 2. This consists of a pointed, thin, lancet-shaped blade which goes in almost painlessly and gives, because it cuts across a great number of capillary vessels a copious flow of blood. The method of making a *sliver pricker* is shown in fig. 2.

We begin by choosing a fairly stout capillary pipette, and preferably one which runs out, as shown in the figure, into an eccentrically disposed stem. We then take a *sharp*



FIG. 1.

glass-cutting knife or a writing diamond and make a *very short* and *very superficial* scratch on the upper surface of the neck of the pipette before it runs out into the stem. This done, we, holding the pipette in the position shown in the diagram, place our thumb-nail underneath the lower wall of the capillary stem about 2 cm. from the scratch and then bring upward pressure to bear here while downward pressure is, so as to make this point the fulcrum, applied to the upper wall of the capillary stem at a point about 2 cm. distally to where the thumb-nail is placed.

By this procedure we split off a sharp-pointed and very thin sliver from the lower wall of the capillary tube. With a pricker of that kind it is quite easy to obtain 1 c.c. or even more of blood from one or more almost painless pricks made at the base of the finger-nail.

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Discussion.—Professor P. A. BUNTON said that in a clean community, with adequate facilities for washing of the person and of underclothes, the body louse could not exist. The body louse was common at present in the tramp community, and he thought it was also more common than was generally suspected in labour camps, among men employed in building jobs and the like, where washing facilities might be small. On the subject of head lice, K. Mellanby had collected statistics which showed that in schoolgirls in cities infestation was as high as 50%, among boys it was never quite so high, while among adolescents the rate remained high in girls but fell away among boys. Opportunities had recently arisen of collecting data with regard to young women going into certain types of employment, and the results were consistent with Mellanby's figures. There were many cities and industrial areas in this country where the infestation rate of head lice among girls and young women was not far short of 50%. The relation of the head louse to typhus fever was not known, but the head louse could not be satisfactorily distinguished from the body louse, and it was known that they were very closely related. In considering the transmission of typhus the safe line to take would be to suppose that the head louse might be dangerous, though perhaps not as dangerous as the body louse.

Dr. FELIX: The previous speakers were not quite correct in stating that the *Proteus* O/19 reaction appears too late in the disease to be of assistance in the early diagnosis of cases. In fact the reaction is positive in about 75% of cases on or before the fourth or fifth day of illness, and the maximum titres reached shortly before or after defervescence are very high. The remaining 25% of cases show a significant O/19 reaction about the sixth or seventh day, and the maximum titres reached are low. The latter group comprises the most severe cases, including the fatal cases, and the very mild, abortive cases. The type of the curve of agglutinin production is thus related to the clinical course of the disease.

It is also not quite correct to state that the agglutinins disappear too early in convalescence to be of value in the search for missed or abortive cases of typhus. How long after the attack a significant O/19 titre is found depends on the height of the maximum titre that has been attained during the attack. In most cases a retrospective diagnosis can be made from the agglutination test during three or four months following the attack of typhus. In some cases a significant agglutinin titre persists for much longer time. The O/19 reaction also is positive in cases of so-called unapparent infection, showing no clinical symptoms at all. Such cases are of particular epidemiological importance in countries where typhus is endemic.

I cannot share the pessimistic view expressed by Dr. Findlay with regard to active immunization against louse-borne typhus. There was little doubt that Weigl's louse vaccine, made from the dissected guts of infected lice, was an effective immunizing agent. The view has long been held that the virus in the infected louse gut has some peculiar properties which tissue virus from man or laboratory animals does not possess. It now appears to be likely that this is due to quantitative rather than qualitative differences, and if this is the case one is justified in expecting further improvement in the methods of growing and preserving Rickettsiae to yield vaccines of an efficacy equal to that of Weigl's louse vaccine.

- (3) *On the Employment of Centrifugalization for Bringing the Microbes and Leucocytes in a Phagocytic Mixture into More Effective Contact than when these are Allowed to Gravitates Down Together in Capillary Tubes Laid on Their Side in an Incubator*

Centrifugalization provides, as becomes obvious the moment the idea suggests itself, a much better agency for bringing the microbes into contact with the leucocytes than did the original technique of laying the capillary pipettes on their side in an incubator and letting the leucocytes settle down, through the microbes suspended in serum.

The advantages of centrifugalization over sedimentation are:

(1) Centrifugalization undertaken at the laboratory temperature and continued for only a minute or even less than a minute gives always a larger phagocytic intake than gravitation conducted at blood heat and prolonged for the usual quarter of an hour.

(2) Again, when we centrifuge phagocytic mixtures containing microbes, normal washed corpuscles and normal salt solution, we obtain always a small amount of spontaneous phagocytosis; while gravitation in the incubator gives an almost completely blank result. This holds true unless the incubation is unduly prolonged when another factor, to which attention will be drawn later, comes into consideration.

(3) Again, when we are dealing with a phagocytic mixture composed of microbes, washed leucocytes and heated serum centrifugalization gives us a bacterial ingest of fully one-third of that given by the original (unheated) serum, whereas gravitation in the incubator gives, when heated serum is employed, an almost negligible phagocytic intake.

(4) Finally, when we are dealing with blood which has given only a small epiphyllactic response to an *in vitro* inoculation, gravitation in the incubator not infrequently gives a blank result, whereas centrifugalization gives one which is definitely positive.

The following points in connexion with centrifugalization of phagocytic mixtures require to be kept in mind:

(1) Centrifugalization can be carried out effectively at ordinary room temperature.

(2) The phagocytic mixture should (it must, of course, be enclosed in a capillary tube which is sealed off at either end) be centrifuged first for about forty-five seconds in one direction and then for the same time in the reverse direction. But the centrifuge may be arrested as soon as ever we have a sharp line of demarcation between the corpuscular sediment and the supernatant fluid of the phagocytic mixture.

- (4) *On the Elimination of all Unphagocytosed Microbes from the Phagocytic Mixtures by Amputating, after Centrifugalization, the Upper Ends of the Capillary Tubes*

The capillary tubes which contain the phagocytic mixtures are, after centrifugalization, amputated at the line of demarcation between the sediment and the supernatant fluid.

But we have now in our amputated tubes a solid and intractable mass of corpuscles; and we must, if we want to make good films, bring our corpuscles into suspension in an equal volume of a not too viscid fluid: Having diluted a small portion of the serum which was pipetted off from our centrifuged defibrinated blood with an equal volume of normal salt solution; and having provided ourselves with an "evacuating pipette" (an evacuating pipette is a simple capillary pipette drawn out into a stem of sufficient fineness to go to the bottom of our amputated tubes) we inscribe upon its stem a fiducial mark at a point which will provide about sufficient room for housing the amount of corpuscular sediment provided in a single tube. We then aspirate from each amputated capillary tube in turn all the corpuscular sediment and then take up into our pipette an equal volume of diluted serum. We then blow these quanta out on to a paraffined slide, and, after mixing, proceed to make our films. In drawing off the corpuscular sediment from our amputated capillary tubes care must be taken to stop off the inflow into our pipette immediately the supply of corpuscles is exhausted, for a sudden indraft of air would scatter the corpuscular deposit all over the interior surface of the pipette.

- (5) *On the Omission from the Phagocytic Count of the Uncountably Large Numbers of Microbes which may be Ingested by Individual Leucocytes when the Microbes in the Phagocytic Mixture are Agglutinated by the Serum Employed*

Attention has not, up to the present, been directed to the fact that staphylococci may be

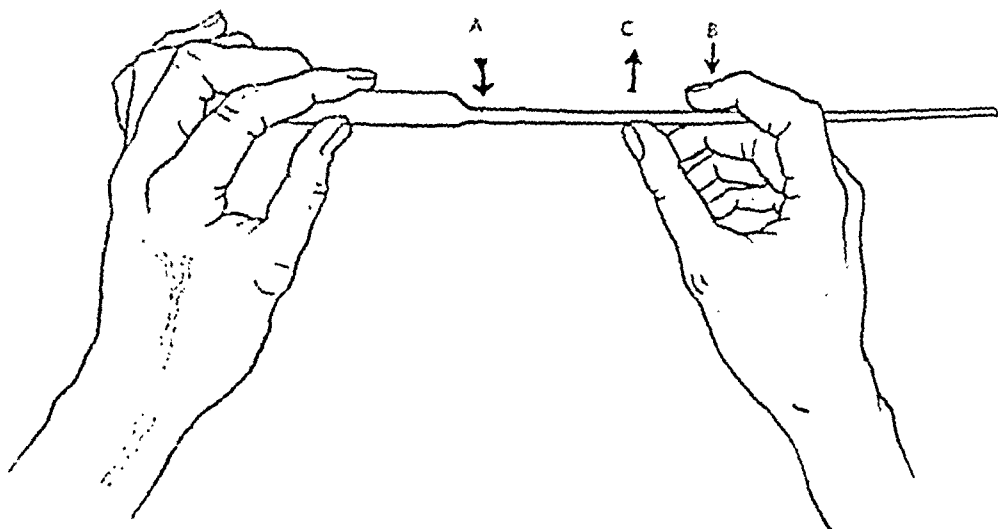


FIG. 2.

A = Superficial cut. B = Point at which downward force is applied. C = Point at which upward force is to be applied.

(2). *On an Improved Technique for Washing Blood Corpuscles on a Centrifuge, and for Obtaining in as Short a Time as Twenty Seconds a Corpuscular Sediment Containing a very Large Percentage of Leucocytes*

The method of washing blood corpuscles prescribed in my "Technique of the Test and the Capillary Glass Tube" consisted in collecting blood direct from the finger into about six or eight times its volume of salt solution; and then centrifuging twice over until in each case all the corpuscles have been brought down. This technique can be improved upon.

One of the demerits of the method just described is that centrifugalization of the unclotted blood will, if it is not arrested betimes, bring down blood platelets into the first, and again into the second corpuscular deposit.

The presence of platelets in a phagocytic mixture must be avoided for platelets reduce, and may—as was shown ten or more years ago by my then fellow-worker Dr. E. J. Storer—entirely abolish phagocytosis.

A corpuscular deposit free from platelets can always be obtained by preparing our washed corpuscles from defibrinated blood instead of from blood taken direct from the finger.

A second blemish on the method is that it involves two centrifugalizations; and complete centrifugalizations occupy a considerable time.

This can be avoided if we, before proceeding to wash our corpuscles, defibrinate our blood and then centrifuge and pipette off the serum.

The corpuscular sediment is then transferred to a centrifuge tube about 10-12 cm. long which holds sufficient salt solution to dilute the corpuscular deposit 25 or more times. Under these conditions a single washing will reduce the serum in the corpuscular deposit to a perfectly negligible amount.

It is important to note that it is unnecessary to continue the centrifugalization for more than twenty to twenty-five seconds. By that time very large numbers of leucocytes will have been carried to the bottom and the corpuscular deposit will be far richer in leucocytes than was the blood with which we started.

The more rapid carrying down of the leucocytes in the centrifugalized salt solution is no doubt due to their somewhat smaller specific gravity being more than offset by their larger dimensions and spherical shape.

quanta of Tuberculin B.E. 8 to 48% of the polynuclear leucocytes were completely filled with microbes.

In later experiments I have seen with similar phagocytic mixtures nearly 100% of the polynuclear leucocytes absolutely gorged with staphylococci.

Results of this sort which previously were unaccountable find now an easy explanation in the fact that both Tuberculin B.E. and heated serum agglutinate the staphylococci of phagocytic mixtures, while both operate also as phagocytic "stimulins".

- (6) *On a Procedure which Admits of a Rough Discrimination of the Role Played in "Phagocytic Mixtures" by (a) the Opsonic Action Exerted by the Serum on the Microbes; (b) a Phago-incitor Stimulin Action Exerted by the Serum upon the Leucocytes; and (c) the Phago-incitor Action Exerted upon the Leucocytes by the Microbes which were previously Credited with being an Inert Phagocytic Pabulum*

The traditional procedure for measuring phagocytic power does not distinguish between those three chemical activities. It does not even contemplate the possibility of any except an opsonic chemical action taking place in phagocytic mixtures.

Much can be learned on this question by carrying out the phagocytic tests in two operations instead of in one.

In the original phagocytic procedure equal parts of washed normal corpuscles, of the patient's serum, and of the microbic suspension were taken, and note was taken of the compendial result achieved.

In the procedure which I propose to substitute for purposes of exploring the triple action just referred to (I propose to call it the *Cæsuri*c Procedure) we—*this constitutes our first operation*—mix equal volumes of (a) serum and microbic suspension, or (b) serum and washed corpuscles, or (c) microbic suspension and washed corpuscles. Let us call these pre-phagocytic mixtures.

We then, after incubating the above pre-phagocytic mixtures for fifteen minutes, pass to our *second operation*. If we are dealing with a "*sero-opsonic*" pre-phagocytic mixture, we add to 2 volumes of it, 1 volume of washed corpuscles. If we are dealing with a "*sero-stimulin*" pre-phagocytic mixture we add to 2 volumes of it, 1 volume of microbes. And if we are dealing with a *microbi-leucocytic pre-phagocytic mixture* we add to 2 volumes of it 1 volume of serum. We then reincubate for fifteen minutes, thus bringing those chemical agents which have been operating in our pre-phagocytic mixtures a *second time* into action in completed phagocytic mixtures.

Tables V and IX show that the *cæsuri*c procedure furnishes valuable information.

Obviously the method would be more valuable if the adventitious chemical action which comes into operation in the secondary incubation could be eliminated.

There are two methods by which this might be done:

The *first* would be to centrifuge the phagocytic mixture, as soon as the third of the triad of operative factors have been added. By thus avoiding second incubation we should cut out most, if not all, the adventitious chemical action.

The *second device*—which could be combined with the first—would be to employ when testing a serum for stimulins, a bacterial pabulum of pre-opsonized microbes.

My experience is that we gain little, if indeed anything, by these devices; and for two reasons, *first* because allowing as we do in the first case, less time for opsonization and stimulin action, we greatly reduce our phagocytic count.

And, if our second device gives us no better results, it is because we, by furnishing the leucocytes with a bacterial pabulum of pre-opsonized microbes, obtain an all-round increase in phagocytosis.

And that, like the all-round decrease of phagocytosis obtained by the first device, largely obliterates phagocytic differences.

PART II

THE EVIDENCE AGAINST THE PRESENT UNIVERSALLY ACCEPTED DOCTRINES OF PHAGOCYTOSIS AND IMMUNIZATION

Introductory

Douglas and I showed in our original paper on the opsonic power of the serum that the phagocytosis which occurred when washed corpuscles, microbes and serum were brought together was due to the serum acting upon the microbes in such a way as to prepare them for ingestion.

agglutinated when either a heated serum, a reconstituted serum,¹ or a heated leucocytic diffusate² is employed in a phagocytic mixture in lieu of an ordinary unheated serum.

The agglutination becomes more marked when Tuberculin B.E. is brought into application upon the microbes along with a heated serum.

These facts touching agglutination would appear to have, in addition to their bearing on phagocytic testing, a general importance; for they obviously suggest that the leucocytes are one, and for all we know the only, source of the agglutinins which appear in the blood of those who are making epiphyllactic response to vaccines, or, as the case may be, auto-inoculations.

Agglutination of microbes shows itself in a phagocytic film in the unduly small percentage of the leucocytes which have ingested microbes, and in the fact that there are often among the leucocytes which have phagocytosed many which have gorged themselves with more microbes than it is possible to count. The blank leucocytes are, of course, those whose environment has been swept clean of microbes by the agglutinating action of the serum; and the phagocytes whose protoplasm is completely full of microbes are, of course, those which have chanced upon a mass of agglutinated microbes.

And when we have this condition of things—in other words, when we have only an unduly small percentage of phagocytosing leucocytes with a considerable proportion of leucocytes brim-full of microbes—it is good practice to discard the whole count as one vitiated by bacterial agglutination.

But if we hesitate to condemn an entire film on the ground of a few leucocytes which have glutinized, we should either leave out of our count these particular leucocytes, or else we should by way of a compromise (and I have in this paper adopted this compromise) accredit them with having ingested each 20, or some arbitrarily chosen, large number of microbes.

Table I gives the results of differential phagocytic counts of films made from phagocytic mixtures containing my unheated non-agglutinating, and my heated and agglutinating, and my reconstituted and agglutinating serum; and the corresponding sera of a colleague.

TABLE I.—DETAILS OF THE STAPHYLOCOCCIC INTAKE OF A.E.W.'s AND K.B.R.'s LEUCOCYTES IN PHAGOCYTIC MIXTURES MADE RESPECTIVELY WITH THEIR UNHEATED, HEATED AND RECONSTITUTED SERUM.

| | A.E.W.'s leucocytes | | | K.B.R.'s leucocytes | | |
|----------------------------------|-------------------------------------------------------------------------------|----------------------------------------------------------------------------------|---------------------------|-------------------------------------------------------------------------------|----------------------------------------------------------------------------------|---------------------------|
| | Percentage of polynuclears which have ingested a countable number of microbes | Percentage of polynuclears which have ingested an uncountable number of microbes | Percentage of blank cells | Percentage of polynuclears which have ingested a countable number of microbes | Percentage of polynuclears which have ingested an uncountable number of microbes | Percentage of blank cells |
| <i>Experiment 1</i> | | | | | | |
| Unheated serum | 94 | 6 | 0 | 96 | 0 | 4 |
| Heated serum | 78 | 2 | 20 | 78 | 4 | 18 |
| Reconstituted serum ¹ | 60 | 11 | 29 | 76 | 18 | 6 |
| <i>Experiment 2</i> | | | | | | |
| Unheated serum | 97 | 3 | 0 | 84 | 0 | 16 |
| Heated serum | 53 | 11 | 36 | 36 | 0 | 64 |
| Reconstituted serum | 37 | 14 | 49 | 57 | 24 | 19 |

NOTE.—I have elsewhere reported (*Lancet*, 1931 (ii), 277, Table XIX without being able to account for it, the almost incredibly large phagocytic ingestions which are obtained in phagocytic mixtures composed of washed corpuscles, staphylococcus suspensions and a heated serum to which effective quanta of Tuberculin B.E. have been added. I cited for instance (*loc. cit.* Table XIX) an experiment in which with different

¹ By a *reconstituted serum* I mean a solution of euglobulin obtained by precipitating normal human serum with 10 volumes of water saturated with CO₂, collecting the precipitate by centrifugalization and re-dissolving it in a volume of normal salt solution which corresponds to the volume of the original serum.

² A *leucocytic diffusate* is an extract of leucocytes such as is obtained by digesting washed corpuscles at blood temperature in normal salt solution.

THERMOLABILITY OF OPSONINS

Only about a year after Douglas and I had shown that opsonins of normal serum are largely thermolabile, Neufeld and Rimpau¹ announced that the heated serum of "immune animals" contained phago-incitor substances, which rendered the pneumococci and streptococci with which he had inoculated his animals, phagocytatable or, as the case may be, more phagocytatable.

There were in the ratiocinative operations of these authors two main false assumptions. The first was the assumption that animals which have been heavily and repeatedly inoculated may without further question be called "*immune*"; and that their sera may properly be described as "*immune*"; or "*anti-sera*".

I pointed out thirty-five years ago² that these so-called "*immune sera*" might in lieu of, or along with, epiphyllactic substances produced in the organism, contain antigenic substances remaining over in the blood from the original inoculations or auto-inoculations.

I have nothing to alter in that except that where I, thirty-five years ago, wrote antigens I should now probably write "*leucocytic stimulins*". And whichever of these descriptive terms I applied to such bacterial substances as might still be circulating in the blood, it would be clear that these would, when transferred to another animal organism, confer on it instead of passive, *active* immunization.

A conspicuous, but now forgotten, practical exemplification of active immunization misinterpreted as a passive one was furnished by the beneficial results which Chantemesse (this was twenty-five or thirty years ago) claimed in typhoid fever from the inoculation of an "*antityphoid serum*". Of this so-called anti-serum he administered in heavily infected cases of typhoid fever reduced, and in still more heavily infected cases absolutely minimal doses—doses of not more than a fraction of a cubic centimetre. (I was unable to procure from him a sample of the serum he was using in order to see whether it contained antigens or antibodies.)

These considerations apply just as much to the sera of Neufeld and Rimpau's laboratory animals as to the sera of horses employed for the treatment of human patients.

A second fallacious assumption made by Neufeld and Rimpau was that convincing proof of a phago-incitor serum operating as an opsonin is furnished when a serum ceases, after it has been digested with heavy doses of the antigenic microbes, to exert its previous phago-incitor effect.

It has to be kept in view in connexion with all such *absorption experiments* that when a serum is digested with a bacterial culture, and the microbes are afterwards removed by centrifugalization, there may remain in the serum such a charge of bacterial toxins as would, even with opsonins present, paralyse phagocytosis.

In view of that consideration "*absorption tests*" can never furnish convincing proof that a serum contained, before it was operated upon, opsonic substances.

A year after the publication of Neufeld and Rimpau's work Reid and I,³ following up the work of these observers, found phago-incitor substances in the heated serum of auto-inoculating tubercular patients. These "*phago-incitor substances*" I—avoiding the term "*bacteriotropins*"⁴ which Neufeld and Rimpau had applied to them—called "*thermostable opsonins*".

We were, in calling the phago-incitor substances we were dealing with "*thermostable opsonins*", beguiled by the self-same fallacies which had misled Neufeld and Rimpau.

¹ Neufeld and Rimpau, *Deutsche med. Wchschr.*, Sept. 1904.

² A Criticism of the Foundations of Serum Therapy, *Clin. Journal*, May 16, 1906. (Reprinted in the Author's "*Studies in Immunisation*", Constable, 1909.)

³ Wright and Reid, *Proc. Roy. Soc. B.*, 1906, 77 (Also reprinted in the Author's "*Studies in Immunisation*", Constable, 1909.)

⁴ I eschewed the term "*bacteriotropins*" because I had already five years before (*Lancet*, Dec. 23, 1899, 1st paragraph) employed that term in its proper generic significance, i.e. as a name for *every and any substance which enters into chemical combination with microbes*. And the use to which Neufeld and Rimpau put this term is also, quite apart from the question of priority of usage, a perfectly improper use. This will be seen when we regard the fact that the limitation of the term *bacteriotropins* to one special variety of "*bacterio-tropic*" substances is a wrong in principle as would be the limitation of a zoological class—let us say the class "*Mammalia*"—to one particular species of mammals.

After establishing the fact that the blood fluids converted the microbes into an attractive pabulum for the leucocytes (whence the word "opsonin"), and further showing that this property is abolished, or all but abolished, by heating the serum to 60° C., we went on to test by what we called the "chiastic procedure" the serum and washed corpuscles of two patients, whom we had successfully treated with staphylococcus vaccine, against the serum and corpuscles of normal men.¹

In the chiastic procedure here in question four separate phagocytic mixtures are made all containing a volume of one and the same staphylococcus suspension. To the volume of staphylococcus suspension employed in capillary Tube 1 are added equal volumes of Normal Serum, and of Normal Washed Corpuscles; to that employed in Tube 2, equal volumes of Normal Serum and of the Patient's Washed Corpuscles; to that in Tube 3, equal volumes of the Patient's Serum and of Normal Washed Corpuscles; and to the staphylococcus suspension employed in Tube 4, equal volumes of the Patient's Serum and of his Washed Corpuscles.

It emerged in experiments done in this way on the bloods of our two patients that there was no improvement in their corpuscles but a distinct improvement in their sera. And we drew from these and from similar experiments in which the serum and corpuscles of tubercular patients were compared with those of normal men, the conclusion that epiphyllactic responses such as those which are induced by therapeutic inoculations that do not cause constitutional disturbances, and by the auto-inoculations of apyrexia patients, increase the opsonic power of the serum, and leave the leucocytes unchanged.

It ought, of course, to have suggested itself to us that what we had found in patients examined ten days after inoculation might not hold true of such patients examined earlier; and further that what we had found to hold true of apyrexia patients would not necessarily hold true of those suffering from pyrexia infections.

Also it ought to have suggested itself to us as possible that the blood of our inoculated or auto-inoculating patients might, or at any rate might in certain cases, contain in addition to bacteriotropic substances (opsonins) leucocytotropic substances which would, acting as stimulins or depressants, raise or lower the efficiency of the phagocytes.

Again, it never even occurred to us that the epiphyllactic response of the blood which follows upon the inoculation of vaccines and upon auto-inoculations, could affect any but the particular variety of microbe which was evoking the response.

And finally, it never dawned upon us that the microbes which were employed to serve as a bacterial pabulum and to furnish a measure of phagocytic power, could play any active part in the phagocytic reaction.

All these a priori inferences were mistaken, and we were, as has only recently emerged, just as much out of our depth with regard to what was happening in our phagocytic mixtures, as to what was happening in our patients.

I do not make light of these errors. "Errors", Goethe tells us, "always matter: but how very much they matter becomes clear to a man only at the end of his way." But I realize that if I were put back into the blinkers which confined my outlook and that of my contemporaries forty years ago, I—do not want to shelter myself by saying we—should almost certainly fall again into the same pitfalls.

It had, at that time, been recently demonstrated by Ehrlich that epiphyllactic response was *antithetic chemical response*. And it seemed to follow from this that inoculations of bacterial vaccines (and I would add the engendering of auto-inoculations) would produce only bacteriotropic as distinguished from leucocytotropic substances. And again it seemed to follow by analogy from the fact that the antitoxins produced by toxins are strictly specific that the same would hold also of bacterial inoculations.

Again, forty years ago, it had not occurred to anyone that epiphyllactic response could be evoked *in vitro*. And much less had it suggested itself that epiphyllactic response *in vitro* would come under regular observation in all phagocytic, and (as is evidenced in the 'Caput and Cauda phenomenon')² in all leuco-bactericidal tests.

And to-day I often ask myself how much water will flow under the bridges before that fundamental fact that epiphyllactic response occurs in the blood *in vitro* succeeds in getting itself into bacteriological textbooks.

Let me now proceed to deal in detail with some of the more important points which have been incidentally referred to.

¹ *Proc. Roy. Soc.*, 1903, 72; *ibid.*, 1904, 73.

² *vide* Wright, New Principles in Therapeutic Inoculation, *Lancet*, Feb. 24, 1923, and also *infra* p. 178⁴.

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I pointed out thirty-five years ago² that these so-called "immune sera" might in lieu of, or along with, epiphyllactic substances produced in the organism, contain antigenic substances remaining over in the blood from the original inoculations or auto-inoculations.

I have nothing to alter in that except that where I, thirty-five years ago, wrote antigens I should now probably write "leucocytic stimulins". And whichever of these descriptive terms I applied to such bacterial substances as might still be circulating in the blood, it would be clear that these would, when transferred to another animal organism, confer on it instead of passive, active immunization.

A conspicuous, but now forgotten, practical exemplification of active immunization misinterpreted as a passive one was furnished by the beneficial results which Chantemesse (this was twenty-five or thirty years ago) claimed in typhoid fever from the inoculation of an "antityphoid serum". Of this so-called anti-serum he administered in heavily infected cases of typhoid fever reduced, and in still more heavily infected cases absolutely minimal doses—doses of not more than a fraction of a cubic centimetre. (I was unable to procure from him a sample of the serum he was using in order to see whether it contained antigens or antibodies.)

These considerations apply just as much to the sera of Neufeld and Rimpau's laboratory animals as to the sera of horses employed for the treatment of human patients.

A second fallacious assumption made by Neufeld and Rimpau was that convincing proof of a phago-incitor serum operating as an opsonin is furnished when a serum ceases, after it has been digested with heavy doses of the antigenic microbes, to exert its previous phago-incitor effect.

It has to be kept in view in connexion with all such absorption experiments that when a serum is digested with a bacterial culture, and the microbes are afterwards removed by centrifugalization, there may remain in the serum such a charge of bacterial toxins as would, even with opsonins present, paralyse phagocytosis.

In view of that consideration "absorption tests" can never furnish convincing proof that a serum contained, before it was operated upon, opsonic substances.

A year after the publication of Neufeld and Rimpau's work Reid and I,³ following up the work of these observers, found phago-incitor substances in the heated serum of auto-inoculating tubercular patients. These "phago-incitor substances" I—avoiding the term "bacteriotropins"⁴ which Neufeld and Rimpau had applied to them—called "thermostable opsonins".

We were, in calling the phago-incitor substances we were dealing with "thermostable opsonins", beguiled by the self-same fallacies which had misled Neufeld and Rimpau.

¹ Neufeld and Rimpau, *Deutsche med. W'chenschr.*, Sept. 1904.

² A Criticism of the Foundations of Serum Therapy, *Clin. Journal*, May 16, 1906. (Reprinted in the Author's "Studies in Immunisation", Constable, 1909.)

³ Wright and Reid, *Proc. Roy. Soc. B.*, 1906, 77 (Also reprinted in the Author's "Studies in Immunisation", Constable, 1909.)

⁴ I eschewed the term "bacteriotropins" because I had already five years before (*Lancet*, Dec. 23, 1899, 1st paragraph) employed that term in its proper generic signification, i.e. as a name for every and any substance which enters into chemical combination with microbes. And the use to which Neufeld and Rimpau put this term is also, quite apart from the question of priority of usage, a perfectly improper use. This will be seen when we regard the fact that the limitation of the term *bacteriotropins* to one special variety of "bacterio-tropic" substances is a wrong in principle as would be the limitation of a zoological class—let us say the class "Mammalian"—to one particular species of mammals.

In other words we had no better warranty for concluding that the heated sera of our auto-inoculating tuberculous patients contained opsonins, than the fact that they induced phagocytosis; and the further fact that the phagocytic power of these sera was lost, when they were digested with heavy doses of tubercle bacilli.

But when I look back on the fact that many of our tuberculous patients were in a very unwholesome condition I cannot help thinking that their blood may not infrequently have contained instead of, or along with, opsonins, tuberculin derived from the tubercle bacilli in their system. And such tuberculin would, as will be shown presently, operate as a phagocytic stimulin.

Further points—and these are new points—which invalidate Neufeld and Rimpau's argument that they had in hand a thermo-stable opsonic serum are referred to below.

I have in view the point that a serum may (as is shown in Table V) be converted into a leucocyte-stimulating serum by heating; and also the point that heating may (as has been shown in Table I) convert sera which do not agglutinate staphylococci into sera which do, with the result that both the percentage of phagocytes and the number of microbes phagocyted may be greatly increased—this being explained by the fact that leucocytes are not attracted by chemotaxis to individual staphylococci to anything like the same degree as they are to agglomerated staphylococci.

The next important happening in the field of phagocytic research was that Shattock and Dudgeon¹—employing the same chiasitic method that Douglas and I had done, but conducting their experiments with a "phagocytic pabulum" consisting of melanin particles—found that the leucocytes of patients suffering from febrile infections differ from those of normal men in that they have usually a *supra*-—but occasionally an *infra*-normal phagocytic efficiency.

And Shattock and Dudgeon pointed out that no one could, in view of their having worked with a phagocytic pabulum of melanin particles, come to any other conclusion than that the increased leucocytic response obtained by auto-inoculations was non-specific.

We shall return to this question of non-specific response to bacterial infections later. In the meantime let us note that the results obtained by Shattock and Dudgeon are irreconcilable with Ehrlich's theory that immunizing response consists *exclusively* in the production of antithetic chemical substances.

For a long time after the date of Shattock and Dudgeon's publication—an interval during which I was working in succession on the study of therapeutic inoculation applied to tuberculosis, prophylactic inoculation against pneumonia, and the treatment of war wounds—I was turning over in my mind not only Shattock and Dudgeon's findings, but also the many new facts about epiphyllactic response which my work was bringing to light. And the conviction was steadily growing in my mind that much of the currently accepted doctrine of immunization was erroneous.

Finally, after much tentative work, I obtained convincing proof of non-specific (i.e. *homo*- and *hetero*-) bactericidal response to the implantation of vaccines into the blood *in vitro*.

It was shown in the papers cited below² that separate implantations of staphylococcus and streptococcus vaccines into the blood *in vitro* resulted in a notable increase in the bactericidal power exerted by serum upon both these microbes.

And let me here remind the reader, though the recent advances in chemotherapy may have deprived this of much of its interest, that in both those papers I drew attention to the fact that the power of inducing epiphyllactic response in extravascular blood had put into our hands a method of transfusion—I called it *immuno-transfusion* from which good results were to be expected in the treatment of septicæmia.

Also I pointed out in this connexion that the blood which was used for immuno-transfusion might be either normal blood subjected to vaccine treatment *in vitro*; or, if this was preferred, blood obtained from a donor who had been inoculated a few hours before

¹ Shattock and Dudgeon, *Proc. Roy. Soc. B.*, 1908, 80.

² Wright, Sur la production de substances bactericides non-specifiques au moyen des vaccine *in vivo* et *in vitro*. *Compt. rend. Acad. d. sc.*, t. 167, 600, Oct. 21, 1918.

Wright, A Lecture on the Lessons of the War, delivered at the Royal Society of Medicine on Feb. 25, 1919, *Lancet*, March 29, 1919.

with a staphylococcus or streptococcus vaccine!—the optimum dosage of the vaccine used having in each case been ascertained by what I call “a vaccine response test”. About the same time I obtained clear evidence of leucocytes killing microbes extracellularly.

EXPERIMENTAL EVIDENCE SHOWING THAT LEUCOCYTES KILL MICROBES EXTRACELLULARLY

The procedure by which it was first established that leucocytes can kill extracellularly was described and figured in my paper “On the Lessons of the War” referred to above.

The procedure consists in taking leucocytes which have emigrated on to glass laths from blood centrifuged before it clots and disposing these laths—one directly as it comes out of the serum, and the other after the serum has been washed off—upon an agar plate inseminated with staphylococcus or streptococcus. When the implanted plates are then incubated the staphylococcus grows out all over the surface of the agar, except under these central strips of each lath where we have a thick belt of emigrated leucocytes. There the agar surface, both in the case where the leucocytes are operating in serum and in the case where they are operating in salt solution, remains absolutely bare.

When the laths are now stained and microscopically examined, it is found that in the case where the leucocytes were operating in serum every leucocyte is full of staphylococci, and these have (we may assume) been inhibited in their growth or killed intracellularly.

On the other hand, in the specimen where the leucocytes were operating in salt solution the leucocytes are empty of microbes, and here the staphylococci have manifestly been inhibited in their growth or killed extracellularly.

I have confirmed the fact that the leucocytes exert their bactericidal power extra- as well as intra-cellularly by five other experimental methods.

I shall label them Procedures 2, 3, 4, 5, and 6.

Procedure 2.—Staphylococci in moderate numbers are implanted in blood drawn off from the finger. (Moderate numbers would in this case mean some thousands or tens of thousands of staphylococci per cubic centimetre.) This blood is immediately centrifuged in flat “emigration tubes”² and is thus divided into an upper portion consisting of plasma, which is presently converted into a *white clot*, and a lower portion consisting of red blood corpuscles surmounted by a cap of leucocytes—this portion being presently converted into a *red clot*. The centrifuged blood is now, after the emigration tube has been very tenderly capped with plasticine, placed upright in a plasticine slide in the incubator; and incubation is continued for twenty-four hours.

When the clot is now microscopically examined, it is found that many of the implanted staphylococci (but not by any means all of them, for a large proportion are killed instantaneously in the blood) have grown out into colonies in the white clot.

But it is the distribution of the colonies, as distinct from the bactericidal power of the blood, which is of special interest. The colonies are found to have grown out in considerable numbers in the distal portion of the white clot; but that none (this will of course depend upon our having hit off the proper measure of *in vitro* infection) will have grown out in the leucocyte invaded proximal area of the white clot or—this is the relevant point—in the area which lies immediately distal to that into which the leucocytes have penetrated. In that “no man’s land” there are, as microscopic examination shows, neither leucocytes nor microbes. The microbes which should by rights have grown out there must have been killed by bactericidal substances excreted by the leucocytes.

Procedure 3.—Here we make “extra-” and “intra-coagular implantations” of staphylococcus into blood in capillary tubes. An *intra-coagular implantation* is made by inoculating staphylococcus into blood drawn from the finger, aspirating this inoculated blood into capillary tubes and allowing it to clot undisturbed.

¹ Immuno-transfusion in this latter form has, as I gather from the French medical literature of the twenty-two years 1918-1940, given very striking results. In England the method has as good as never come into application. And I myself have had only one good opportunity of administering to a patient blood inoculated *in vitro*. This was at the end of the last war, when I was dealing with a severe case of septicemia following a wound infection. An account of the very strikingly beneficial result achieved in that case was included in the second of the two papers referred to above.

² The method of making these is described in the “Technique of the Teat and Capillary Pipette”, Wright and Colebrook, Constable, London, 1921.

In an *extra-coagular implantation* a similar volume of *unimplanted* blood is drawn up into a capillary tube, and we then, after the clot has contracted and the serum has exuded, implant into this serum the same volume of *staphylococcus* suspension as was employed for the *intra-coagular implantation*—let us say 5 c.mm. of a 10,000-fold dilution of a twenty-four-hour broth culture. We make the implantation with the aid of a capillary pipette drawn out into a hair-line extremity.

Along with the two foregoing implantations a similar quantum of *staphylococcus* suspension is implanted into another capillary tube which contains only serum. These tubes—let me for convenience number them 1, 2, and 3—are, after sealing up, incubated horizontally (horizontally to prevent the colonies running together) upon a plasticine slide.

After twenty-four hours' incubation the *staphylococci* colonies in Tube 3, which contains only serum, are seen to be many times more numerous than those in the *extra-coagular* serum in Tube 2. These are so few that they can quite easily be counted by the naked eye.

Dealing now with Tube 1, the colonies which have grown out in the interior of the clot are enumerated as follows: The clot is blown out into a test tube of water. This is then placed in a water bath standing at 60° C., and the clot is left there until it is fully decolorized. After that it is transferred to a slide; is fixed down upon this by drying, and is then lightly stained with methylene blue. This done the colonies are counted under the low power of the microscope.

These, instead of being, as one might have expected, definitely fewer than those in the *ex-coagular* serum, are generally a little more numerous.

With regard to the interpretation of the findings, those in Tube 2 point to stimulus deriving from the microbes implanted into the serum operating upon the leucocytes of the clot, and causing these to pour out bactericidal elements. These will now diffuse into the serum, rendering this bactericidal. And the fact that the colonies which grow out in the clot are, despite the presumptively better opportunity for phagocytosis there afforded, fewer than those which have grown out in the serum in Tube 2, is no doubt due to the bactericidal substances which have been excreted by the leucocytes coming less effectively into operation upon the microbes which are lodged in the clot than on the microbes which are floating free in the serum.

Procedure 4.—We draw up some melted agar into the stem of a capillary tube and then immediately blow it out. We leave by this procedure a coating of agar upon the wall of our capillary tube. We then draw up into this a volume of blood from the finger and set this aside to clot and contract. By this contraction the agar lining is pulled off from the walls of the tube and the serum now exudes through the agar capsule into the *extra-coagular* space. Into this space we introduce by the same procedure which was employed for *extra-coagular implantations*, a small measured quantum of *staphylococcus* suspension.

At the same time we—to provide ourselves with a control—make a similar implantation of *staphylococci* into two samples of serum and draw these up the one into a naked and the other into an agar-lined tube.

After twenty-four hours' incubation we find that the colonies which have grown out in the control tubes containing only serum are much more numerous than those which have grown in the serum in the tube containing the agar-enveloped clot. And we must, as in Procedure 3, assume that the leucocytes of the clot have (under the impulsion of bacterial stimulus which have traversed the agar capsule) secreted bactericidal substances which have killed the microbes which are implanted into the serum outside.

Procedure 5.—Here we prepare one or more *leucocyte-lined capillary tubes*. This is done by drawing up into our capillary tube first a very small quantum of normal salt solution and then about sufficient blood to occupy about one-half of the stem, and after this another very small quantum of normal salt solution. The purpose of flanking the blood above and below with salt solution is to prevent the clot drying and fixing itself on to the walls of the tube.

After filling in our pipette as explained we seal up its distal end, mark off with a blue pencil the limits of our blood-clot, and then incubate. After some twenty to thirty minutes we blow out the clot and run enough normal salt solution through the tube to carry away loose leucocytes and red blood corpuscles. We then aspirate into our tube some serum lightly implanted with *staphylococcus*, disposing this so that the serum

shall fill up the space between our two blue pencil marks, and extend on each side a little distance beyond them. We then take exactly the same amount of our staphylococcus implanted serum and draw this up into an unlined capillary tube. This done we seal up the distal ends of our pipettes in the flame, and provide against evaporation from the proximal ends by blocking the neck of our capillary pipette either with a little mercury, or with a little salt solution. We then place our pipettes in the incubator, fixing them down horizontally upon a plasticine slide.

Comparison of the tubes twenty-four hours after shows that a great number of staphylococci have been killed in the leucocyte-lined tube, and it will be noticed that the bactericidal effect exerted by the leucocytes extends some little distance on each side beyond the blue pencil marks.

The experiment can be repeated with even more striking results with capillary tubes which have been furnished with a double lining of leucocytes—such a double lining being obtained by refilling the tube after Clot No. 1 has been blown out with a new lot of blood and then carrying out the rest of the technique exactly as in the single leucocyte-lined tube.

Procedure 6.—We here first prepare some defibrinated blood and then add to 100 c.mm. of this blood one-tenth of its volume of approximately a 30,000-fold dilution of a twenty-four-hour broth culture of staphylococcus. 10 c.mm. of such a 30,000-fold dilution will represent an implantation of about 100 staphylococci per cubic centimetre of blood.

We now take three to six capillary tubes; draw up into each 100 c.mm. of the implanted blood; amputate the barrels of the pipettes; and then centrifuge until the corpuscles have been carried down completely. That done, and there will now be just as many microbes in the upper as in the lower layers of the serum, we block the proximal ends of the tubes with plasticine and then incubate them upright in plasticine slides for one and a half to two hours.

We now take a series of evacuating pipettes (one for each tube) and draw out the distal end of each stem into a throttle—taking care to make this sufficiently wide. This done we mark off in each tube the middle point of the contained serum with a blue pencil, and then, working very cautiously, draw up into our evacuating pipette, first the upper, and then, after interposing an air-bubble, the lower half of the serum.

This done, we seal up the stems of our evacuating pipettes above and below, and arrange them horizontally upon a plasticine slide; and now incubate for twenty-four hours. After that we count the colonies which have developed in each portion of the serum (Table II).

TABLE II.

| | Number of staphylococcus colonies which developed in centrifuged serum taken from | |
|---------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-------------------------------|
| | the distal half of the tube | the proximal half of the tube |
| Defibrinated blood heavily implanted with staphylococci | Uncountable | 60 |
| Defibrinated blood implanted with half as many staphylococci | { 100 | { 0 |
| | { 100 | { 11 |
| Defibrinated blood implanted with roughly a quarter as many staphylococci | { 41 | { 8 |
| | { 23 | { 10 |

The point that leucocytes can be killed quite as surely extracellularly as intracellularly settled, let me now take up the question as to whether the doctrine of Shattock and Dudgeon that the leucocytes of pyrexia, i.e. auto-inoculating patients have generally acquired a leucocytic efficiency superior to that of the leucocytes of the normal blood, should prevail over the doctrine which was enumerated by Douglas and myself. I mean the doctrine that the leucocytes are unaffected by inoculation.

QUESTION AS TO WHETHER THE LEUCOCYTES ARE OR ARE NOT AFFECTED BY INOCULATION

With a view to clearing up this point I, employing as before, the chiasitic procedure, compared my own defibrinated blood untreated with my defibrinated blood inoculated *in vitro* with doses of $\frac{1}{1000000}$ to $\frac{1}{2000000}$ of its volume of a Tuberculin B.E.¹

The inoculated and the control bloods were then incubated side by side for longer or

¹ The Tuberculin B.E. which was employed here and in all the other experiments reported in this paper contained 1 mg. of dried and ground up tubercle bacilli per cubic centimetre of fluid.

shorter periods. After that they were centrifuged; the serum pipetted off, and the corpuscles washed in normal salt solution. Chiasitic phagocytic mixtures were then made of the washed corpuscles and the sera in the manner particularized in the table below.

TABLE III.—SHOWING THE EFFECT OF CHIASITIC EXPERIMENTS IN WHICH THE WASHED LEUCOCYTES AND SERUM OF NORMAL DEFIBRINATED BLOOD WERE COMPARED WITH THE WASHED LEUCOCYTES AND SERUM OF THE SAME DEFIBRINATED BLOOD TREATED WITH TUBERCULIN B.E.

The test-microbes employed were staphylococci, and the interpretation of the Initials employed is given at the foot of the table.

| Amount of Tuberculin B.E. | Period for which the inoculated and control bloods were incubated | Constitution of the phagocytic mixtures | | Phagocytic count | Summary of results |
|---------------------------------------------------------------------------|-------------------------------------------------------------------|-----------------------------------------|--------|------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1 cc. of a suspension containing 1 mg. of dried tubercle powder in 1 c.c. | 25 minutes | NS + NC + | Staph. | 3.29 | Phagocytic efficiency of my blood increased as 1:2.4; that of my leucocytes as 1:2; and the phago-incitor power of my serum as 1:1.2 |
| | | NS + IC + | Staph. | 6.70 | |
| | | IS + NC + | Staph. | 3.96 | |
| | | IS + IC + | Staph. | 8.01 | |
| Ditto | 20 minutes | NS + NC + | Staph. | 1.09 | Phagocytic efficiency of my blood increased as 1:2; that of my leucocytes as 1:2; the phago-incitor power of my serum remaining as 1:1 |
| | | NS + IC + | Staph. | 2.16 | |
| | | IS + NC + | Staph. | 1.04 | |
| | | IS + IC + | Staph. | 2.06 | |
| Ditto | Not noted (Probably between 20 and 40 minutes) | NS + NC + | Staph. | 3.24 | Phagocytic efficiency of my blood increased as 1:1.7; that of my leucocytes as 1:1.4; the phago-incitor power of my serum remaining as 1:1, nearly |
| | | NS + IC + | Staph. | 6.72 | |
| | | IS + NC + | Staph. | 5.20 | |
| | | IS + IC + | Staph. | 5.60 | |
| Ditto | Not noted (Probably between 20 and 40 minutes) | NS + NC + | Staph. | 1.34 | Phagocytic efficiency of my blood increased as 1:2.1; and that of my leucocytes as 1:1.2; and the phago-incitor power of my serum increased as 1:1.7 |
| | | NS + IC + | Staph. | 1.70 | |
| | | IS + NC + | Staph. | 2.38 | |
| | | IS + IC + | Staph. | 2.80 | |
| Ditto | 40 minutes | NS + NC + | Staph. | 7.1 | Phagocytic efficiency of my blood increased as 1:1.1, and that of my leucocytes as 1:1.3; and the phago-incitor power of my serum reduced as 1:0.9, nearly |
| | | NS + IC + | Staph. | 9.7 | |
| | | IS + NC + | Staph. | 6.4 | |
| | | IS + IC + | Staph. | 8.1 | |
| 1 mg. | 40 minutes | NS + NC + | Staph. | 3.9 | Phagocytic efficiency of my blood increased as 1:1.5; that of my leucocytes remains as 1:1, nearly; and the phago-incitor power of my serum is increased as 1:1.4 |
| | | NS + IC + | Staph. | 5.1 | |
| | | IS + NC + | Staph. | 6.4 | |
| | | IS + IC + | Staph. | 6.0 | |

NS=Normal Serum; NC Normal Corpuscles; IS=Immune Serum; IC Immune Corpuscles.

Epitome of Results.—Phagocytic efficiency of the whole blood improved on the average as 1:1.7; Phagocytic efficiency of the leucocytes improved on the average as 1:1.5; Phago-incitor power of the serum improved on the average as 1:1.2; and in two of the experiments as 1:1.7 and 1:1.4 respectively.

These results show that Douglas and I were wrong in concluding that the leucocytes are not affected by bacterial vaccines, and that Shattock and Dudgeon were, on the whole, right in concluding that they were.

Further, the results set forth in the above table show—and this is in agreement with

the inferences of Shattock and Dudgeon—that the increase of phagocytic efficiency which is obtained by bacterial vaccines is non-specific.

More direct confirmation of Shattock and Dudgeon's inferences has been obtained by my sometime pupil—Dr. Ronald Hare of Toronto—by the phagocytic testing of the leucocytes with different kinds and strains of microbes. These unpublished results, which have been very generously placed at my disposal, are set out in the table below.

TABLE IV.—SHOWING THE NON-SPECIFIC RESPONSE OF LEUCOCYTES TO SEPTICÆMIC INFECTIONS.

Here the Washed Corpuscles of Infected Patients and of Normal Controls were incubated with 1 volume of normal serum and 1 volume of a suspension of 2, 3, or 4 different strains or species of microbes.

The results of the phagocytic counts so made are shown in the table below in the form of ratios—the phagocytic intake of the normal man being in each case represented by 1.

| Name of patient: | Strain 1 | Streptococcus Strain 2 | Strain 3 | Staphylococcus |
|-----------------------------------------------------|----------|---------------------------|----------|----------------|
| Boon (mild streptococcus infection) | 1·8 | 1·8 | | 1·7 |
| Ellis (fatal streptococcal septicæmia) | 0·73 | 0·76 | | 0·8 |
| Same patient (a week later) ... | 0·2 | 0·3 | 0·17 | 0·1 |
| Hatcher (Whitlow) ... | 0·49 | 0·51 | | 0·47 |
| Barker (streptococcal infection) ... | 1·3 | | | 0·8 |
| Allan (acute fatal streptococcus septicæmia) ... | 1·9 | 2·5 | | 2·3 |

THE EFFECT EXERTED BY HEATING THE SERUM FOR TEN MINUTES TO 60° C.

The problem of the nature of the thermostable phago-incitor substances found by Neufeld and Rimpau in the heated serum of their "immune animals" and by Reid and myself in the heated sera of tuberculous patients, calling for further investigation, my choice of methods lay between testing the heated sera by the "Absorptional" and by the "Cæsuriæ Method". And inasmuch as the former method is, as has been indicated above, very fallacious when employed as a prelude to phagocytic tests, I naturally employed the latter.

I took a series of samples of my serum and tested these unheated and heated, both by the "sero-microbic", and by the "sero-corpuscular cæsuriæ procedure". Table V shows the results yielded by these two different methods.

TABLE V.—SECTION 1 SETS OUT THE RESULTS OF PHAGOCYTIC TESTS OBTAINED BY MAKING, TO BEGIN WITH, "SERO-MICROBIC, PRE-PHAGOCYTIC", AND SECTION 2, THE RESULTS BY MAKING, TO BEGIN WITH, "SERO-CORPUSCULAR, PRE-PHAGOCYTIC MIXTURES"; AND THEN AFTERWARDS COMPLETING THESE MIXTURES.

Phagocytic counts obtained in consecutive experiments
Serial No. of the Serum Test:

| SECTION 1 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | Average |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|------|-----|-----|------|------|------|------|------|---------|
| <i>Sero-microbic Cæsuriæ method</i> | | | | | | | | | | |
| Here the UNHEATED SERUM was incubated first for 15' with a suspension of staphylococci; and then, after leucocytes had been added, the completed phagocytic mixtures were incubated for another 15' | 1·75 | 3·9 | 6·0 | 3·5 | 6·6 | 1·83 | 2·1 | 1·7 | 6·7 | 3·8 |
| Here the same procedure was repeated with HEATED SERUM | 0·12 | 1·15 | 1·6 | 1·5 | 0·85 | 0·42 | 0·38 | 1·3 | 3·2 | 1·3 |
| <i>SECTION 2.</i> | | | | | | | | | | |
| <i>Sero-corpuscular Cæsuriæ method</i> | | | | | | | | | | |
| Here the UNHEATED SERUM was incubated first for 15' with a suspension of washed corpuscles; and then after staphylococci had been added the completed phagocytic mixtures were incubated for another 15' | 1·61 | 1·38 | 4·7 | 1·8 | 0·64 | 3·0 | 1·2 | 0·21 | 2·0 | 1·83 |
| Here the same procedure was repeated with HEATED SERUM | 1·85 | 3·4 | 7·3 | 2·0 | 2·9 | 5·0 | 1·95 | 1·9 | 2·85 | 3·1 |

In the first section of Table V it will be seen that the opsonic power of the serum is, as was previously found by Douglas and myself, very much reduced by heating. The figures set forth in the second section of the table throw, however, fresh light upon the question, for they show that heating does a great deal besides reducing the opsonic index of the serum. It, as the figures show, converts the serum into a powerful leucocytic stimulin. It is therefore probable that *some* (one does not know how much) of the phago-incitor action, which was attributed by Neufeld and Rimpau to a "bacteriotropin" and by Reid and myself to "thermostable opsonins" may have been attributable to "thermo-generated stimulins".

I leave that question for a moment to consider another fundamental point which I and my fellow-workers elicited with regard to phagocytosis in our original researches on that subject—researches which we may, using an apt expression of Francis Bacon, call our *Vindematio Prima* or "First Grape-harvesting".

We had concluded in that *Vindematio Prima* that leucocytes did not ingest microbes in the absence of serum; in other words that leucocytes could not be credited with the faculty of spontaneously phagocytosing microbes.

That question obviously required to be re-examined. For our original findings might quite well hold of normal leucocytes, and yet not hold of the leucocytes of auto-inoculating patients. And that, as the results displayed in Table VI (Columns 2 and 3), and VII. and VIII show, is what we do actually find.

TABLE VI.—SPONTANEOUS PHAGOCYTOSIS TO STAPHYLOCOCCI—COMPARISON OF NORMAL LEUCOCYTES WITH THOSE OF AUTO-INOCULATING PATIENTS.

Technique.—The washed corpuscles derived from defibrinated bloods are mixed in each case with an equal volume of an 8 to 12-fold dilution of a twenty-four-hour broth culture of staphylococcus. These mixtures are then centrifuged for forty-five seconds in one direction and then for forty-five seconds in the opposite direction. The supernatant fluid is then amputated and film preparations made from the corpuscular deposit.

About 200 leucocytes are then counted in each specimen and the average staphylococcic intake of the individual leucocytes is determined in the ordinary way by dividing the total number of microbes ingested by the number of leucocytes counted.

| Single observations of normal men | | Single observations of febrile and non-febrile patients suffering from: | | | |
|-----------------------------------|------|-------------------------------------------------------------------------|--------------|-------------------------------------|------|
| A. S. | 0.04 | Gonorrhoea | 0.86 | Strept. septicaemia | 0.25 |
| D. S. | 0.03 | Gonococcal arthritis | 0.88 | Puerperal fever—Case 1 | 0.25 |
| C. W. M. | 0.01 | Rheumatism—Case 1 | 0.25 | Puerperal fever—Case 2 | 0.22 |
| F. H. | 0.02 | Rheumatism—Case 2 | 0.48 | Puerperal fever—Case 3 | 0.15 |
| A. F. W. | 0.02 | Rheumatism—Case 3 | 0.11 | Puerperal cellulitis | 0.20 |
| A. H. | 0.02 | Chancre | 0.60 | Septic pneumonia | 0.19 |
| E. A. B. | 0.05 | Secondary syphilis | 0.22 | Febrile cellulitis | 0.20 |
| K. B. R. | 0.05 | Endocarditis—Case 1 | 0.26 | Mastoid infection | 0.55 |
| | | Endocarditis—Case 2 | 0.70 | Appendicitis with peritonitis | 0.27 |
| | | Endocarditis—Case 3 | 0.70 | Cervical abscess | 1.00 |
| | | Tubercular peritonitis | 0.35 | Hæmorrhagic colitis | 0.16 |
| Average | 0.03 | Average | 0.4 (nearly) | | |

TABLE VII.—SPONTANEOUS PHAGOCYTOSIS IN (FOR THE MOST PART FEBRILE) PHTHISIS.

The technique here used was the same as that employed above.

Average result of the observations made with the leucocytes of normal men

0.03

Results of single observations made upon febrile and non-febrile phthisical patients

| | | | |
|--------|------|---------|------|
| Case 1 | 1.59 | Case 10 | 0.29 |
| " 2 | 0.54 | " 11 | 0.37 |
| " 3 | 0.21 | " 12 | 0.37 |
| " 4 | 0.54 | " 13 | 0.10 |
| " 5 | 1.80 | " 14 | 0.54 |
| " 6 | 0.50 | " 15 | 1.50 |
| " 7 | 0.13 | " 16 | 0.74 |
| " 8 | 0.10 | " 17 | 1.20 |
| " 9 | 0.06 | " 18 | 0.86 |

Average 0.65

Average 0.03

In view of these data it is obvious that we should, if we had not already been compelled to do so by Shattock and Dudgeon's work and by the data set out in Table III, have had

to delete from our immunological code the pronouncement that leucocytes are unaffected by inoculation.

And it emerges from comparative observations which now run into hundreds that we get on the whole much better information about immunizing response by testing the leucocytes of an infected patient than by testing his serum. Also we get our results with much less trouble.

An example of epiphyllactic response to an inoculation of Tuberculin B.E. is furnished in the table below.

TABLE VIII.—SPONTANEOUS PHAGOCYTOSIS OF STAPHYLOCOCCUS BY THE WASHED CORPUSCLES OF A PATIENT IN THE LAST STAGES OF PHTHISIS: BEFORE AND AFTER THE INOCULATION OF TUBERCULIN B.E.

| | | |
|----------|--------------------------------------------|------|
| 25.10.32 | Before inoculation | 1.58 |
| | Inoculation of $\frac{\text{B.E.}}{3,000}$ | |
| 25.10.32 | Two hours after inoculation | 0.63 |
| 27.10.32 | | 3.26 |
| 31.10.32 | | 2.03 |
| 9.12.32 | Six hours before death | 0.32 |

The point of superiority which this method has over all others is that incubation is avoided, and with that, almost all the possibility of auto-inoculation *in vitro*.

Another point of very practical interest which emerges from consideration of the data set forth in the tables above is that we have here an unflattering method of determining what dose of vaccine to administer to a patient to evoke the best epiphyllactic response.

STIMULINS

The term "*stimulin*" was introduced by Metchnikoff; and what he had in view when he spoke of a "*stimulin*" was a product of active immunization which circulated in the blood of immune animals and men and which operated by "training" the leucocytes to attack the species of microbes which had supplied the immunizing ictus. In other words a "*stimulin*" as conceived by Metchnikoff was a kind of hormone engendered in the animal body in response to an inoculation or auto-inoculation.

It will be appreciated that thermo-generated stimulins of serum which have been considered above belong to a category of stimulins which have nothing in common with those which Metchnikoff had in mind, except only the fact that they are leucocytotropic and not bacteriotropic—that is to say they operate on the leucocytes instead of combining chemically with the microbes. But they differ from Metchnikoff's notion of a *stimulin* is not being generated in response to an immunizing ictus. And this holds true even more emphatically of the bacterial antigens which come under consideration in the next table.

That bacteria and bacterial derivatives function as leucocytic stimulins may be said to be *a priori* probable, and experimental confirmation of this deduction is as a matter of fact very easily obtained.

One of the simplest ways of doing this is to make caesuric phagocytic experiments such as those set out in Table IX. Here there was made (A) an ordinary compendial phagocytic mixture; and (B) a phagocytic mixture made by adding 1 volume of serum to 2 volumes of a microbi-corpuscular pre-phagocytic mixture which had been incubated for fifteen minutes.

TABLE IX.

| | Phagocytic counts obtained in experiments No. | | | | | |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|------|------|------|------|---------|
| | 1 | 2 | 3 | 4 | 5 | Average |
| A. Here the microbes, leucocytes and serum were incubated, all of them together for 15 minutes | 3.9 | 1.4 | 0.63 | 0.44 | 1.4 | 1.55 |
| B. Here the microbes and leucocytes were incubated first for 15 minutes by themselves; and this microbi-corpuscular pre-phagocytic mixture was, after it had been by the addition of serum, converted into a phagocytic mixture, reincubated for another 15 minutes | 6.2 | 2.58 | 2.1 | 1.35 | 2.58 | 2.96 |

We here have evidence that the microbes which are added to a phagocytic mixture to provide pabulum for the leucocytes operate upon these as vaccines. If further evidence of vaccines acting as leucocytic stimulins is required we have it in the subjoined table. And this table further shows that the *stimulin* action exerted by vaccines is non-specific.

TABLE X.—COMPARISON OF THE PHAGOCYTOTIC INTAKE OF (A) LEUCOCYTES INCUBATED IN NORMAL SALT SOLUTION; AND (B) OF LEUCOCYTES INCUBATED IN NORMAL SALT SOLUTION CONTAINING GRADUATED QUANTA OF TUBERCULIN B.E.

The stimulin effect was judged of by the number of microbes ingested when the two kinds of leucocytes in question were incubated or centrifuged with a suspension of staphylococcus.

The figures for the different samples are expressed below as the ratios, the figure for the Control being arbitrarily represented as 1.

| No. of Expt. | Length of incubation | Phagocytic efficiency of the leucocytes operating in plain normal saline taken as | Phagocytic efficiency of the leucocytes operating in normal saline containing B.E. in the following quantities: | | | | | |
|--------------|------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------|------------------|---------------------|---------------------|---------------------|-----------------------|
| | | | $\frac{1}{2M}$ to | $\frac{1}{M}$ to | $\frac{1}{100T}$ to | $\frac{1}{300T}$ to | $\frac{1}{100T}$ to | Above $\frac{1}{10T}$ |
| | | | $\frac{1}{15M}$ | $\frac{1}{65M}$ | $\frac{1}{400T}$ | $\frac{1}{100T}$ | $\frac{1}{10T}$ | |
| 1 | 9 minutes | 1 | - | - | - | - | 17 | - |
| 2 | 35 minutes | 1 | 1.5; 5.6 | - | - | 8.5; 1 | - | 1 |
| 3 | 60 minutes | 1 | 2 | - | 3.1 | 3.7 | - | - |
| 4 | Not noted | 1 | - | - | - | 12 | 24; 24 | - |
| 5 | 20 minutes | 1 | 4.6; 4 | 10 | 3 | 1 | - | - |
| 6 | 60 minutes | 1 | - | - | 2.5 | - | - | - |
| 7 | immediate centrifuging | 1 | 11 | 17 | - | - | - | - |
| 8 | 1 hr. 45 min. | 1 | - | - | - | 3 | 2 | 5; 10 |
| 9 | 30 minutes | 1 | 5; 6 | 15 | 18 | - | - | - |

M = 1 millionth, T = 1 thousandth of a tuberculin suspension containing 1 mg. per c.c. of powdered tubercle bacilli.

Where the two separate figures appear in a single column of the table these indicate that I was here dealing with two specimens of blood which fell within the particular rubric.

We have still to consider more than one other class of stimulins. Among these there have to be considered drugs. And the drugs which especially invite examinations are the sulphanilamides. I have made experiments with one of these—M & B 693.

The results obtained are set out in the next table.

TABLE XI.—SHOWING THE PHAGOCYTOTIC COUNTS OBTAINED WITH NORMAL BLOOD, SERUM, AND CORPUSCLES, AND THOSE OBTAINED WITH THE SAME BLOOD, SERUM OR CORPUSCLES, INCUBATED WITH "693"; TOGETHER WITH (THESE BEING ADDED FOR THE SAKE OF COMPARISON) THE PHAGOCYTOTIC COUNTS OBTAINED WITH SERUM, BLOOD OR CORPUSCLES TREATED WITH VACCINES.

| Serial No. of Experiment | Length of time during which the drug or vaccine operated upon the blood | Amount of the drug or of the vaccine which was added to the different blood specimens | | | Phagocytic indices of blood specimens | | | Procedures by which the figures here recorded were obtained |
|--------------------------|-------------------------------------------------------------------------|---------------------------------------------------------------------------------------|---------------|------------------------|---------------------------------------|-------|-------|-------------------------------------------------------------|
| | | No. 1 | No. 2 | No. 3 | No. 1 | No. 2 | No. 3 | |
| Expt. 1 | 75 minutes | Nil | 693 100000 | B.E. 3000000 | 1 | 1.3 | 1.4 | Procedure 1 |
| Expt. 2 | 30 minutes | " | " | " | 1 | 1.8 | - | " 1 |
| | | " | " | " | 1 | 2.5 | - | " 2 |
| | | " | " | " | 1 | 0.7 | - | " 3 |
| | | " | " | " | 1 | 2 | - | " 4 |
| Expt. 3 | 40 minutes | Nil | " | " | 1 | 1 | 1.6 | " 1 |
| | | " | " | " | 1 | 1.4 | 1.7 | " 3 |
| | | " | " | " | 1 | 1.4 | 1.2 | " 4 |
| Expt. 4 | 30 minutes | " | " | 3,300 Staph. per c.c. | 1 | 1.2 | 1.4 | " 1 |
| | | " | " | " | 1 | 1.6 | 1.5 | " 3 |
| | | " | " | " | 1 | 2.1 | 1.1 | " 4 |
| Expt. 5 | 33 minutes | " | " | 10,000 Staph. per c.c. | 1 | 1.2 | 1.4 | " 1 |
| | | " | " | " | 1 | 1.6 | 1.7 | " 3 |
| | | " | " | " | 1 | 1.3 | 2.4 | " 4 |
| Expt. 6 | 30 minutes | " | " | B.E. 3000000 | 1 | 0.9 | 1.3 | " 1 |
| | | " | " | " | 1 | 0.8 | 0.6 | " 2 |
| | | " | " | " | 1 | 1 | - | " 3 |
| | | " | " | " | 1 | 1.3 | 1.1 | " 4 |

Procedure 1: The separated washed corpuscles of the different samples of blood were incubated for fifteen minutes with normal serum and staphylococci suspended in normal saline.

Procedure 2: The separated sera of the bloods were incubated for fifteen minutes with normal washed corpuscles and staphylococci.

Procedure 3: The sera were incubated for fifteen minutes with staphylococci suspended in normal saline, and then after normal washed corpuscles had been added to the sero-microbic mixture, the completed phagocytic mixtures were incubated for another fifteen minutes.

Procedure 4: The sera were incubated for fifteen minutes with washed normal corpuscles and after staphylococci had been added to the sero-corpuscular mixture the completed phagocytic mixture was incubated for another fifteen minutes.

Epitome of the data set out in the foregoing table.

It will be seen on study of the table that $\frac{1}{50000}$ of M & B 693 operates upon the leucocytes as a stimulin—we may call such stimulins "*pharmaco-stimulins*"—and that it increases their phagocytic avidity in the ratio of 1:1.4 (this being the average of six experiments); and sometimes as much as 1:2.5. This result compares with an average stimulin effect of 1:1.27 obtained under the same conditions with blood inoculated with $\frac{1}{500000}$ B.E.; and 1:3 for blood inoculated with 3,300 staphylococci per c.c.

Going back now to Tables IX, X and XI it will be seen that we have had demonstration in these of three different kinds of stimulins: in IX of *stimulins produced by heating serum*; in X of *bacterial stimulins*; and in XI of both these and of *pharmaco-stimulins*. To these may now be added a fourth kind of stimulins—those which can be extracted from leucocytes by incubating them in normal salt solution. These may be conveniently called *endo-leucocytic stimulins*.

I shall discuss these stimulins on another occasion for there are certain points more relevant to our subject matter which still require to be considered. The first of these is:

POSSIBLE BEARING OF THE FACTS IN TABLES IX, X AND XI ON THE TREATMENT OF BACTERIAL INFECTIONS IN WOUNDS

In the researches on the infections of granulating wounds which were carried out during the last war by my fellow-worker Prof. A. Fleming, striking results were obtained by making impression preparations from wound surfaces which had been untreated for some hours. The cover-glasses which were used for making the specimens were then immediately turned down upon agar plates, and were then incubated.

Here the ordinary result was for fairly numerous colonies—for the most part colonies of staphylococci and streptococci—to grow out under the cover-glasses wherever there were any vacant spaces between the blobs of pus.

And when impression preparations were taken from a wound after it had been carefully washed, leucocytes were here, as is natural, entirely wanting; but the bacterial colonies were now more numerous than before for the microbes could now, in the absence of leucocytes, grow out unrestrained.

When specimens were taken a little later, and freshly emigrated leucocytes had occupied the surface of the wound there were only few, and in many cases no surviving microbes.

And finally after a considerable interval of time microbic colonies again grew out in the impression preparations in the interspaces between the blobs of pus—this showing that the sterilization of the wound by the leucocytes had been somewhere or other left incomplete.

The important point is that when the corrupted discharges have been carefully removed from the wound surface, and this is maintained in a condition favourable to the emigration of leucocytes and to their bactericidal action, the wound is so nearly sterilized that only a slight reinforcement of the bactericidal forces already in action would seem to be required to effect complete sterilization.

Now that reinforcement might possibly, as the results set out in Tables IX, X, and XI show, be supplied by a local application of a leucocytic stimulin either in the form of a sulphanilamide or a bacterial vaccine.

The local application of sulphanilamide has of course become the routine treatment of bacterial infections of wounds.

But we do not know whether the drug should be employed in the strength which gives the most effective specific bactericidal action, or in the strength which gives the best results as a non-specific leucocytic stimulin.

That vaccines might also be employed in the treatment of bacterial infections of wounds would seem clear not only from the results set out in the tables just referred to, but also from *in vitro* experiments carried out as follows:

Two filter paper discs are taken and soaked—one in a very high dilution of a broth culture of staphylococcus (a watery dilution containing 1,000 to 3,000 staphylococci per c.c.); and the other in the same dilution of staphylococcus culture to which an appropriate dose of vaccine has been added.¹ Both discs are drained of all superfluous fluid and are then flooded with the same amount of defibrinated blood, or blood taken direct from the finger, and the discs are then covered in with cover-glasses and incubated for not less than 3 hours.

After that the paper discs are taken from under the cover-glasses; the red blood corpuscles are dissolved in hot water; and the now decolorized discs are lightly stained with methylene blue. A comparison now shows that always fewer—and generally very many fewer—staphylococcus colonies have grown out in the vaccinated than in the control disc.

There is nothing fundamentally new in this result. For exactly the same result is obtained when an appropriate dose of vaccine is added to the outside blood instead of being added to the paper disc.

In the case where we put the vaccine into the paper disc we are doing the same experiment as we should be doing if we put vaccine into a patient's wound; and when we put the vaccine into surrounding blood we are doing the same experiment as we should be if we were applying the vaccine in the ordinary way.

Let me now as a preface to what I want to say about vaccines dwell for a moment on the, at first sight, paradoxical fact that the blood, when it is tested under conditions in which epi-phylactic response is excluded, has no staphylo-phyllactic power; that is to say it kills either no staphylococci or at best a minimal number of these.

I have dealt with the facts which lead up to this conclusion (I think it is a conclusion which applies generally to all serophytes) in a series of three papers, the first two of which were published in 1902² and in 1915.³

In the first paper I showed that the serum exerts absolutely no bactericidal power upon the staphylococcus or streptococcus or the *M. melitensis* or the plague bacillus. In the second I emphasized the fact that the serum furnishes an ideal cultivation medium for the whole of septicæmia-producing or as I prefer to call them, *serophytic microbes*.

And finally I showed in a paper published in 1923⁴ that the normal whole blood exerts upon the staphylococcus practically no bactericidal power; and that we can never, as we do when we are speaking of the typho-bactericidal power of the normal serum, say of the non-epiphyllactic blood that it will kill a definite number of staphylococci or other serophytes.

My whole blood, for example, exerts no bactericidal power when it is implanted with minimal numbers of staphylococci. It begins to develop bactericidal power only when more than 200 staphylococci per cubic centimetre are implanted. And it develops its maximum bactericidal power when it is implanted with between 4,000 and 5,000 staphylococci per cubic centimetre.

I have called that the *Caput and Cauda phenomenon* because when the figures for bactericidal power achieved by graduated doses of vaccine are set out in the form of a table we find that the percentage of microbes killed in the more heavily implanted bloods which occupy the head or caput of the table is always much greater than those for the lightly implanted bloods which occupy the whole tail of the cauda of the table.

¹ I have always used a vaccine consisting of 1:100,000 dilution of B.E. suspension which contained 1 mg. of desiccated tubercle.

² *Journal of Hygiene*, 1902, 2, No. 4 (reprinted in the Author's "Studies on Immunisation", Constable).

³ *Proc. Roy. Soc. Med.*, October 8, 1915.

⁴ *Lancet*, February 24, 1923, p. 368.

This fact that the blood, which had originally none, develops a bactericidal power to staphylococcus under the stimulus of a sufficient bacterial implantation, furnishes, as we shall see later, a possible explanation of the fact that the effects of prophylactic inoculation persist long after the manifest blood changes have passed off.

From that I pass to put before you certain general considerations about vaccines. Let me deal first with the question of dosage.

DOSAGE OF BACTERIAL VACCINES

An all-important first principle to appreciate in connexion with the testing of the phagocytic power developed in the blood by vaccines applied therapeutically *in vivo* or *in vitro* is that the phagocytic intake is never a simple function of the strength of the vaccine brought into application. Epiphyllactic response is powerfully affected (this comes out with special clearness when we measure the phagocytic response to a vaccine implanted into blood *in vitro*) by the larger or smaller number of microbes we employ as "*phagocytic pabulum*".

The smaller the number of microbes employed as "*pabulum*" the larger is the dose of vaccine required to give a maximal epiphyllactic response. And the larger the number of microbes employed as phagocytic pabulum the smaller is the quantum of vaccine required to give the same measure of response. In other words the principle of dosage which has to be borne in mind is that the epiphyllactic ictus brought to bear upon the leucocytes is always composed of two or three factors—two in the case of blood inoculated *in vitro* (one being the microbes supplied to serve as a phagocytic pabulum, and the other the amount of vaccine superadded); and three in the case where an infected patient is inoculated (the first being the number of microbes harboured in his body; the second being the dose of vaccine administered; and the third being, when his phagocytosis is being tested, the phagocytic pabulum employed in the test).

The influence of the different amounts of staphylococcal pabulum employed in the phagocytic tests comes out clearly in Table X. The quantitatively different epiphyllactic responses there recorded in different experiments in which one and the same dose of tubercle vaccine was employed would seem to be due to the different strengths of bacterial pabulum used in the different experiments.

Experiments made *ad hoc* bring confirmation of the principle in question. In the experiments set out below equal samples of my defibrinated blood were incubated with two different doses of Tuberculin B.E. vaccine there being, in the two sets of experiments, added to the phagocytic mixtures by way of pabulum the same quanta of staphylococcus culture.

TABLE XII.

| Serial No. of expt. | Composition of Phagocytic mixtures employed | Phagocytic index of the vaccinated blood |
|------------------------|------------------------------------------------------------------------------------------|------------------------------------------------|
| 1 | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.7 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.8 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.1 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.2 |
| 2 | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.0 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.1 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.1 |
| | 2 vols. blood inoculated with B.E. per c.c. + 1 vol. $\frac{\text{Staph.}}{\text{T.M.}}$ | 1.5 |

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⁴ *Lancet*, February 24, 1923, p. 368.

(1) Do the bacteriotropic substances which are produced by and any stimulins which are set free in the body in response to the inoculation of a bacterial vaccine give protection only against microbes which are culturally, clinically, chemically and immunologically indistinguishable?

(2) Do these substances protect not only against the microbes which constitute the prime target of inoculation, but in addition against microbes which have slightly different cultural, clinical and immunological properties, but would be included under one and the same microbic species?

(3) Do the substances in question operate not only upon *homonymous* but also upon *heteronymous* microbes?

To the *first question* a definite answer is given in the fact that prophylactic inoculation—I limit myself for the moment to anti-typhoid and anti-cholera vaccination—would seem to be everywhere effective, i.e. effective upon all the different strains or varieties of typhoid and cholera which exist. And we have striking confirmation of this applying generally in the fact that vaccine therapy with stock vaccines is—at any rate in the majority of cases—as effective as treatment with autogenous vaccines.

With regard to the *second question*, a unique opportunity for deciding whether a bacterial inoculation is specific in the narrowest sense, or whether it protects also against homonymous microbes of different kinds, presented itself in the 1914-18 war. For there then offered themselves for observation: a large population of soldiers who were inoculated (in the early years of the war) with a simple typhoid vaccine; and side by side with these a large population of soldiers who were (in the later years of the war) inoculated with a typhoid vaccine in conjunction with a paratyphoid A and a paratyphoid B vaccine.

That statistical opportunity was missed because it was thought necessary to add to the typhoid vaccine, without reducing the dose of that vaccine, half as much paratyphoid A vaccine and half as much paratyphoid B vaccine—this of course resulting in the second population of soldiers being inoculated with double the number of microbes given to the first population.

And at any rate the question under investigation remains unresolved for the statistics which were collected showed that there was no significant difference between the number of cases of paratyphoid A and B in the two populations.

And further evidence pointing to non-type specific immunization is furnished by the favourable prophylactic results against pneumococcus which were obtained by myself and my fellow-workers in the Transvaal with vaccines made with untyped pneumococcus.

The very striking results in question are set forth in a series of papers which were published in the *Lancet* in 1912 and were republished in book form.¹

Facts which would seem to conduct to a directly contradictory conclusion have been put on record by many—chiefly American—investigators.

The experiments of these investigators would seem to indicate that animals which are inoculated with one particular strain of pneumococcus are protected only against that particular strain. But these, being purely statistical experiments have, like all such experiments, the vice of taking cognizance of only one particular effect—in this case *survival or non-survival after inoculation with supra-lethal doses*. And when this is borne in mind it becomes plain that all the statistical results I am referring to really establish is that better protection is given against the strain of pneumococcus which was employed as a vaccine than against other strains.

And what we have to settle before we call a pneumococcus type-specific or non-type-specific is *how much* protection is afforded against “homologous” and how much against “heterologous” strains.

Again most of the facts which are relied upon as proof of non-specific response to pneumococci are results obtained by serum therapy on animals and man. And there is, as we shall see in a moment, great difficulty in referring the favourable results obtained by pneumococcal serum-therapy to the administration of epiphyllactic substances elaborated in the organism of the donor animal.

¹ “On Pharmacotherapy and Preventive Inoculation applied to Pneumonia in the African Native”. Constable, 1914.

We have in this, and the previous tables, the answer to the question of dosage which should, when treating a patient by vaccine therapy, be kept constantly in mind. These tables show that the initial dose of vaccine to be administered to an infected patient must always be a matter of clinical judgment—the heavier the patient's infection the smaller should be the amount of vaccine given for a first inoculation. And the rule for subsequent inoculations is this: When the first dose is adjudged to have been operative and to have exerted a favourable result, and when we have good reason to think (but never before) that the population of microbes in the patient's body has been reduced, we may correspondingly increase the dose of vaccine administered. And when the infection is very nearly extinguished we may use a dose of vaccine little less than that which would be administered for prophylaxis to a healthy man.

QUESTION AS TO WHETHER BACTERIAL VACCINES OPERATE SPECIFICALLY OR NON-SPECIFICALLY

As the terms "specific" and "non-specific" carry different meanings for different persons it will be necessary to begin by saying something about the terminology.

If only the single strain of microbes out of which the vaccine has been manufactured is killed or impeded in its growth by the vaccine inoculated the effect of the inoculation would be *specific in the narrowest sense*, i.e. type or strain-specific. If on the other hand the bacterial vaccine operates upon all microbes which are "*homonymous*" with that from which the vaccine was made the effect would be *non-specific in the sense which the term is customarily used*. And if, in addition to homonymic, heteronymic microbes are killed or are impeded in their growth by the vaccine inoculated, the immunizing effect exerted would be *non-specific in the widest sense*.

Homonymous microbes would be those which are called by the same name because they have similar cultural characters; produce similar clinical symptoms; manufacture similar chemical products; generate, when used as vaccines similar epiphytactic elements; and have, subject to the qualification below, similar chemical susceptibilities.

It is necessary to appreciate that when microbes which are called by the same name are found to differ in any cultural, clinical or immunological character; and also when they have been found to produce different chemical products; and to have different chemical susceptibilities; they can by the aforesaid criteria be distinguished into *sub-species*, or *types*, or *variants* or strains which finally receive distinguishing appellations of some sort.

There have in this way been differentiated and this concerns the specific action of vaccines, the human, bovine, avian and reptilian strains of tubercle bacillus; and the sub-species or variants of the typhoid bacillus which are known as paratyphoid A and paratyphoid B.

Heteronymous microbes would be microbes which are called by different names because they have different cultural characters, produce different clinical symptoms, and different immunological responses; and have—this being subject to the reservation now to be made—different chemical susceptibilities.

Susceptibility or insusceptibility to one particular reagent cannot be regarded as a trustworthy criterion of generic affinity or lack of such affinity. This comes out clearly in the fact that M & B 693 operates with conspicuous effect only upon one particular strain of streptococci, while it operates also upon microbes as generally distinct as the meningococcus and the pneumococcus.

And further proof that the susceptibility or insusceptibility to one particular reagent is not of classificatory importance, is furnished by the fact that it is possible to breed out so-called "arsenic-fast" types of trypanosomes which do not as far as it is known differ from unmodified trypanosomes except only in the matter of being resistant to one particular arsenical.

These points of terminology having been made clear it will be seen that the question as to whether bacterial vaccines operate specifically or non-specifically resolves itself into three different questions.

(1) Do the bacteriotropic substances which are produced by and any stimulins which are set free in the body in response to the inoculation of a bacterial vaccine give protection only against microbes which are culturally, clinically, chemically and immunologically indistinguishable?

(2) Do these substances protect not only against the microbes which constitute the prime target of inoculation, but in addition against microbes which have slightly different cultural, clinical and immunological properties, but would be included under one and the same microbic species?

(3) Do the substances in question operate not only upon *homonymous* but also upon *heteronymous* microbes?

To the *first question* a definite answer is given in the fact that prophylactic inoculation—I limit myself for the moment to anti-typhoid and anti-cholera vaccination—would seem to be everywhere effective, i.e. effective upon all the different strains or varieties of typhoid and cholera which exist. And we have striking confirmation of this applying generally in the fact that vaccine therapy with stock vaccines is—at any rate in the majority of cases—as effective as treatment with autogenous vaccines.

With regard to the *second question*, a unique opportunity for deciding whether a bacterial inoculation is specific in the narrowest sense, or whether it protects also against homonymous microbes of different kinds, presented itself in the 1914-18 war. For there then offered themselves for observation: a large population of soldiers who were inoculated (in the early years of the war) with a simple typhoid vaccine; and side by side with these a large population of soldiers who were (in the later years of the war) inoculated with a typhoid vaccine in conjunction with a paratyphoid A and a paratyphoid B vaccine.

That statistical opportunity was missed because it was thought necessary to add to the typhoid vaccine, without reducing the dose of that vaccine, half as much paratyphoid A vaccine and half as much paratyphoid B vaccine—this of course resulting in the second population of soldiers being inoculated with double the number of microbes given to the first population.

And at any rate the question under investigation remains unresolved for the statistics which were collected showed that there was no significant difference between the number of cases of paratyphoid A and B in the two populations.

And further evidence pointing to non-type specific immunization is furnished by the favourable prophylactic results against pneumococcus which were obtained by myself and my fellow-workers in the Transvaal with vaccines made with untyped pneumococcus.

The very striking results in question are set forth in a series of papers which were published in the *Lancet* in 1912 and were republished in book form.¹

Facts which would seem to conduct to a directly contradictory conclusion have been put on record by many—chiefly American—investigators.

The experiments of these investigators would seem to indicate that animals which are inoculated with one particular strain of pneumococcus are protected only against that particular strain. But these, being purely statistical experiments have, like all such experiments, the vice of taking cognizance of only one particular effect—in this case *survival or non-survival after inoculation with supra-lethal doses*. And when this is borne in mind it becomes plain that all the statistical results I am referring to really establish is that better protection is given against the strain of pneumococcus which was employed as a vaccine than against other strains.

And what we have to settle before we call a pneumococcus type-specific or non-type-specific is *how much* protection is afforded against “homologous” and how much against “heterologous” strains.

Again most of the facts which are relied upon as proof of non-specific response to pneumococci are results obtained by serum therapy on animals and man. And there is, as we shall see in a moment, great difficulty in referring the favourable results obtained by pneumococcal serum-therapy to the administration of epiphyllactic substances elaborated in the organism of the donor animal.

¹ “On Pharmacotherapy and Preventive Inoculation applied to Pneumonia in the African Native”. Constable, 1914.

The *third issue* as to whether bacterial vaccines can, or cannot, afford protection against heteronymous microbes, can be put to the test in a number of different ways.

We have already in this connexion considered the observations which were made by Shattock and Dudgeon on the one hand, and by (Table IV *supra*) Dr. Ronald Hare on the other.

And what looks like clear statistical evidence of the protective effect exerted by a vaccine on heteronymous microbes is that set out in the following table, which shows the results obtained by pneumococcal vaccination in the Premier Mines in the Transvaal in 1911.

TABLE XIII.—SHOWING FOR THE WHOLE NATIVE POPULATION OF THE PREMIER MINE THE INCIDENCE AND DEATH-RATE FOR PNEUMONIA; THE INCIDENCE AND DEATH-RATE FOR "OTHER DISEASES"; AND ALSO THE NUMBER OF WORKING DAYS LOST THROUGH ILLNESS; FOR THE MONTHS OF FEBRUARY TO MAY, IN 1911, 1912, AND 1913 RESPECTIVELY.

| | 1911 | 1912 | 1913 |
|------------------------------------------------------|--------|-----------|-----------|
| Population (daily average strength) | 16,426 | 12,549 | 15,284 |
| Proportion of the population inoculated | 0 | About 50% | About 92% |
| Incidence-rate of pneumonia | 4% | 1.28% | 0.74% |
| Death-rate from pneumonia | 0.07% | 0.31% | 0.14% |
| Incidence-rate of other diseases | 31% | 20.7% | 14.4% |
| Death-rate from other diseases | 0.51% | 0.38% | 0.34% |
| Number of working days lost per 100 native labourers | 275 | 177 | 131 |

* In 1912 the incidence-rate was 0.86% for the inoculated, and 1.7% for the uninoculated. In 1913 it was 0.5% for the inoculated, and 3% for the controls.

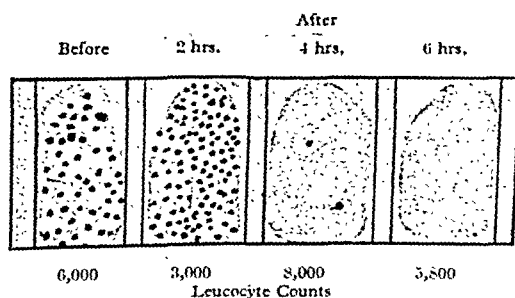
Further, convincing clinical evidence of the non-specific effect of vaccines is available in the fact that the intra-vascular inoculation of typhoid and coliform vaccines has proved itself an effective treatment for many different infections.

In particular it is credited with having given strikingly favourable results in gonococcal infections.

That these results find their most natural explanation in a non-specific immunizing response and not in a newly-invented type of reaction which has been christened "Protein Shock," will not be doubtful to anyone who has, from laboratory experimentation, learned to expect non-specific immunization *in vitro*.

Passing now from clinical to clinico-laboratory experiment I may lay stress on an experiment I published ten years ago. This showed that an intravenous inoculation of coliform vaccine first markedly diminished the staphylo-bactericidal power of the patient's blood and then formidably increased it.

FIG. 3.



Non-specific Immunization. Epiphyllactic effect of 200 million coli vaccine administered intravenously. Tested in a slide cell by implanting the same number of staphylococci into each of the four successive 50 c.mm. volumes of blood.

The first sample of the patient's blood, taken before inoculation, killed off (see fig. 3) all except some 50 of the implanted staphylococci. In the second sample drawn two hours after inoculation, about double that number of staphylococci grew out into colonies. The third sample, drawn two hours after that killed all but two of the staphylococci implanted. And the fourth and last sample of blood, drawn from the patient six hours after inoculation, killed all the implanted and might easily, if it had been tested higher, have killed many more.

Non-specific epiphyllactic responses of that sort are obtained whenever blood which has been inoculated with vaccine *in vitro* is tested by hæmo-bactericidal methods, i.e. by the slide cell or the capillary clot methods.

HAS CONVINCING PROOF OF ANY PURELY SPECIFIC IMMUNIZATION EVER BEEN BROUGHT FORWARD?

I shall confine myself to my own work and that of my fellow-workers. To test the specificity of the response to typhoid vaccine, I took the sera of six laboratory workers who had been inoculated against typhoid and tested their bactericidal power both to cholera and typhoid. I found in these experiments that the sera I was dealing with showed a greatly increased bactericidal effect to typhoid but no increased bactericidal power to cholera. I inferred from that that bacterial vaccines produce only specific epiphyllactic effects. But in coming to this conclusion I had neglected to think of the leucocytes.

I may instance the further fact that when my fellow-worker, Dr. John Freeman, massaged a gonococcal knee with a view to determining whether the auto-inoculation produced would have a specific or non-specific effect, he found¹ that it markedly increased the phago-incitor power of the serum to gonococcus, but left the phago-incitor power of the serum to the tubercle bacillus quite uninfluenced.

Here again we all in my laboratory, quite forgetting that a different result might have been obtained if the leucocytes had been tested, rashly assumed that auto-inoculations operate only upon homonymous microbes.

Finally coming back to the fact that we get, as pointed out above, better results by testing leucocytic than by testing serum response, I may draw attention to the fact that when my colleagues and I were working on the pneumonia which was then epidemic in the Transvaal mines, we very laboriously searched for evidence of immunizing response to pneumococcus in the sera of ourselves, of healthy natives, and of natives who were suffering with pneumonia, and also of healthy natives inoculated with pneumococcus vaccine. We did this in order to test the effects of vaccine therapy, and also to choose a proper dose of vaccine for prophylactic inoculation. And the upshot of all this work was that satisfying evidence of the development of either increased bactericidal or phago-incitor power in the serum was conspicuously lacking.

The results obtained in our thousands of phago-incitor tests are too complicated to set forth here but the bactericidal results of our large numbers of serum tests are shown in Table XIV.

TABLE XIV.—SHOWING THE RESULTS OBTAINED BY TESTING THE PNEUMO-BACTERICIDAL POWER OF THE SERA OF FOUR LARGE GROUPS OF PERSONS.

| Source from which the sera were derived | Total number of observations | Number of observations in which no microbes were killed by the serum | Cases in which 30 pneumococci were killed by 50 c.mm. of serum | Cases in which 30-90 pneumococci were killed by 50 c.mm. of serum | Average number of pneumococci killed by 50 c.mm. of serum from the group |
|--------------------------------------------------------------|------------------------------|----------------------------------------------------------------------|----------------------------------------------------------------|-------------------------------------------------------------------|--------------------------------------------------------------------------|
| Healthy Europeans | 41 | 39 | 4 | 1 | 2 (approx.) |
| Healthy tropical natives | 27 | 19 | 8 | 0 | 4 (approx.) |
| Tropical natives after inoculation with pneumococcus vaccine | 176 | 161 | 15 | 0 | 1 (approx.) |
| Natives suffering from pneumonia | 20 | 11 | 9 | 0 | 5 (approx.) |

¹ Studies in Connexion with Therapeutic Immunisation, *Lancet*, November 2, 1907. (Reprinted in the Author's "Studies on Immunisation", p. 425, Constable, 1909).

And the very startling negative or practically negative results here recorded may be usefully contrasted with the fact that when at a later date the bloods of normal Europeans were tested by hæmo-bactericidal methods (i.e. slide cell or capillary clot methods) they gave instead of the small figures recorded above, figures of 50,000 and over of pneumococci killed per 50 c.mm. of blood.

The data in Table XIV suggest that the current interpretation of the favourable results of pneumococcal serum therapy may not be the right one. For if the sera of auto-inoculating pneumonic patients, and healthy men inoculated with pneumococcus vaccine, contain no bactericidal and seemingly no increased phago-incitor substances, it becomes difficult to believe that the sera of inoculated animals contain such substances.

IS THE ACCREDITED INTERPRETATION OF THE GOOD RESULTS OBTAINED WITH SERUM THERAPY OF PNEUMONIA THE ONLY POSSIBLE ONE?

There are two other possible interpretations of the recorded facts. The first is that antigens, possibly antigens which act as stimulins, may be contained in the pneumococcal serum administered. This general possibility has already been considered in connexion with Neufeld and Rimpau's reported thermostable phago-incitor substances.

And the second possible explanation of the favourable results obtained in human serum therapy is that the processes of concentration and re-solution which have been applied to the serum administered to man may have converted these sera into stimulins. Many of the results which I have obtained with re-constituted sera seem to suggest that.

One is driven to this desperate resource of guessing at the explanation of the favourable results of pneumonic serum therapy by the fact there would seem never to have been any proper evaluation of the epiphyllactic power of the anti-pneumococcus sera administered, or any proper evaluation of the blood of the patients before and after the administration of the serum.

PERSISTENCE OF THE PROTECTIVE EFFECTS OF PROPHYLACTIC VACCINATION

Everyone who has considered the evanescence of the increased bactericidal power which is achieved by antityphoid inoculation is agreed that the persistent specific immunization which is achieved must be due to some of the cells of the body remaining, long after all constitutional effects of inoculation have passed off, sensitive to re-immunization by such minimal typhoid inoculations as would inevitably occur in patients who were living in places where they are from time to time exposed to typhoid infection.

What cells in the body perform this work of re-immunization to typhoid is one of the important unresolved questions of bacteriology.

It may quite well be that it is the leucocytes which respond to infection; for that is not precluded by the fact that it is confidently asserted that that function is performed either by fixed cells distributed all over the body, or by fixed cells closely associated with the blood, and the ascription of this function to the leucocytes would at any rate harmonize with the fact that extravascular blood that possesses either none or only minimal staphylobactericidal power acquires when inoculated plentiful bactericidal power.

Further, it looks as if the question could easily be settled by direct experiment. It would be perfectly easy to take a series of samples of blood of patients who have been inoculated with typhoid (the taking of the first of these samples being deferred until the constitutional disturbance produced by the inoculation has died down) and to compare (using for this purpose very small doses of typhoid vaccine) the sensitiveness of the leucocytes of these bloods with that of the leucocytes of normal bloods.

If I were not too far "declined into the vale of years", and if I had not more important matters to investigate, I should myself undertake such a series of experiments. As things are I commend the suggested procedure to younger research workers.

Finally I subjoin, by way of a key to the reasoning in this paper, the following tables:

TABLE A.—PASTEURIAN DOCTRINE OF IMMUNIZATION

(This was based on what was known or believed to hold true of Jennerian vaccination; and it constituted the foundation of all the Pasteurian extensions of that system of immunization.)

- (1) Immunization can be undertaken whenever the pathogenic microbe of a disease has been isolated, or when material which contains that microbe is available.
- (2) It is a *sine qua non* for successful vaccination that it should be carried out with living microbes.
- (3) Immunity is established only after the expiration of ten days.
- (4) Immunization is practicable only in advance of the infection—with a possible exception in the case of any disease in which the incubation period lasts sufficiently long for immunization to be completed before the symptoms of the disease become manifest.
- (5) The amount of vaccine which is administered need not be minutely regulated. All that is required is to make sure that the microbe which is employed has been adequately and permanently attenuated.
- (6) Success or failure of an immunization procedure can be judged only by statistical methods.

That involves in the case of animals, inoculating them and their controls with a virulent culture of the specific microbe of the disease. And it involves in the case of man the registering of the incidence and death-rates of formidable numbers of vaccinated persons and controls who have been equally exposed to the infection against which it is hoped to give protection.

CONTRARY GENERALIZATIONS ENUNCIATED BY THE AUTHOR IN PAPERS
PUBLISHED BETWEEN 1897 AND 1901

- (1) Inoculations can be successfully carried out with sterilized vaccines.
The fact that agglutinins are elaborated in response to bacterial inoculations has been previously shown by Richard Pfeiffer.¹
- (2) An increase in the bactericidal powers of the blood can be registered very shortly; to wit, within one day or less after vaccination.²
- (3) Vaccines can be employed not only prophylactically but also therapeutically in the treatment of infected patients; and here the phagocytic power of the blood is very rapidly increased.³
- (4) The measure of immunity conferred by an inoculation procedure can be gauged by measuring the changes effected in the bactericidal and phagocytic power of the blood.

¹ Wright and Semple—"On vaccination against Typhoid Fever", Brit. M. J., January 30, 1897.

Wright and Leishman—"On the Results which have been Obtained by the Anti-typhoid Inoculations, and on the Methods which have been Employed in the Preparation of the Vaccine", Brit. M. J., January 20, 1900.

² Wright—"On the Changes Effected by Anti-typhoid Inoculations in the Bactericidal Power of the Blood, with Remarks on the Probable Significance of these Changes", Lancet, September 14, 1901.

³ Wright—"Notes on the Treatment of Furunculosis, Sycosis and Acne by the Inoculation of a Staphylococcus Vaccine, and Generally on the Treatment of Localized Bacterial Invasions by Therapeutic Inoculations of the Corresponding Bacterial Vaccines", Lancet, March 29, 1901.

TABLE B.—DEALING WITH THE MACHINERY OF IMMUNIZATION.

Rival Doctrines dating from 1890 or before.

Humoral Theory.—Immunization consists in the elaboration of bactericidal substances and in the conveyance of these into the blood-stream.

Phagocytic Doctrine of Metchnikoff.—Immunization consists in training the leucocytes to confront and phagocytose the specific pathogenic microbes against which the animal has been inoculated.

Doctrine of the author with regard to the machinery of immunization set out in papers published between 1907 and 1908.

Logical grounds which were felt to justify the conclusions set out in column 1

Revision in the light of new data of the doctrine set out in column 1

- | | | |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| (1) Phagocytosis of microbes takes place only in the presence of serum | When leucocytes have been washed free from serum phagocytosis of microbes is abolished | <i>Vide</i> new data set out in Tables VI, VII, and VIII, pp. 14 and 15. |
| (2) The chemical reaction which takes place in a phagocytic mixture (i.e. mixture of corpuscles, serum and microbes) is an opsonic action | Of the three <i>a priori</i> possibilities (1) that the serum acts opsonically on the microbes; (2) that the serum may function as a leucocytic stimulin; and (3) that the microbes which are added as phagocytic pabulum may vaccinate the leucocytes; only the first appears to be admissible | <i>Vide</i> new data showing that the microbes which are contained in a phagocytic mixture operate as a vaccine on the leucocytes of that mixture (<i>vide</i> Table IX, p. 15) and that a serum may exert a stimulin effect on the leucocytes. <i>Vide</i> Table V, Sects. 1 and 2, p. 13. |
| (3) Serum is rendered inert by heating to 60° C.—this result being due to the destruction of the opsonins | Comparison of the effects of heated serum with that of unheated serum (<i>vide</i> Table V, Sect. 1, p. 13) | <i>Vide</i> new data set out in Table V, Sect. 2, p. 13. |
| (4) Immunizing response of the patient to the inoculation of bacterial vaccines affects his serum and leaves his leucocytes unchanged | <i>Vide</i> Chiastic Experiments, <i>Proc. Roy. Soc.</i> , 73, 1903, and 74, 1904, republished in the Author's "Studies on Immunisation", pp. 87, 90 and 117. | <i>Vide</i> new data set out in Chiastic Table III, p. 12. |
| (5) The whole of the increased phagocytosis which is obtained by the inoculation of bacterial vaccines is to be ascribed to an elaboration of opsonins in the serum and not to an elaboration of leucocytic stimulins | This was inferred from Ehrlich's axiom that inoculations lead always to the elaboration of anti-thetic chemical substances, and as a corollary from that that bacterial vaccines lead to the elaboration of <i>bacteriotropic</i> as distinguished from <i>leucocytotropic</i> substances | <i>Vide</i> Tables X and XI for evidence of stimulin action exerted on leucocytes by bacterial vaccines, and also by M & B 603, pp. 16 and 17. |
| (6) Leucocytes kill microbes only intracellularly | <i>A priori</i> assumptions | Leucocytes can kill microbes extracellularly. <i>Vide</i> p. 9. |
| (7) The blood <i>in vitro</i> will not respond to inoculation | <i>A priori</i> assumptions | The blood does, when inoculated <i>in vitro</i> , exhibit increased bactericidal and phagocytic power, and does this often instantaneously; often in the course of a few minutes; and it would seem always in the course of two hours. <i>Vide</i> papers published by the Author*. |
| (8) The anti-bacterial substances which are elaborated in the body in response to bacterial vaccines operate only on the kind or sort of microbes which have been inoculated | <i>Vide</i> Immunization Curve No. 53 originally published in the <i>Lancet</i> , November 2, 1907, and republished in the Author's "Studies on Immunisation" (Chart 44) | <i>Vide</i> results of prophylactic inoculation against pneumonia in the Transvaal: Table XIII, p. 22; and non-specific clinical effects of intravenous inoculations of vaccines; p. 22. <i>Vide</i> also direct laboratory experiments — in particular Slide Cell Experiment p. 22, fig. 3. Shattock and Dudgeon's results p. 13, and Dr. R. Hare's results, Table IV, p. 13, also inoculations on blood <i>in vitro</i> . <i>Vide</i> papers referred to on this page.* |

* *Lancet*, March 29, 1919; February 24, 1923 (p. 365) and August 1, 1931 (p. 225); August 8 (p. 277); and August 15 (p. 333)

Clinical Section

President—G. DE BEC TURTLE, M.D.

[November 14, 1941]

Partial Thrombosis of the Right Internal Carotid at Level of Ophthalmic Artery, and Probably also a Thrombus in the Upper Dorsal Region of the Pons on Right Side.—
WILFRED HARRIS, M.D.

M. B., male, aged 61. Has been professional pugilist, and has served in the Canadian Mounted Police, and in the Navy. Wounded by a bullet, right anterior parietal bone, in 1929, resulting in left hemiplegia. Decompressed, and recovered. Wounded at Dunkirk in May 1940, and given prophylactic antitetanic serum. Three months ago wounded in left shin on board ship.

September 21, 1941: Admitted to St. Mary's Hospital with well-marked tetanus, generalized twitchings, trismus, risus sardonicus, and opisthotonos when stimulated. Given 191,000 A.T.S. units intrathecally and intravenously. Rapid recovery, and in ten days was allowed up for walks. In mid-October, when in a shop near the hospital, sudden momentary faintness occurred, left leg dragged, and there was mistiness of right vision, which has increased. Tremor of head noticed since recovery from tetanus. Two days after the faint, right pupil observed to be widely dilated. Right vision fingers only. Cerebrospinal fluid pressure 210.

On examination.—Weakness of left arm and leg, but no rigidity or increase of deep reflexes; left plantar reflex: flexor. Well-marked left hemianæsthesia below angle of jaw, and also over right trigeminal area. Right vision still fingers only; colour of right disc definitely paler than left, with contracted central artery. Blood-pressure 200/100.

Present condition.—The colour of the right disc has improved to normal, and the central artery is nearly equal to the left. The vision remains unaltered in the right eye, with a contracted visual field.

The prism test demonstrated that the right amblyopia was functional, the ascertained visual acuity of the right eye being nearly equal to the left.

The functional amblyopia is probably due to auto-suggestion following a temporary occlusion of the arteria centralis retinae.

JAN.—CLIN. I

Adie's Syndrome (Tonic Pupils).—HELEN DIMSDALE, M.R.C.P. (for WILFRED HARRIS, M.D.).

Female, married, aged 22, though feeling in perfect health, with no history of any illness beyond childish complaints, when applying in August 1941 for a post in an industrial firm in the suburbs, was refused by the doctor examining for the firm, who made the diagnosis of neurosyphilis.

On examination.—The pupils are unequal, the right pupil medium large, the left considerably smaller, resembling the Argyll-Robertson pupils in that neither reacts to light in the ordinary way, but, unlike the Argyll-Robertson pupil, contracting slowly to accommodation (tonic reaction).

After she has been in a dark room for half an hour, both pupils contract partially to a bright light. She states that she has known as long as she can remember that her pupils have been unequal. The ciliary muscles function normally, and the fundi and the optic discs are also normal. No ptosis.

The knee-jerks, Achilles and forearm-jerks are all absent. There is no ataxy, no Romberg sign. She denies having any pains in the limbs, and there is no sphincter disturbance.

The teeth are good, the upper central incisors being perfect. The blood Wassermann and Kahn tests were completely negative.

(Since the Meeting on November 14, the cerebro-spinal fluid has been examined and found normal in all respects. Wassermann reaction and Kahn test, cells, protein, and colloidal gold test negative.)

Hyperpiesis with Polycystic Kidneys treated by Sympathectomy and Adrenal Gland Reduction.—A. DICKSON WRIGHT, M.S.

Mrs. B. S., aged 40, first seen in February 1939, referred by Dr. Wilfred Harris, complaining of:

Increasing throbbing, right-sided, occipital headache for four months requiring morphia and other drugs for its relief. Vertigo for a similar period; this symptom together with the headache had kept her more or less constantly in bed. Dyspnoea and angina pains for eighteen months. Epistaxis for four years. Five-minute attacks of hemi-paræsthesia during last four months.

On examination.—A pale hyperpietic woman, prematurely grey. Blood-pressure averaged 220/100. Heart enlarged, with duplicated second sound. The blood-pressure fell in sleep to 140/100 and under deep pentothal anaesthesia to 158/110. Examination of renal functions showed them to be depressed, and the blood urea was sometimes as high as 70, but the urine contained no albumin or casts. The eye grounds showed some venous nipping, silver wire arteries and blurring of the disc edges.

At the first operation on the left side (11.5.39) the kidney was discovered to be polycystic and about twice the normal size. One cyst at the upper pole was as large as a billiard ball. All the splanchnic nerves were excised with half the celiac ganglion; the second and third lumbar ganglia were removed and half the suprarenal gland.

After operation the pressure fell to 145/100 and a week later rose to 165/110. The patient was so pleased with the improvement in her symptoms that she asked for the other side to be done and following this (15.6.39) the pressure became 105/65. The kidney on this side was polycystic like the other. Half the right suprarenal gland was removed.

On discharge on 3.7.39 the blood-pressure was 138 when standing and 154 when lying. Her headache was the last symptom to disappear, but now she is well and works in twenty-four hour shifts as a telephonist and has no hyperpietic symptoms two and a half years later.

COMMENT.—The possibility of the hyperpiesis being essential and not secondary to the polycystic kidneys is worth considering. The reason for showing this case is to draw

attention to the fact that if the kidney condition had been discovered before operation, sympathectomy would probably have been withheld, and so a very suitable case would have been left unaided. In the search for contra-indications it is possible to be too selective and cases which could be helped are sometimes left.

Dr. F. PARKES WEBER thought that the association of polycystic kidneys with hyperpiesia was only a coincidence, since extreme hyperpiesia was not usually a sign of polycystic kidneys until the terminal period, when operation would be hopeless.

Aneurysm of Circle of Willis.—A. DICKSON WRIGHT, M.S.

Mrs. M. S., aged 67. Since childhood has been troubled with migraines which were relieved by vomiting. They occurred only occasionally and were not localized.

About 1930 the headaches became more severe and were localized to the right frontal region "behind the eyes" and to the right face. Occasional vomiting.

May 1937: Attended hospital and was given "electrical treatment". During this treatment a right facial palsy developed which has persisted, and she also suffered occasionally from earache on the right side.

1939: Headache constant. Latterly she noticed the right eyelids opening "lazily".

6.10.41: Headache became almost unbearable, and this attack was preceded by vomiting. One week later the right eye closed. She occasionally felt dizzy during this last attack—had never felt so before.

About 14.10.41: Seen by Dr. Wilfred Harris who reported almost total 3rd nerve palsy, anosmia on right side, severe pain in first and second divisions of trigeminal nerve and slight reduction in sensation in the same area.

24.10.41: The right internal carotid was injected with thorotrast and an aneurysm the size and shape of a butter-bean was revealed. It was situated in the anterior part of the circle of Willis. The artery was immediately tied above the needle puncture just above the bifurcation of the common carotid.

After operation the foot of the bed was kept raised for a week, and the patient's position was very gradually altered until she sat up after three weeks. During this time ephedrine was administered (gr. $\frac{1}{2}$ t.d.s.) and bellergal, the first to raise blood-pressure and the second to prevent arteriospasm.

The pain has practically gone, and the 3rd nerve palsy is rapidly disappearing.

Ulcerative Colitis Complicated by Polyarthritis treated by Total Colectomy.—A. DICKSON WRIGHT, M.S.

A. H. S., male, aged 31, first seen on May 8, 1938, with symptoms of ulcerative colitis. The diagnosis was confirmed by sigmoidoscopy and an appendicostomy was done. Yatren instillations resulted in great improvement, and the colitis settled down to a chronic state; X-rays showed stricture formation. Pyrexial arthritis developed and the large joints of the body and the spine were involved, the knees and the wrists being most affected. After consultation it was decided to remove the colon as no other focus of infection could be found.

This was done in one stage and the ileum was anastomosed to the recto-sigmoid junction; although without a colon, he now has one regular formed action every day. There are occasional attacks of proctitis with a little bleeding and mucus formation. The patient is much mutilated by the arthritis, but the disease has been quiescent since the colectomy and he is now having joint adhesions broken down under anaesthesia. He

recently had a laparotomy for a strangulation of the small intestine by a band, and this he negotiated satisfactorily.

Constrictive Pericarditis.—G. W. PICKERING, F.R.C.P.

Mrs. F. F., aged 46 years. Occupation: Home duties.

Past history.—Left pleurisy with effusion at 14 years of age; "rheumatic pains" in ankles and left shoulder for many years. No rheumatic fever, scarlet fever, tonsillitis or tuberculosis. Two miscarriages, 1921-22.

History: 1926: Complained of breathlessness and palpitations when walking on the level, but no swelling of ankles. Symptoms disappeared after three weeks' rest in bed.

1937: Again complained of breathlessness on slight exertion, and of a "puffed-out feeling" in epigastrium, but still no swelling of ankles. In hospital for three months and given digitalis. From then until November 1940 her only symptoms were breathlessness going upstairs and fullness after meals.

November 1940: Noticed that her navel was pushed out. This was relieved by rest at home.

February 1941: Admitted to hospital; abdomen tapped; given digitalis for three months and then discharged. May and June 1941: Given intravenous salyrgan 2 c.c. twice weekly, but with little effect. July 14, 1941: Seen in Out-patients' Department complaining of breathlessness and swollen abdomen. She had walked to hospital and was breathless but not grossly so.

On examination.—Gross ascites prevented the liver being felt; neck veins engorged to 6 in. above angle of Louis, but collapsing for brief moment in each cardiac cycle; forcible impulse in 5th left space 5 in. from mid-line, and pulsation in 4th left space just internal to impulse, and in 3rd left space near sternum. All these pulsations appeared to be systolic retraction. The right heart border was $2\frac{1}{4}$ in. from mid-line. No dullness in 2nd space, but dullness at inner end of 3rd left space. Heart rhythm regular. The second sound was reduplicated at the impulse and at the second left cartilage. Blood-pressure 108/90. No abnormal physical signs in lungs or central nervous system.

July 21, 1941: Admitted as in-patient. Ascites tapped on July 24 and 12 pints removed. This enabled the liver to be felt to 3 in. below the costal margin in the mammary line. It was firm and pulsating.

Throughout August venous pressure was 12-15 cm. above angle of Louis. Heart-rate 60-70. Fluid intake restricted to 30 oz.; daily urine output 10-20 oz. Tincture digitalis 60 m per day for four days, then 45 m for four days. It produced slowing to 36 beats per minute without diuresis, and was then stopped. Ascites tapped on August 30 (20 pints) and September 6.

Investigations.—X-rays, 26.7.41: "No evidence of pericardial calcification. Appreciable degree of ventricular hypertrophy apparent; vigorous hypertrophic type of pulsation of left ventricular origin." Blood urea, 29.7.41: 34 mg. per 100 c.c. Wassermann reaction negative. Urine: Sp. Gr. 1020-1024; always slight cloud albumin; occasional hyaline cast present. Blood, 29.7.41: Hb. 98%; C.I. 1.0. Electrocardiogram: Auricular fibrillation; low potential all leads. Vital capacity: 1,250 c.c.

Operation, September 8, 1941, under cyclopropane anaesthesia, in semi-recumbent position (Mr. A. Dickson Wright).

Mr. A. Dickson Wright: The operation was not especially difficult. A large pectoral flap was turned back and three chondral cartilages and the inner end of the ribs removed. There was no safe space between the pleural cavities so the left pleural cavity was opened. The heart was firmly encapsuled with fibrous pericardium and the two layers firmly fused together were dissected piecemeal from the surface of the heart. The right ventricle was slightly torn and had to be stitched. At the completion of the operation the heart seemed to be expanding freely in diastole. The convalescence was

straightforward and seemed to be greatly helped by a special oxygen mask, recommended by the anaesthetist, Dr. L. H. Morris.¹

Post-operatively: A left pleural effusion developed to the level of the lower angle of the scapula. This slowly absorbed and had gone by October 17. Venous pressure 9 cm. four days after operation, and by October 17 it was varying from 5 to 7 cm. Abdomen tapped on September 11 (4½ pints) and on September 25 (10½ pints) since when it has remained undistended.

Post-operative electrocardiogram: Auricular fibrillation; greater potential in all leads than before.

Discharged 18.10.41. Patient now on 1 pint per day fluid intake; she has mersalyl 2 c.c. twice weekly. Urine output after mersalyl 70-90 oz. and about 20 oz. on intervening days. Now walks half a mile without dyspnoea.

¹ This mask was described by Barach and Eckman in *Anæsthesiology*, 2, No. 4, 421. In appearance it is similar to the B.L.B. mask, except that the respiratory bag is larger, and its capacity one gallon. By means of an injector attached to the oxygen regulator, the percentage of oxygen in the inspired air delivered to the patient is constant, irrespective of the rate of flow to the mask. The oxygen concentration can be varied between 40 to 100%, in ordinary use 40 to 60% is sufficient for the needs of most patients. No rebreathing occurs, thus the carbon dioxide percentage does not rise above 0.2 and no increased pressures are developed in the mask during respiration. This is obtained by the insertion of an inspiratory valve between the respiratory bag and the nose mask and a light expiratory valve placed on the mask opposite the nose. Control of the apparatus is simple, the rate of flow of the mixture is set so that the respiratory bag remains comfortably full, a flowmeter is not required. It is economical in use, a large 100 ft. oxygen cylinder lasts about sixteen hours. Drying up of the oral, nasal and pharyngeal mucous membranes, commonly met with in prolonged oxygen therapy, does not seem to occur, this is, no doubt, due to the fact that the injector draws in atmospheric air.

The mask was in continual use for eight days following the operation and gave definite relief to the patient. It is of interest to note that on the second night a delay occurred in changing the oxygen cylinders and as the patient became slightly delirious, a B.L.B. mask was substituted. This she did not tolerate for long, the original mask was restored to her and the anoxæmia was relieved.

This mask has three advantages over the B.L.B. mask: (1) The delivery of a constant proportion of oxygen and air irrespective of the rate of flow. (2) No increased pressures are developed during respiration. (3) No rebreathing and consequently no building up of carbon dioxide.

The mask was kindly lent by the Medical and Industrial Equipment Co., 12, New Cavendish Street, W.1.

Pulmonary Hypertension with Pulmonary Regurgitation Possibly due to Interatrial Septal Defect.—W. D. W. BROOKS, D.M., F.R.C.P.

Mrs. T. G., Jewess, aged 40.

Family history not significant.

Personal history.—Hæmoptysis (varying in amount up to ½ pint) on five occasions since January 1939. Slight dyspnoea on exertion recently. Apart from occasional colds in the winter, she has had no illness of note.

Physical signs.—A well-built, rather stout woman, dyspnoeic on slight exertion. Cyanosed; no clubbing of fingers and no evidence of congestive heart failure. Blood pressure, 125/80. The heart, however, is enlarged, beats regularly, and presents the clinical signs of pulmonary regurgitation. Râles are occasionally audible at the bases of the lungs. Nothing else abnormal.

Investigations.—Blood-count: R.B.C. 7,000,000; Hb. 131%; C.I. 0.93; W.B.C. 9,800. Sedimentation rate: 3 mm. (Westergren). Sputum: Tubercle bacilli absent (3 occasions); no significant infection. Urine: No abnormality found.

X-ray examination.—Lungs: Generalized gross vascular congestion. Heart: Increased in size due to enlargement of both ventricles; conus and pulmonary artery considerably

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Section of Orthopædics

President—C. LAMBRINUDI, F.R.C.S.

[October 11, 1941]

MEETING HELD AT HORTON HOSPITAL

The Foot Problem in Service Cases

By H. L.-C. WOOD, M.S., F.R.C.S.

INTRODUCTION

DURING the last eighteen months we have seen a great number of foot cases referred for advice. We have tried to obtain the view-point of the Battalion Medical Officer by maintaining close touch through letters, interviews at the time of consultation, and by visits of my colleagues and myself to local units and training establishments. It has been our aim, by personal discussion, to see how best we can help.

Whereas in civil practice a major surgical procedure may be indicated for the relief of a certain foot condition, in circumstances of conscript military service the surgical condition and its treatment is largely subsidiary to the co-operation and willingness of the patient to become an efficient soldier. In other words, in an unwilling patient surgery may do more harm than good. For this reason I shall say little about the relief of major disabilities.

TYPES OF DISABILITY

In the terminology of the Medical Board Category Sheets, the foot troubles we encounter fall into three main groups:

- (1) *Severe*.—Not remediable by operation to the extent of making an efficient soldier.
- (a) Severe hallux rigidus and hallux valgus.
- (b) Rigid flat feet.
- (c) Severe claw foot.

I know that in a modern army men in this group can do useful work of a sedentary nature, but it is only recently that Medical Boards have been retaining their services for this purpose. We have been rightly advised not to operate on these conditions, but to submit the men for regrading or discharge, and our experience confirms this ruling.

- (2) *Moderate disability*.—Remediable by operation.
- (a) Hammer toe.
- (b) Removal of exostoses, including the slicing operation for early hallux valgus.
- (c) Amputation of fifth toes.
- (d) Manipulations.

The only observation I would make upon this group is that when an arthrodesis for

enlarged. Electrocardiogram: Slight right axis deviation, curves otherwise within normal limits.

Dr. PARKES WEBER said he thought that the pulmonary regurgitation was probably due to atheromatous disease of the pulmonary valve, which occasionally occurred when the valve was the site of a slight congenital (developmental) abnormality, for instance, two of the cusps being congenitally more or less fused together, the valve thus being a *lucus minoris resistencie*.

Scleroderma and Dermatomyositis.—C. G. BARNES, M.D.

Man, aged 66. Tongue became swollen and hard in October 1940 after dental extractions, and scleroderma of the lips and cheeks developed. Legs became oedematous and painful, and dermatomyositis spread, involving the whole body except the chest. All the affected muscles were firm and tender, with contractures in the limbs. Sclerodactyly of hands without Raynaud's phenomenon or pigmentation. Afebrile, and no cardiac or renal lesion.

Oral aperture now becoming smaller, and face losing expression. Arms and hands greatly improved after treatment with thyroid extract and salyrgan, but trunk and limbs still greatly oedematous, and abdominal muscles board-like. Tongue very hard and almost immovable.

Investigations.—Urine, renal function, blood-count and cardiogram all normal. Wassermann reaction negative. No creatinuria. Basal metabolic rate: -14% . Plasma proteins 5.8 g./100 c.c. Albumin 4.0%, globulin 1.8 g.%.

Glucose tolerance curve: fasting 90 mg., $\frac{1}{2}$ hr. 130 mg., 1 hr. 148 mg., 2 hr. 140 mg. per 100 c.c. after 50 g. of glucose.

[Sections of muscle removed at biopsy, and radiograms of soft tissues were shown.]

Dr. PARKES WEBER thought that the case was an extreme example of one type of dermatomyositis, namely, the type formerly known as the generalized symmetrical ("hypertrophic") form of scleroderma. He knew of one case in which the general "puffy" appearance was at first so marked as superficially to resemble renal oedema.

DEMONSTRATION

Dr. J. Skládal (Prague) demonstrated a case showing expiratory reduplication sound which arises on auscultation when the author uses his method of a sudden expiration instead of provoked coughing (See "Pleuro-subpleural Zone", by J. Skládal, Cambridge University Press, 1942.) This has been found clinically to be a simple test for subpleural consolidation.

to the clinic, and only returned to the cobbler for completion, after the fitting has been checked. Owing to the hard wear to which insoles are subjected, it is desirable to provide two pairs. The work is done by a cobbler, an ex-patient who was the victim of an air raid. It is cheap—for example, an insole with sponge-rubber metatarsal bar can be made for about ninepence.

Arrangements exist for similar clinics to be held at some of the outlying Sector hospitals, but at present the men have to attend here for the cobbling to be done. We hope to make some provision for this at these outlying hospitals, in order to save time and transport.

Follow-up.—The idea of a modern soldier going into battle wearing a valgus insole or a metatarsal bar, may seem ludicrous. When possible, we have followed up our cases and have established the fact that these men do get real benefit, and can return to full duty, often without need for lowering of category. There is usually a period of bitter complaint for ten days or a fortnight, but if the Unit Medical Officer will encourage the man to continue with his appliance it is found that he soon becomes comfortable and carries on.

Two practical points have emerged:

(1) The football bar or rocking bar used for hallux rigidus tends to wear out rapidly; consequently an inside sponge rubber bar is preferable. Studding the football bar with nails might be an alternative solution.

(2) The recommendation of civilian-type boots is seldom necessary. The army-type boot is excellent and with minor adjustments can be made comfortable. We find civilian shoes worn frequently, but their type and shape are usually unsuitable, and, also, it is against the Regulations.

Regrading for minor foot disabilities.—To lower a man's category is demoralizing. So-called "light" work in the Army would be heavy work to some of us, and may consist of fatigue duties, or prolonged standing in a cook-house. The disability remains unremedied and unrelieved. Hence we avoid regrading where possible.

SUMMARY

We wish to build up an organization in the Sector which will give a rapid and efficient service to Unit Medical Officers stationed in the locality of our outlying hospitals. This service to consist of:

(1) Advice. (2) The provision of simple appliances. (3) Remedial treatment under the direction of competent physiotherapists. To this end, the establishment of "local" foot clinics, complete with a chiropodist, cobbler, and physiotherapist is desirable.

In addition, a rapid means of obtaining boot refits must be worked out in connection with the Army authorities. For instance, at the large centres a small stock of boots might be held in charge of the Military Registrar. We hope shortly to have such a stock.

We believe that with a team consisting of surgeon, physiotherapist, chiropodist and cobbler, working on a clinic system of the type outlined, we can avoid the hospitalization of many men and at the same time give efficient treatment. The decentralization of the clinics will save time in travelling and will lift the load from the main orthopædic centres which must continue to treat the cases of major disability.

Finally it appears that we must initiate the scheme ourselves and, when once it is in being, hope that it will receive the support of E.M.S. and military authorities.

Psychological Reactions to Injury

By LOUIS MINSKI, M.D., M.R.C.P., D.P.M.

MANY members of the medical profession are rarely able to visualize all the needs of the individual whom they are called upon to treat. Probably the commonest example of this failing is seen in the case of the physically disabled. The deformity is in itself so obvious that the surgeon, nurses, and friends may direct all their attention and energies towards its alleviation and the question of mental hygiene is neglected. The result is that morbid psychological factors are introduced because of over-emphasis on the purely physical problem. This is sometimes seen in the case of children who are admitted to

hammer toe is done, the man should be hospitalized and the toe splinted for not less than six weeks. I have seen several who were not, with disastrous results to the arthrodesis.

(3) *Minor disabilities*.—Remediable without operation. This is the most important group and it includes:

- (a) *Metatarsalgia* with flat anterior arch. (Most common of all.)
- (b) Corns, callosities and verrucae.
- (c) Acute foot strain.
- (d) Early degrees of flat foot.
- (e) Moderate hallux valgus.
- (f) Short tendo Achillis.
- (g) Mild claw toe.
- (h) That most intractable condition, epidermophytosis pedum.

This last is common, and is the source of much minor discomfort, but, although difficult to cure, it should be dealt with efficiently by the Battalion Medical Officer.

Perhaps I should add to the lengthy list the "mauling foot".

MINOR DISABILITIES

The first problem to tackle is how best these men can be treated.

This hospital is primarily a consultation centre to which men are sent for *advice*, and in some cases a note to the medical officer is all that is needed. It often happens that the medical officer does not want advice; the diagnosis is obvious; what he wants is *treatment for his patient*. It is not because he is incapable of carrying this out himself, but because no facilities exist for so doing. This is confirmed in conversation; nor do adequate facilities exist at the subsidiary E.M.S. hospitals of this Sector. For example, the only mechanism for obtaining an insole is to order one as a "surgical appliance," through the routine military channels. The same applies in a lesser degree when a boot is found to be too small and a refit is necessary.

I am sure that it is wrong to admit a man to hospital if out-patient treatment is possible. Once he is admitted, the hospital complex develops and cure is uncertain.

A solution has been found at Horton by the institution of an out-patient "foot clinic"

The working is as follows:

- (1) General out-patient consultations are held twice weekly for all types of case.
- (2) Foot cases are sorted into two groups: (a) Those needing *in-patient* treatment: (i) Remedial exercises; (ii) manipulation; (iii) operation. (b) Those who can be treated as out-patients.

(a) In particular the cases of acute foot strain benefit from a *short* period in hospital, but this should not exceed three weeks or the man will be unable to return to his unit, because of the operation of the "Y" list. This is undesirable and should be avoided, as it leads to convalescent depôts, and psychological complications.

(i) In regard to *remedial exercises*, there is no doubt that if these could be carried out under the supervision of the Unit Medical Officer, or at a nearby hospital, it would be ideal; but it is seldom possible to arrange this, and a short stay in hospital is the next best thing. While we aim at rapid restoration to normal, a further difficulty arises in that the man may have to wait for discharge after treatment is ended; and during this time his morale deteriorates and other disabilities appear.

The indications for operation and manipulation I have already touched upon.

(b) In the *out-patient group* are men with metatarsalgia, corns, flat foot, short tendo Achillis, &c., in other words, those men who can be benefited by *simple* expedients, such as the alteration of the size of boots, the provision of heel wedges, insoles, and metatarsal bars, or the attention of a chiropodist. These men are sent direct from the out-patient department to the "foot clinic".

Here, a routine method is adopted.

(i) The feet are measured in the weight-bearing position and the measurement checked against the size of boot.

(ii) Socks are inspected; the *shrunk* sock is apt to be forgotten as a cause of claw toes.

(iii) If wedges or insoles are needed, they can be made and fitted the same day.

The fitting of insoles and metatarsal bars must be personally checked by the surgeon, and adjustments made at the time. To facilitate this, the partly finished product is sent

not to include this type of case, about which there is some doubt as to the relationship between the physical injury and psychological reaction, but to study those patients who are apparently normal before the accident producing the physical disability and who develop abnormal mental attitudes afterwards. Many such cases of disability and deformity are seen nowadays as a result of war injuries and among the commonest are loss of limbs, blindness and facial disfigurement.

It is not proposed to discuss the psychological disturbances occurring as a result of sepsis, exhaustion, &c., which may take the form of delirious states and toxic confusional reactions, and are not psychological reactions to the physical deformities.

The first adjustment which loss of a limb entails is to that of the phantom limb. This may be regarded as an hallucination occurring in persons with a normal mental setting. Although the limb appears to be very real, the patient has insight into the fact that it is illusory in nature. By subjective perception the limb is perceived as a real one, while by objective perception this impression is corrected mainly as a result of visual impressions. The peripheral theory for the phantom limb assumes that the sensation originates from excitation of nerve ends in the scar and anæsthetization of the stump has abolished the phantom which returns when the anæsthesia has passed away. The cerebral theory maintains that the limb is still represented in the total body image. Head showed that the phantom limb disappeared after injuries of the posterior central convolution and parietal region of the brain. Phantoms are missing in congenital absence of limbs and this is thought to be due to the fact that they have never been represented in the body image. Again, the fact that the arm is much less marked in phantom limbs than the hand may be due to the fact that central representation of the hand is much more extensive than the arm.

Phantom limbs which are not always present may appear immediately after amputation or not for some time. The limb usually feels the same size as the normal one while feelings of irritation and itching and alterations in temperature are not uncommon. For instance, one patient with an amputation through the lower third of the thigh complained of continuous itching below the heel and a feeling as if hot water were being poured down the leg. Pains and cramps in the phantom are not uncommon, the patient often complaining that the toes or fingers are bent in uncomfortable positions. A characteristic of the pain appears to be that it is most marked and persistent where the patient has had a painful wound or scar before amputation. For instance, a patient who had had a wound of the knee with subsequent amputation of the leg felt this painful wound for some time, while another patient whose leg was amputated for senile gangrene was kept awake at nights by a pain in the big toe which had been present before amputation. A woman whose eye had been removed some years before because of severe corneal ulceration which had been extremely painful, developed a painful phantom eye which persisted for some time. On the other hand, those patients who are, like many air-raid casualties, severely shocked at the time they have been injured and have not had to endure for some considerable time the pain of chronic ulceration, gangrene, &c., do not appear to be troubled so much by pain in the phantom limb which, however, is still definitely present. Again, some patients only have phantom limbs when they have pains in them. When an artificial limb is fitted this may be identified with the phantom and may become animated although the latter may be felt to be independent of the position of the body. Adjustment to the phantom limb takes place fairly rapidly, although the pains and cramps in it may give rise to periods of insomnia as they are usually more marked at night. In some cases the insomnia and resulting fatigue tend to produce mild temporary anxiety states.

When the patient is ready for discharge from hospital he now has to adjust himself to his new circumstances. The degree of adjustment which the patient makes depends on a number of factors. The younger the person, as a rule, the better the adjustment, provided that, as previously mentioned, the environment is favourable and the patient is correctly handled. It is found that after the prime of life adaptability is much more difficult although even in younger persons other factors must be taken into consideration. For instance, in the case of a young, attractive, unmarried girl the loss of a leg or facial disfigurement may cause a much more difficult task in adjusting than the loss of an arm, whereas the loss of an arm in the breadwinner of a family who is a manual labourer would be more difficult to adjust than the loss of an eye or severe facial disfigurement. Here again, it is obvious that rehabilitation for the latter in the form of vocational

hospital because of some physical injury which is likely to take long to cure, such as fractures as a result of air raids. Because of lack of separate children's wards they are admitted to wards with adult patients, where they are not unnaturally pampered and fussed by patients and staff, and, in fact, spoiled. At the end of some months, when they are fit for discharge, they return to a poor home where they are no longer the centre of attraction, with the result that many of them become difficult and resentful of their surroundings and develop behaviour disorders which are often extremely difficult to eradicate. It will be seen, therefore, that even among children who are not permanently crippled or deformed abnormal psychological reactions may readily occur, while among those permanently disabled "crippled personalities" may be even more easily formed. Many of them show behaviour disorders characterized by aggressiveness, temper tantrums, day-dreaming, stealing, &c., and one of the most important causal factors in symptomatic behaviour in handicapped children is the attitude of the parents towards the child and his disability. Over-protective parents tend to be more protective while an athletic father may show his disappointment in a crippled child by making disparaging remarks to him and increasing the child's feeling of inadequacy. Again, such children often do not enjoy the privileges of other members of the family because "they are too much trouble". The over-protective attitude tends to create "the crippled personality" by making the child more dependent and infantile and totally incapable of living his life as a self-maintaining member of society. Not only parents but teachers, nurses, doctors and all with whom he comes in contact must bear their share of responsibility. The tendency to be sentimental and pity the child may also be associated with a feeling of repulsion for the handicap.

Among children of his own age, however, the crippled child may find greatest unhappiness, as he is often friendless and an object of ridicule. This results in feelings of inferiority and shame with consequent aggressiveness and resentment, or a regression to a more childish level. Over-compensation for these inferiority feelings may often be seen in boasting and attitudes of superiority. All handicapped children, however, are not maladjusted; in fact, many of them show no psychological disturbances at all. Lesions which are acquired early or are congenital are usually well compensated physically, while psychological adjustment is also easier in childhood.

These children have been wisely handled by understanding parents, a tolerant teacher and a well-trained social worker. Something constructive has been offered to compensate for physical handicaps. This may be in the form of adequate recreational facilities and occupational therapy which must be something more than an aid to physiotherapy. It must interest the patient, develop pride in accomplishment and give a feeling of independence. By such means feelings of inferiority and inadequacy, of self-pity and resentment against environment will tend to disappear.

Lesions of the central nervous system such as birth trauma, encephalitis, &c., which produce physical disability often cause at the same time a disturbance of personality which is not a psychological reaction to the physical disablement at all. The personality disturbance and probable intellectual impairment are the direct results of the cerebral lesion which has caused the physical disability. In the same way, in the adult who suffers a cerebral injury or vascular accident, emotional and intellectual disturbances often occur at the same time as the physical symptoms. Patients with partial cerebral lesions may be much more distressed than those with complete ones. In the latter, insight is often lacking and intellectual impairment amounting to dementia may be present. For instance a patient with hemianopia may be very distressed by his disability, whereas a patient with a complete central blindness may be quite unaffected by his loss. Most of these patients, both children and adults, not only require the services of an orthopaedic surgeon to attend to the physical deformity but also those of a neuro-psychiatrist.

Among adults there is the group of patients who have lost a limb as a result of an accident which relatives regard as the causal factor in a consequent mental illness. For example, a girl of 25 was said to have fallen under a tube train while trying to enter it and she sustained a laceration of the scalp together with a severed leg. She was an attractive girl, intelligent and artistic, and when convalescent from the physical illness she began to be depressed, said people were staring at her and talking about her. Since that time, viz., two years ago, she has shown signs of an insidiously developing schizophrenia and it is not at all unlikely that the psychotic illness had started before the so-called accident which may have been in the nature of a suicidal attempt. It is wise

not to include this type of case, about which there is some doubt as to the relationship between the physical injury and psychological reaction, but to study those patients who are apparently normal before the accident producing the physical disability and who develop abnormal mental attitudes afterwards. Many such cases of disability and deformity are seen nowadays as a result of war injuries and among the commonest are loss of limbs, blindness and facial disfigurement.

It is not proposed to discuss the psychological disturbances occurring as a result of sepsis, exhaustion, &c., which may take the form of delirious states and toxic confusional reactions, and are not psychological reactions to the physical deformities.

The first adjustment which loss of a limb entails is to that of the phantom limb. This may be regarded as an hallucination occurring in persons with a normal mental setting. Although the limb appears to be very real, the patient has insight into the fact that it is illusory in nature. By subjective perception the limb is perceived as a real one, while by objective perception this impression is corrected mainly as a result of visual impressions. The peripheral theory for the phantom limb assumes that the sensation originates from excitation of nerve ends in the scar and anæsthetization of the stump has abolished the phantom which returns when the anæsthesia has passed away. The cerebral theory maintains that the limb is still represented in the total body image. Head showed that the phantom limb disappeared after injuries of the posterior central convolution and parietal region of the brain. Phantoms are missing in congenital absence of limbs and this is thought to be due to the fact that they have never been represented in the body image. Again, the fact that the arm is much less marked in phantom limbs than the hand may be due to the fact that central representation of the hand is much more extensive than the arm.

Phantom limbs which are not always present may appear immediately after amputation or not for some time. The limb usually feels the same size as the normal one while feelings of irritation and itching and alterations in temperature are not uncommon. For instance, one patient with an amputation through the lower third of the thigh complained of continuous itching below the heel and a feeling as if hot water were being poured down the leg. Pains and cramps in the phantom are not uncommon, the patient often complaining that the toes or fingers are bent in uncomfortable positions. A characteristic of the pain appears to be that it is most marked and persistent where the patient has had a painful wound or scar before amputation. For instance, a patient who had had a wound of the knee with subsequent amputation of the leg felt this painful wound for some time, while another patient whose leg was amputated for senile gangrene was kept awake at nights by a pain in the big toe which had been present before amputation. A woman whose eye had been removed some years before because of severe corneal ulceration which had been extremely painful, developed a painful phantom eye which persisted for some time. On the other hand, those patients who are, like many air-raid casualties, severely shocked at the time they have been injured and have not had to endure for some considerable time the pain of chronic ulceration, gangrene, &c., do not appear to be troubled so much by pain in the phantom limb which, however, is still definitely present. Again, some patients only have phantom limbs when they have pains in them. When an artificial limb is fitted this may be identified with the phantom and may become animated although the latter may be felt to be independent of the position of the body. Adjustment to the phantom limb takes place fairly rapidly, although the pains and cramps in it may give rise to periods of insomnia as they are usually more marked at night. In some cases the insomnia and resulting fatigue tend to produce mild temporary anxiety states.

When the patient is ready for discharge from hospital he now has to adjust himself to his new circumstances. The degree of adjustment which the patient makes depends on a number of factors. The younger the person, as a rule, the better the adjustment, provided that, as previously mentioned, the environment is favourable and the patient is correctly handled. It is found that after the prime of life adaptability is much more difficult although even in younger persons other factors must be taken into consideration. For instance, in the case of a young, attractive, unmarried girl the loss of a leg or facial disfigurement may cause a much more difficult task in adjusting than the loss of an arm, whereas the loss of an arm in the breadwinner of a family who is a manual labourer would be more difficult to adjust than the loss of an eye or severe facial disfigurement. Here again, it is obvious that rehabilitation for the latter in the form of vocational

training for some other form of employment and psychological guidance, and useful creative employment for the former will help to make the mental adjustment more easy.

The previous personality of the patient is also an important factor in the psychological attitude towards an injury. Those who previously showed evidence of neurotic traits are more likely to show abnormal reactions than the stable type of individual. This is well typified in the case of a soldier of 26 who was admitted on 22.6.40 with a compound comminuted fracture of the left tibia and fibula complicated by gas gangrene. The left leg was amputated below the knee. He was quite cheerful while in hospital, made a good recovery, and was fitted for an artificial limb on 16.4.41. He is now at home and doing his previous work of accountancy with the same firm. He is morose, depressed, and apathetic and makes little attempt to interest himself in things generally. It appears that he has always been spoiled by his mother at home, that he is selfish and self-centred and now his mother is very over-sympathetic and over-protective, and she is always giving in to him. This is just the type of environment to produce such a state of despondency in a spoiled and pampered individual who obviously requires a firm but kindly attitude to remove the feelings of dependence and hopelessness.

The comparatively few amputation cases treated at this hospital have made good adjustments and are in employment or doing their housework, apart from one who passed through a severe phase of depression from which he made a good recovery, and the other case already mentioned. None of them show any feelings of resentment, unless sympathetic strangers try to help them, while only one shows any signs of self pity.

It is not only patients with total or partial amputations who are liable to develop abnormal psychological reactions; very often severe or even minor injuries may be opportunities for patients to perpetuate symptoms for some unconscious motive and for which there is no longer any organic basis. This is seen especially in war-time where Service cases who have been wounded continue to have painful scars or stiff joints long after physical factors have ceased to play a part. Very often this is the result of a hysterical prolongation or fixation of symptoms, the motive for the symptoms being that they keep the patient in hospital or at any rate out of active service. These neurotic reactions resemble the compensation neuroses of peace-time in that the symptoms may improve or disappear when the ends are achieved and an adequate pension has been provided. Hysterical exaggeration of symptoms may also occur even when the physical factor is still operative, all these hysterical symptoms arising out of the patient's previous personality; the number of such cases, however, is comparatively few. Psychotherapy in the form of persuasion, suggestion or hypnosis should be started immediately as the longer the symptoms are allowed to develop the more intractable do they become and the more difficult are they to remove.

These symptoms are not consciously produced and the patient does not realize that they are functional in origin. In fact were such symptoms conscious in origin they would express a deliberate intention on the part of the patient to achieve some motive which would help his ends, such a condition being malingering. Often conscious and unconscious motives are combined, and the malingerer may easily persuade himself of the reality of his symptoms.

The following case illustrates the neurotic reaction to a physical injury and at the same time partially reveals the underlying psychopathology. A married woman, aged 34, was admitted to hospital on 24.11.40. She was travelling in a bus with a girl friend during an air raid when a bomb fell near. She felt a severe pain in the left arm and also her friend lying on her, and on looking round she saw the battered blood-stained face of her friend who was dead. She sustained a comminuted fracture of the left ulna and a punctured wound of the neck. The arm was cleaned, the fracture set, and the limb put up in plaster. Union and alignment were good, and when the plaster was removed massage and remedial exercises were ordered. The patient refused to co-operate, became aggressive towards the masseuse and yelled with pain. It was noticed that when attempts were made to move the arm the antagonists contracted at the same time and went into spasm. Manipulation was attempted on 29.3.41 with little result. The arm was abnormally wasted, but no neurological signs were present. She was seen by a psychiatrist as it was considered that a functional element was present. Hysterical contracture was diagnosed and she was given intravenous sodium amytal in an attempt to relieve this and also to make her more receptive of suggestions to aid movements. This was unsuccessful, and

when an attempt was made to straighten the arm she called the doctor a brute, screamed, and wept copiously. As it was considered that there might be some underlying organic cause also, her arm was again manipulated on 29.4.41 when many adhesions in both the shoulder and elbow joints were broken down. She was again given amytal and on this occasion movements of the arm took place. She was given therapeutic talks and left the hospital improved on 11.5.41.

The relevant psychiatric history was as follows: The mother had a withered arm which resulted in her obtaining more sympathy from her husband than she would otherwise have done. The patient had previously had a good personality with no neurotic traits but her husband was a bully and went with other women. He was only kind to her when she was ill and he gave the sister of the ward the impression that he was not anxious to have her home. In addition the patient's children had been evacuated and although she wanted to join them there she was afraid to leave her husband as it would allow him more liberty with other women. There was also the suggestion that the friend who was travelling in the bus with her and who was killed was one of her husband's girl friends. The unconscious motivation for the hysterical illness therefore appeared to arise from (a) the fact that her husband was only kind when she was ill, (b) guilt feelings associated with the death of her friend owing to its probably being associated with an unconscious wish for this to occur as a result of her husband's relationship with her, (c) the conflict over wishing to join her children and (d) the mother having a withered arm which served as an example to her.

This case demonstrates very well the mixture of neurotic and organic symptoms, together with the underlying psychopathology. It should be emphasized that when a physical injury is not making satisfactory progress and physical signs to account for the condition are absent, or when it is definitely suspected that there is a functional component, the services of a psychiatrist should be called upon at once in order that any underlying factors for such a neurotic superstructure may be exposed and dealt with.

It cannot be too firmly stated that a patient may give an impression of having made a good adjustment while in hospital, but after being at home for some time, as in the case of the patient with the amputated leg, psychological difficulties may occur. A wise precaution would be to have all such patients seen by a psychiatrist while in hospital to ensure that there are no personality difficulties, &c., and also to have a social report on the environment to which he is going in order that unsuitable influences can be removed or another more suitable environment found.

I have recently had an opportunity of seeing ex-Service men disabled in the last war. Many of these men have been leading invalid lives for twenty-five years as a result of fractured spines, &c., and the outstanding characteristics of their mental state are resignation and apathy, self-centredness and a tendency to talk about themselves indefinitely. There is however a definite *esprit de corps* and feeling of comradeship, they are willing to help each other and the home whenever they are able, they will do handicrafts if encouraged, but if *ordered* to do anything or strict discipline is enforced they become difficult and obstructive.

Men wounded in this war likely to be permanently disabled are already being sent to such homes and hospitals. This is a great psychological error as these patients quickly realize that in another twenty years they will probably be like the others and they rapidly become depressed, refuse to stay, and are often extremely difficult to manage. Separate homes when the number warrants it should be provided, and until such time arises, beds in other hospitals could be allotted where they would receive psychological encouragement by the admission and discharge of the other patients.

Conditions of the Back Simulating Visceral Disease

By J. H. KELLGREN, F.R.C.S.

FROM the experimental studies on pain carried out by Sir Thomas Lewis and others, we now know that the deep-lying somatic and visceral structures give rise to the same type of pain which is poorly localized and referred over a common segmental pattern. The superficial and peripheral structures give rise to local pain, while from intermediate structures the pain presents every gradation from the full segmental to the purely local type of distribution.

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The structures of the back illustrate this general rule very well. The important supraspinous ligament together with the lumbodorsal fascia give rise to local pain. The deep-lying structures surrounding the vertebrae give rise to pain of full segmental distribution, while the intermediate mass of muscle composing the erector spinae gives rise to pain of

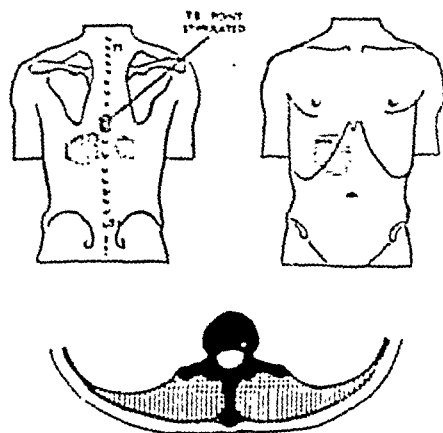


Figure illustrates the distribution of pain arising from structures in the back at the level of the 8th thoracic vertebra. The supraspinous ligament and lumbodorsal fascia give local pain (dots). The deep-lying structures surrounding the vertebrae give rise to pain of full segmental distribution (horizontal hatching). The intermediate mass of muscle comprising erector spinae gives pain of modified segmental distribution (vertical hatching).

modified segmental distribution. Besides this variation in pain distribution there is also a variation in the sensitivity of these structures. Thus the supraspinous ligament is intensely sensitive; the structures surrounding the neural arch are somewhat less so, and the vertebral bodies and discs are relatively insensitive. From this it is clear that disease of the insensitive vertebral bodies rarely produces pain unless there is a secondary disturbance of the sensitive neural arch.

Apart from these considerations the clinical picture of spinal disease is determined by the distribution of the segmental pain areas at different levels. Thus from T.11 to L.2, the segmental areas have a very large posterior component, so that backache is the presenting symptom in both spinal and visceral disease at this level.

In the mid-thoracic region the anterior component is by far the greater, so that pain in the chest or abdomen is paramount and such patients are usually investigated for visceral disease to the exclusion of the back; it is this type of patient that provides such diagnostic difficulties.

This is not surprising when we remember that not only is the character and the distribution of the pain identical in both visceral and spinal disease, but pain arising in the back may be associated with the appropriate visceral symptoms and signs; for instance, substernal pain simulating angina may be associated with shortness of breath and palpitations, and the pain may be aggravated by exercise. Similarly abdominal pain may be associated with nausea and even vomiting, and when the appropriate abdominal tenderness and rigidity are added, the clinical picture of visceral disease may be almost exactly reproduced.

Spinal disease might be expected to give rise to pain on movement, but the thoracic spine is easily held rigid during the ordinary movements of the trunk, and such a history is frequently lacking. Pain on movement is also produced by any visceral disease involving the parietes and may therefore be misleading.

It is a safe rule to investigate the back thoroughly as well as the viscera in every patient with pain in the chest or abdomen. The thoracic spine is notoriously difficult to examine, so that I will describe in some detail the method I employ.

First the patient is asked to map out the distribution of his pain, and by comparison

with the segmental pain areas, we can deduce the level of the supposed spinal lesion. The forced movements of the spine are then examined segment by segment, and if the pain is somatic in origin, it will be produced by moving the appropriate segment. Lastly the relevant portion of the back is explored with a needle and novocain when the site of the lesion is further defined and the spinal origin of the symptoms finally demonstrated.

This test of local anæsthesia is not as simple as it sounds. In the first place the pain must be severe at the time of examination for the test to be at all practicable. Secondly it is often difficult to define the exact site of the lesion. The finding of tender spots in the back is frequently misleading as there is always more or less tenderness within the posterior pain areas, and anæsthetizing this referred tenderness leads to nothing but confusion. If, however, we remember that it is only the deep-lying structures surrounding the articular processes and laminæ which give rise to this type of pain, and if we always deduce the level of the lesion from the segmental pain areas, these difficulties are easily overcome.

In this way the spinal origin of much thoracic and abdominal pain can be clearly demonstrated, and it remains only to consider the nature of the lesion. I believe this is usually a simple sprain resulting from some underlying deformity. In the middle-aged the most frequent deformity is a localized kyphosis with retained mobility, but in younger subjects rotation deformities are more frequent. Apart from these sprains every kind of inflammatory or neoplastic disease may be present, but in such cases the diagnosis can usually be established by the X-rays, except perhaps in the early cases of ankylosing spondylitis.

It is important to recognize these cases as they frequently respond to the simple procedure of novocain infiltration followed by hyperextension exercises, and they undoubtedly form the basis of many osteopathic cures to the detriment of our profession. Lastly I would sound a word of warning: these patients are usually middle-aged people in whom visceral disease is common, and although we may relieve their pain by treating their backs, we have in no way excluded the presence of visceral disease.

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Comment (C. Jennings Marshall, M.S., M.D.).—Chronic pain in the trunk without obvious physical signs of organic disease is perhaps the commonest diagnostic problem in the out-patient department—there may be back pain due to visceral disease, there may be anterior pain due to disease in posterior structures, there may be pain both posterior and anterior. For many years I have taught that the questioning of the patient as to the relationship of the pain, relief or aggravation, to visceral functions, and to rest, exercise and locomotion will frequently settle the question whether we have to deal with disease in viscera or in the musculo-skeletal system. There are, however, many cases in which doubt will survive such investigation—visceral symptoms may co-exist with skeletal disease, some visceral diseases may produce pains related positively to rest and motion, and with but few if any visceral symptoms, and the production of referred pain may result in visceral symptoms without organic visceral disease (the numerous forms of abdominal crisis in *tubæ dorsalis* form a useful reminder of such possibilities). The referred pains of such gross conditions as spinal caries, aneurysmal erosion, spinal tumours and metastases offer few difficulties in diagnosis; those of spinal and paraspinal chronic inflammatory lesions of “rheumatic” type are in a rather different category. In the first place the radiographic verification is not so simple as is usually taught; it has been dominated by the idea that osteophytes are pressing on spinal nerves on their emergence from the canal. The osteophytic masses so commonly obvious in X-rays are anterior to the neural foramina and cannot exert pressure on the nerves; even the grossest instances of osteo-arthritis of the spine show little encroachment on the foramina which in addition, compared with the size of the nerve, are very large indeed. (The same knowledge of pathology should be applied to osteo-arthritis as a common excuse for a failure of lumbar puncture!) We very commonly see patients showing great osteophytes in lumbar radiographs in whom questioning gives no history whatever of pain in the appropriate region—the front of the lower limb. The conception of these outgrowths being painful is fundamentally wrong—spinal pains in the vast majority of cases, as

with joint pains in general are not bone pains but ligament pains. (A similar error attributes the pain in cases of avulsion of lateral processes wrongly to the detached piece of bone.) It is of course quite well known that the pain of spondylitis is worst in the early stages when the X-ray signs are minimal or nil. The pains are referred by either, or both, of the classical routes: (1) Direct excitation of the sensory nerve by implication in the inflammatory process (and by infiltration in most cases of carcinomatous metastases), as is seen also in instances of traumatic neuromata, and in implication of nerves in scar tissue; (2) excitation of the central nervous system via the filaments supplying ligaments, &c., and reference out from the segment involved, as in the typical phrenic shoulder pain—the neurologists decline even now to be categorical as to the precise mechanism. When such excitation implicates the region between the 4th and 11th dorsal segments it becomes clear that splanchnic as well as parietal and cutaneous symptoms may arise, simulating visceral disease.

Many referred pains in the trunk arise from conditions farther along the course of the nerves than the neural foramina—the intercostal nerves and particularly the subcostal nerve very commonly are implicated in fibrositic patches, the last-named giving loin-to-groin pain simulating kidney disease and often being associated with a disappointing appendicectomy scar. For over ten years I have taught and practised novocain nerve blocks as both diagnostic and therapeutic measures of the highest value; I published a description in 1936 in *Lancet* (ii), 242.

In dozens of cases the diagnosis has been established clinically in a few minutes by this method in patients who have been submitted to numerous special investigations, often repeated several times—barium meals, cholecystographs, pyelography, barium enemas, test meals, and so on. The value of such tests applied with discrimination is not disputed but the expense, time-wasting and mental deterioration of the patient is far too often overlooked; such repeated examinations, it must be insisted, are one of the most certain ways of rendering permanent a functional disorder.

Diagnostic analgesia is of most certain application where the site of nerve implication is the intercostal space or the lateral parietes; with paraspinal and spinal forms of irritation it becomes distinctly more difficult—all the more as several segments are apt to be involved; where the irritation has permeated the neural foramen it becomes inapplicable in most cases, as it becomes almost impossible to block the affected area to its proximal limits.

Some Spinal Cases of Interest

By E. J. RADLEY SMITH, M.S., F.R.C.S.

THESE cases have shown either a prolapse of the intervertebral disc or a thickened ligamentum flavum. The former condition is, perhaps, better recognized than the latter, but even the prolapsed disc can closely mimic other spinal conditions as two of these cases will demonstrate.

First are two cases of thickened ligamentum flavum.

(i) A man of 34 fell some 20 ft. off scaffolding thirteen years ago. Ever since he has had pain in the right buttock and leg, sometimes of great severity and increasing in the last two years. His symptoms were strongly suggestive of pain arising by pressure on a nerve root. Pressure on a tender area over the 5th lumbar region caused numbness above the right ankle; standing on the left leg, with the right swinging free, caused tingling all down the right leg. The lumbar curve was found to be flattened; pain was experienced when the sciatic nerve was stretched, and the muscular power of the leg was diminished; sensation on the inner side of the ankle was diminished (4th lumbar); the ankle-jerk was unaffected. The cerebrospinal fluid revealed no abnormality.

The history and signs suggested to us a prolapse of the intervertebral disc on the right side, probably between the 4th and 5th lumbar vertebrae. Lipiodol injection was made, using only 2 c.c., and the column studied as the patient was tilted under the screen. The antero-posterior films showed a constant narrowing between 4th and 5th lumbar vertebrae (fig. 1). Lateral films were taken in the prone position, and these showed that the indentation in the lipiodol was chiefly at the back (fig. 2). In this way, the diagnosis of thickened ligamentum flavum was made before operation.

Laminectomy was performed, and the ligamentum flavum between the 4th and 5th lumbar vertebrae found to be about three times its usual thickness and also notably white

instead of yellow. The dura was much compressed, but expanded normally when the ligament was removed. No prolapse of the disc was seen or felt. The patient was up in one month, and practising golf shots in two.

(ii) The second case is, in many respects, similar. The patient is a schoolmaster, aged 42, who first began to suffer low lumbar pain five years ago, after he had been chilled while gardening. The pain was entirely lumbar for some ten days and then suddenly radiated to the left leg. For the next four years he suffered from intermittent "sciatica" and numbness of the foot, of such severity that he submitted himself to most forms of physical treatment, manipulative stretching of the sciatic nerve and epidural injection.

When admitted in 1940 he had severe left-sided sciatic pain. The usual signs were all present: wasting of calf and thigh; absent ankle-jerk; diminished sensation on the outer side of the ankle; and pain on stretching the sciatic nerve. The cerebrospinal



FIG. 1.



FIG. 2.

fluid showed no abnormality. The lipiodol studies showed a narrowing opposite the disc between the 4th and 5th lumbar vertebrae, but unfortunately attention was not directed, during screening, to the possibility that this narrowing might be posterolateral, rather than antero-lateral. Changes similar to those described in the preceding case were found at operation. This man has now remained well for some eighteen months.

At the moment it does not seem possible to differentiate clinically, with any degree of certainty, between the prolapsed disc and the thickened ligamentum flavum. Although the so-called "hypertrophy" of this ligamentum flavum was described by Elsberg as long ago as 1913, little attention seems to have been paid to it in this country until recent years. Careful screening, or radiography, in the lateral plane, after lipiodol injection, appears to be the only method of separating these two groups. The lateral films are unhappily not so easy to take with advantage, since the lumbar curve causes the lipiodol

with joint pains in general are not bone pains but ligament pains. (A similar error attributes the pain in cases of avulsion of lateral processes wrongly to the detached piece of bone.) It is of course quite well known that the pain of spondylitis is worst in the early stages when the X-ray signs are minimal or nil. The pains are referred by either, or both, of the classical routes: (1) Direct excitation of the sensory nerve by implication in the inflammatory process (and by infiltration in most cases of carcinomatous metastases, as is seen also in instances of traumatic neuromata, and in implication of nerves in scar tissue); (2) excitation of the central nervous system via the filaments supplying ligaments, &c., and reference out from the segment involved, as in the typical phrenic shoulder pain—the neurologists decline even now to be categorical as to the precise mechanism. When such excitation implicates the region between the 4th and 11th dorsal segments it becomes clear that splanchnic as well as parietal and cutaneous symptoms may arise, simulating visceral disease.

Many referred pains in the trunk arise from conditions farther along the course of the nerves than the neural foramina—the intercostal nerves and particularly the subcostal nerve very commonly are implicated in fibrositic patches, the last-named giving loin-to-groin pain simulating kidney disease and often being associated with a disappointing appendicectomy scar. For over ten years I have taught and practised novocain nerve blocks as both diagnostic and therapeutic measures of the highest value; I published a description in 1936 in *Lancet* (ii), 242.

In dozens of cases the diagnosis has been established clinically in a few minutes by this method in patients who have been submitted to numerous special investigations, often repeated several times—barium meals, cholecystographs, pycelography, barium enemata, test meals, and so on. The value of such tests applied with discrimination is not disputed but the expense, time-wasting and mental deterioration of the patient is far too often overlooked; such repeated examinations, it must be insisted, are one of the most certain ways of rendering permanent a functional disorder.

Diagnostic analgesia is of most certain application where the site of nerve implication is the intercostal space or the lateral parietes; with paraspinal and spinal forms of irritation it becomes distinctly more difficult—all the more as several segments are apt to be involved; where the irritation has permeated the neural foramen it becomes inapplicable in most cases, as it becomes almost impossible to block the affected area to its proximal limits.

Some Spinal Cases of Interest

By E. J. RADLEY SMITH, M.S., F.R.C.S.

THESE cases have shown either a prolapse of the intervertebral disc or a thickened ligamentum flavum. The former condition is, perhaps, better recognized than the latter, but even the prolapsed disc can closely mimic other spinal conditions as two of these cases will demonstrate.

First are two cases of thickened ligamentum flavum.

(i) A man of 34 fell some 20 ft. off scaffolding thirteen years ago. Ever since he has had pain in the right buttock and leg, sometimes of great severity and increasing in the last two years. His symptoms were strongly suggestive of pain arising by pressure on a nerve root. Pressure on a tender area over the 5th lumbar region caused numbness above the right ankle; standing on the left leg, with the right swinging free, caused tingling all down the right leg. The lumbar curve was found to be flattened; pain was experienced when the sciatic nerve was stretched, and the muscular power of the leg was diminished; sensation on the inner side of the ankle was diminished (4th lumbar); the ankle-jerk was unaffected. The cerebrospinal fluid revealed no abnormality.

The history and signs suggested to us a prolapse of the intervertebral disc on the right side, probably between the 4th and 5th lumbar vertebrae. Lipiodol injection was made, using only 2 c.c., and the column studied as the patient was tilted under the screen. The antero-posterior films showed a constant narrowing between 4th and 5th lumbar vertebrae (fig. 1). Lateral films were taken in the prone position, and these showed that the indentation in the lipiodol was chiefly at the back (fig. 2). In this way, the diagnosis of thickened ligamentum flavum was made before operation.

Laminectomy was performed, and the ligamentum flavum between the 4th and 5th lumbar vertebrae found to be about three times its usual thickness and also notably white

instead of yellow. The dura was much compressed, but expanded normally when the ligament was removed. No prolapse of the disc was seen or felt. The patient was up in one month, and practising golf shots in two.

(ii) The second case is, in many respects, similar. The patient is a schoolmaster, aged 42, who first began to suffer low lumbar pain five years ago, after he had been chilled while gardening. The pain was entirely lumbar for some ten days and then suddenly radiated to the left leg. For the next four years he suffered from intermittent "sciatica" and numbness of the foot, of such severity that he submitted himself to most forms of physical treatment, manipulative stretching of the sciatic nerve and epidural injection.

When admitted in 1940 he had severe left-sided sciatic pain. The usual signs were all present: wasting of calf and thigh; absent ankle-jerk; diminished sensation on the outer side of the ankle; and pain on stretching the sciatic nerve. The cerebrospinal

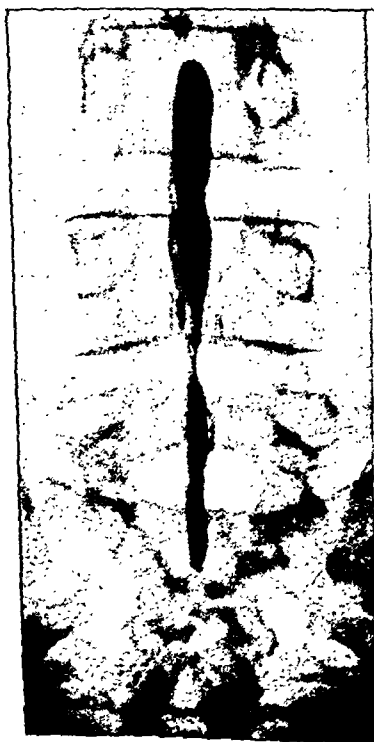


FIG. 1.



FIG. 2.

fluid showed no abnormality. The lipiodol studies showed a narrowing opposite the disc between the 4th and 5th lumbar vertebræ, but unfortunately attention was not directed, during screening, to the possibility that this narrowing might be postero-lateral, rather than antero-lateral. Changes similar to those described in the preceding case were found at operation. This man has now remained well for some eighteen months.

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The prolapsed intervertebral disc in the commonest situation—the lower lumbar spine—produces sciatic pain with, usually, low backache. If the prolapse occurs in other regions of the spine, a quite different picture is produced.

(iii) The next case is of a prolapsed disc in the common site. He was a stoker, aged 29, who had suffered no single severe back injury, but had "sprained" the back slightly many times while shovelling coal with the back bent. The typical history of low backache, for three months, followed by left sciatic pain and pins and needles on the outer side of the left foot, both for nine months, was given. He showed the usual signs of flattening of the lumbar spine; absence of ankle-jerk; pain on stretching the sciatic nerve and on pressure on the tender 5th lumbar spinous process; slight wasting of the calf and blunting of sensation over the outer side of the foot, ankle and calf. The cerebrospinal fluid was normal. The lipiodol films showed an enormous indentation of the column on the left side, opposite the disc between the 5th lumbar and 1st sacral vertebrae. Laminectomy confirmed the existence of a large prolapse of the disc in this situation.

(iv) The next case, diagnosed by Professor Nevin as one of spinal tumour, was a lorry driver, aged 37, who had had no injury but who started his heavy lorry by "swinging" it. For four months before admission, both legs had felt cold and heavy; walking was becoming increasingly difficult. He showed a spastic paraplegia, with a skin level pointing to a lesion in the region of the 7th or 8th thoracic spinal segment.

The cerebrospinal fluid findings are illuminating. On 3.3.41 the lumbar fluid contained 1 cell and 80 mg. of protein, with very little rise in the fluid pressure on jugular compression. On that date, 2 c.c. of lipiodol were put in by the lumbar route. Films taken with the patient sloping, head downwards, were not satisfactory. Therefore on 6.3.41 1 c.c. of lipiodol was injected by cistern puncture, so that the upper level of any spinal block could be seen. The fluid drawn off from the cistern—3 days after lipiodol had been injected into the lumbar subarachnoid space—contained 35 cells! This film of the cistern lipiodol revealed a block opposite the space between the 5th and 6th thoracic vertebrae, and the outline was said to suggest an intramedullary spinal lesion. On the day following cistern puncture, retention of urine developed, and laminectomy was performed at once. The ligamentum flavum was found to be normal in thickness and yellow in colour. The dura and cord were displaced considerably backwards by a tumour in front of the canal, and a prolapse of the disc between the 5th and 6th thoracic vertebrae was found on the left side. No other spinal tumour was found. Another, but rather smaller, prolapse was found on the right side of the same disc. For a few days after operation paralysis of the legs, incontinence and abdominal distension—doubtless due to operative trauma—caused some anxiety, but he made an excellent convalescence, and left hospital fully recovered four months after operation.

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The incidence of nuclear prolapse in a total series of patients claiming relief for pain of sciatic distribution is limited to a small percentage. At the neuro-surgical centre of which I have charge, of all the patients admitted with the provisional diagnosis of "sciatica, probable hernia of the disc", only about one in every ten is finally subjected to operation.

The isolation of the patient harbouring a prolapsed disc is no simple clinical problem; rather does it depend on careful clinical selection amplified by apposite investigation. At the present time I am not aware of any specific clinical syndrome of truly pathognomonic import, yet it may be said that a clinical impression can be formulated. In an analysis

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Lumbar puncture, with manometric determinations of cerebrospinal fluid pressure and estimation of the total protein content of the fluid, may produce valuable information. An absolute spinal block would be obvious, but such a disclosure is unusual with disc prolapse, rather indicating the likelihood of an underlying tumour; certainly such evidence would exclude neuritis. The employment of Queckenstedt and inverse Queckenstedt tests appears inconclusive. As for protein content, in the absence of a complete block, the percentage elevation bears no relation of consequence to either prolapsed disc or neuritis, though a normal content favours prolapsed disc rather than neuritis.

Foci of infection having been eradicated, there remains observation on the reaction of the patient to simple remedial measures, e.g. radiant heat and massage, diathermy, epidural or perineural injections of saline or novocain, or, in suitable cases, spinal manipulation together with stretching of the sciatic nerve. Some cases indicate resort to more drastic measures from the onset, but never have operative means been undertaken unless conservative methods have been applied and failed to produce lasting relief.

At this stage it is felt that the adoption of surgical treatment must be seriously considered. First of all, however, it is demanded that the patient is incapacitated from full normal activities by his affliction. Before resort to surgery is undertaken confirmatory evidence is sought by employing contrast radiography, utilizing air as the contrast medium. Air myelography is no certain procedure, for satisfactory radiographs cannot be produced in patients of heavy build; nevertheless it merits employment. The use of lipiodol, or other similar preparation, is avoided until immediately before exploration, and it is utilized then only for purposes of study, in the hope that finally a form of investigation may be evolved to the exclusion of such methods. I should like to appeal for the relinquishing of the generalized use of lipiodol as a diagnostic medium, because unpleasant reactions are apt to follow its injection, such reactions varying between increase of the original pain and the initiation of a diffuse meningeal irritation. The removal of lipiodol through a wide-bore needle has been effected by several observers, but only after prolonged manipulations not devoid of risk. That this can be done does not, in my opinion, justify its indiscriminate application.

The operative appearances vary. A hypertrophic state of the ligamentum subflavum is encountered frequently in the operative exposure. When other causes fail to be revealed, the pain is ascribed to this thickened ligament. I am not convinced that this is an adequate explanation. Rather do I believe that it is consequent on the scoliosis and is therefore the result of the lesion and not the pathological basis of the pain.

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occurs. This protrusion contains a substance of the consistency of caseous matter; moreover the periphery is apt to consist of a calcified capsule. Such calcification may, in fact, cast a shadow of such density on the radiograph as to allow of recognition on straight films.

Some Points in the Diagnosis of Osteochondritis of the Knee

By C. D. LANGTON, F.R.C.S.

A SMALL series of 13 cases of osteochondritis dissecans of the knee was treated at Horton Emergency Hospital, Epsom, in the fourteen months, April 1940 to May 1941. During this period a total of 276 cases of knee disorders were admitted, traumatic, infective, degenerative and neoplastic. Thus osteochondritis constituted 4·7% of the total. This large proportion is probably accounted for by the fact that the greater part of our knee cases are military, and that trauma is, by far, the most common cause of their disability. It must also be stated that 6 of the 13 cases had a torn intra-articular cartilage as well as the condition under review, and in 3 of these the osteochondritis was unsuspected, as it did not show in the X-ray.

All but one case gave a history of trauma, though two were a little vague—one stating that he had played a good deal of football and had hurt both knees many times, though never seriously, while the other's peace-time occupation involved considerable kneeling. The type of injury was nearly always a twist—in one case the man fell 12 feet through a skylight on to his flexed knees. Nearly all the histories, however, tended to conform to a definite pattern. Following an injury to the knee, there was pain and effusion, which soon subsided, though the joint did not become quite normal—some pain on movement, aching at rest, recurrent small effusion, or instability of the joint—any or all of these symptoms persisting for a time varying from nine years to four months, with an average of three years, until suddenly, either spontaneously or following a second injury, all the symptoms were aggravated, with, in some instances, the addition of locking. In only one case could this latter be attributed to the liberation of a loose body into the joint; while in three, it was unassociated with either a medial meniscus injury or the presence of a loose body. The locking can be explained by the fragment becoming more mobile, though still attached to the femoral condyle, the new mobility allowing it to get caught between the femur and the intact medial meniscus. Admittedly, lateral meniscus injury is not entirely excluded as the cause of the locking, though in every case it was examined, as well as possible, from the incision on the internal side of the joint, and apparently was intact. However, with such a small total number of cases, it may well be that 3 out of 13 had a damaged external meniscus, without physical signs indicating it, though no knee locked after operation in spite of ample opportunity during the subsequent rehabilitation and physical training. Unfortunately all the histories did not conform to this cut-and-dried pattern, but it does seem that there is something diagnostically helpful in the sequence outlined above.

On examination of the joint, little reliable evidence of the presence of the condition was detected. The only constant sign was wasting of the quadriceps. Limitation of movement and crepitus occurred in some cases. Tenderness on deep pressure over the affected area was present in the majority, but as this included all 6 cases with torn internal meniscus, it is impossible to say how frequently it is present in uncomplicated cases.

In the literature on this subject, X-ray examination has usually been accepted as settling the diagnosis, and so, of course, it does when positive. In 5 of our 13 cases, however, the X-ray was negative. The obvious reason for this is that the loose fragment contained only cartilage, but this was found to be incorrect. I have suggested two other reasons besides the absence of bone for a negative X-ray:

(1) The actual fracture line is too thin to show on the film. An exact parallel can be seen in fracture of the carpal scaphoid. Lachmann (1938) showed experimentally that a gap of at least 1 mm. between the opposite bony surfaces is necessary before the line of separation in the knee is visible. He excised a piece of bone and cartilage from the internal femoral condyle of a cadaver, replaced it, and took antero-posterior and lateral X-rays, gradually increasing the separation of the fragment by coating its bony surface with a measured thickness of a radio-translucent medium. The line of separation showed first in the lateral view when there was 1 mm. of separation, and in the antero-posterior with slightly more.

(2) To show, with even 1 mm. or more of separation, providing there is not gross dis-

placement, the rays from the tube which will show the lesion on the film must be tangential to the femoral condyle at the point of the lesion. This is commonly so with the central ray when the knee is X-rayed in the conventional positions, but if the lesion happens to be a little higher on, or more to one side of the condyle than usual, it may not show in the X-ray, unless special positions are taken.

Accordingly I submit that a normal pair of X-ray photographs will not rule out a diagnosis of osteochondritis; careful examination is necessary, many films requiring to be taken with a tube shift accurately measured, before there is even a reasonable probability that there are no bony changes.

Perhaps here is a reason for some of the lack of knowledge as to the origin of loose bodies in joints; further, as we know that flakes of cartilage in a knee can be absorbed, is it stretching the imagination too far to postulate that eventual arthritis can originate in this way, and that, with more accurate diagnosis and earlier operation, we may be able to diminish its incidence from this cause?

How long does it take after the causal injury for evidence to show on X-ray? That is, how long does the sodden, fibrous tissue or fibro-cartilage, found in the base of the crater in the femoral condyle, take to attain a thickness of 1 mm. Our figures do not lead to any definite conclusion, though in one case the separation is visible, in the lateral view only, in six months.

As final points in the X-ray appearances of this condition, I would mention that the general description of a small, dense area of the medial femoral condyle, surrounded by a translucent zone, is not always correct. As is well known, the bone in the partially separated piece may be alive—nourished through a tag of the posterior cruciate ligament—and it will not necessarily appear denser than the normal condyle. In fact it sometimes seems more translucent, even though completely necrosed, the explanation being that with the X-rays tangential at the site of the lesion, they have so much less depth of bone to traverse than the rays passing through the much thicker normal condyle.

It is not intended to discuss the treatment of osteochondritis, though the bearing which an earlier diagnosis would have on this may be mentioned. The onset of arthritis has already been referred to, and it is only reasonable to suppose that early operation would lessen its incidence, though a number of knees may be opened unnecessarily. Further, Axhausen (1914), Kappis (1920) and Moreau (1923) have recorded cases of spontaneous cure. What should be done for a patient giving a history on the lines sketched earlier, who has tenderness over the condyle and a wasted quadriceps, but a normal X-ray, is a subject for further work, which should not be on the "look and see" principle. Few of my points are precise, and this should serve to restrain those of very radical tendencies.

A last personal opinion is that the transverse incision along the joint line from the medial collateral ligament to the ligamentum patellæ, is inadvisable for removal of the medial meniscus, as 6 operations—out of a total of 49—for removal of a torn medial meniscus were found to be complicated by osteochondritis. The condyle can be inspected well enough through this incision, though at the expense of extending the knee during operation, but it is impossible satisfactorily to remove a partly attached fragment and to bevel the edges of the resulting crater.

To summarize, a series so small as 13 proves nothing but the need for more accurate diagnosis. Many more cases are necessary before definite conclusions can be reached about the points which suggest themselves. However, as far as they go, it seems that a history conforming to type, tenderness over the point on the condyle likely to be affected, crepitus, especially if restricted to a small arc in the total range of movement, and a wasted quadriceps—if all present together—should make one suspect the presence of osteochondritis in the absence of X-ray changes. Finally, some research in the realms of radiology is definitely indicated.

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occurs. This protrusion contains a substance of the consistency of caseous matter; moreover the periphery is apt to consist of a calcified capsule. Such calcification may, in fact, cast a shadow of such density on the radiograph as to allow of recognition on straight films.

Some Points in the Diagnosis of Osteochondritis of the Knee

By C. D. LANCION, F.R.C.S.

A SMALL series of 13 cases of osteochondritis dissecans of the knee was treated at Horton Emergency Hospital, Epsom, in the fourteen months, April 1946 to May 1947. During this period a total of 276 cases of knee disorders were admitted, traumatic, infective, degenerative and neoplastic. Thus osteochondritis constituted 4.7% of the total. This large proportion is probably accounted for by the fact that the greater part of our knee cases are military, and that trauma is, by far, the most common cause of their disability. It must also be stated that 6 of the 13 cases had a torn intra-articular cartilage as well as the condition under review, and in 3 of these the osteochondritis was unsuspected, as it did not show in the X-ray.

All but one case gave a history of trauma, though two were a little vague—one stating that he had played a good deal of football and had hurt both knees many times, though never seriously, while the other's peace-time occupation involved considerable kneeling. The type of injury was nearly always a twist—in one case the man fell 12 feet through a skylight on to his flexed knees. Nearly all the histories, however, tended to conform to a definite pattern. Following an injury to the knee, there was pain and effusion, which soon subsided, though the joint did not become quite normal—some pain on movement, aching at rest, recurrent small effusion, or instability of the joint—any or all of these symptoms persisting for a time varying from nine years to four months, with an average of three years, until suddenly, either spontaneously or following a second injury, all the symptoms were aggravated, with, in some instances, the addition of locking. In only one case could this latter be attributed to the liberation of a loose body into the joint: while in three, it was unassociated with either a medial meniscus injury or the presence of a loose body. The locking can be explained by the fragment becoming more mobile, though still attached to the femoral condyle, the new mobility allowing it to get caught between the femur and the intact medial meniscus. Admittedly, lateral meniscus injury is not entirely excluded as the cause of the locking, though in every case it was examined, as well as possible, from the incision on the internal side of the joint, and apparently was intact. However, with such a small total number of cases, it may well be that 3 out of 13 had a damaged external meniscus, without physical signs indicating it, though no knee locked after operation in spite of ample opportunity during the subsequent rehabilitation and physical training. Unfortunately all the histories did not conform to this cut-and-dried pattern, but it does seem that there is something diagnostically helpful in the sequence outlined above.

On examination of the joint, little reliable evidence of the presence of the condition was detected. The only constant sign was wasting of the quadriceps. Limitation of movement and crepitus occurred in some cases. Tenderness on deep pressure over the affected area was present in the majority, but as this included all 6 cases with torn internal meniscus, it is impossible to say how frequently it is present in uncomplicated cases.

In the literature on this subject, X-ray examination has usually been accepted as settling the diagnosis, and so, of course, it does when positive. In 5 of our 13 cases, however, the X-ray was negative. The obvious reason for this is that the loose fragment contained only cartilage, but this was found to be incorrect. I have suggested two other reasons besides the absence of bone for a negative X-ray:

(1) The actual fracture line is too thin to show on the film. An exact parallel can be seen in fracture of the carpal scaphoid. Lachmann (1938) showed experimentally that a gap of at least 1 mm. between the opposite bony surfaces is necessary before the line of separation in the knee is visible. He excised a piece of bone and cartilage from the internal femoral condyle of a cadaver, replaced it, and took antero-posterior and lateral X-rays, gradually increasing the separation of the fragment by coating its bony surface with a measured thickness of a radio-translucent medium. The line of separation showed first in the lateral view when there was 1 mm. of separation, and in the antero-posterior with slightly more.

(2) To show, with even 1 mm. or more of separation, providing there is not gross dis-

placement, the rays from the tube which will show the lesion on the film must be tangential to the femoral condyle at the point of the lesion. This is commonly so with the central ray when the knee is X-rayed in the conventional positions, but if the lesion happens to be a little higher on, or more to one side of the condyle than usual, it may not show in the X-ray, unless special positions are taken.

Accordingly I submit that a normal pair of X-ray photographs will not rule out a diagnosis of osteochondritis; careful examination is necessary, many films requiring to be taken with a tube shift accurately measured, before there is even a reasonable probability that there are no bony changes.

Perhaps here is a reason for some of the lack of knowledge as to the origin of loose bodies in joints; further, as we know that flakes of cartilage in a knee can be absorbed, is it stretching the imagination too far to postulate that eventual arthritis can originate in this way, and that, with more accurate diagnosis and earlier operation, we may be able to diminish its incidence from this cause?

How long does it take after the causal injury for evidence to show on X-ray? That is, how long does the sodden, fibrous tissue or fibro-cartilage, found in the base of the crater in the femoral condyle, take to attain a thickness of 1 mm. Our figures do not lead to any definite conclusion, though in one case the separation is visible, in the lateral view only, in six months.

As final points in the X-ray appearances of this condition, I would mention that the general description of a small, dense area of the medial femoral condyle, surrounded by a translucent zone, is not always correct. As is well known, the bone in the partially separated piece may be alive—nourished through a tag of the posterior cruciate ligament—and it will not necessarily appear denser than the normal condyle. In fact it sometimes seems more translucent, even though completely necrosed, the explanation being that with the X-rays tangential at the site of the lesion, they have so much less depth of bone to traverse than the rays passing through the much thicker normal condyle.

It is not intended to discuss the treatment of osteochondritis, though the bearing which an earlier diagnosis would have on this may be mentioned. The onset of arthritis has already been referred to, and it is only reasonable to suppose that early operation would lessen its incidence, though a number of knees may be opened unnecessarily. Further, Axhausen (1914), Kappis (1920) and Moreau (1923) have recorded cases of spontaneous cure. What should be done for a patient giving a history on the lines sketched earlier, who has tenderness over the condyle and a wasted quadriceps, but a normal X-ray, is a subject for further work, which should not be on the "look and see" principle. Few of my points are precise, and this should serve to restrain those of very radical tendencies.

A last personal opinion is that the transverse incision along the joint line from the medial collateral ligament to the ligamentum patellæ, is inadvisable for removal of the medial meniscus, as 6 operations—out of a total of 49—for removal of a torn medial meniscus were found to be complicated by osteochondritis. The condyle can be inspected well enough through this incision, though at the expense of extending the knee during operation, but it is impossible satisfactorily to remove a partly attached fragment and to bevel the edges of the resulting crater.

To summarize, a series so small as 13 proves nothing but the need for more accurate diagnosis. Many more cases are necessary before definite conclusions can be reached about the points which suggest themselves. However, as far as they go, it seems that a history conforming to type, tenderness over the point on the condyle likely to be affected, crepitus, especially if restricted to a small arc in the total range of movement, and a wasted quadriceps—if all present together—should make one suspect the presence of osteochondritis in the absence of X-ray changes. Finally, some research in the realms of radiology is definitely indicated.

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Lumbar Puncture Injuries

By A. D. EVERETT, M.S., F.R.C.S.

Injury to an intervertebral disc is still rarely thought of as a complication of lumbar puncture. In the three cases on which this paper is based, the condition was discovered while investigating the cause of persistent low back pain following this procedure.

Billington (1924) reported twelve cases of a similar nature which he described as spondylitis following cerebrospinal meningitis. Pease (1935) wrote an authoritative article based on three cases. Milward and Grout (1936) recorded five cases following spinal anaesthesia, and one further case was described by Gellman (1940). In all these cases the main signs and symptoms were essentially the same as in the present series.

Clinical features.—In each of the cases described the patient complained of persistent low back pain coming on a few days after the lumbar puncture. This pain was made considerably worse by exercise, and especially by any flexion of the spine. A certain degree of stiffness after sitting for any length of time was also experienced. On examination, a marked degree of rigidity of the lumbar spine was present, with a flattening of the normal lumbar lordosis. In one case there was actual angulation backwards, with the apex at the 4th lumbar spine. Radiographs showed collapse of the intervertebral disc relating to the space used for lumbar puncture. This was evidenced by gross narrowing of the space between the vertebral bodies. Individual features were also present in each of the three cases. In one there was clinical and radiographic evidence of infection of the bodies of the vertebra above and below the disc involved; this was sufficiently marked for the radiologist to query tuberculous infection of the spine. In a second case osteophytic outgrowths on the anterior surface of the bodies of adjacent vertebrae were a conspicuous feature, while in a third the radiographic evidence of collapse was not present for the first two months.

Treatment and results.—One patient was treated on a plaster bed in extension for three months, and then for a further three months by graduated exercises and physiotherapy. He made an exceedingly good recovery, and was boarded back to the Army category "C" with a recommendation that he should be upgraded in a further three months. The second patient refused all treatment except fourteen days' rest in bed. This patient undoubtedly showed the least improvement, and when seen three months after discharge had still not returned to work and was complaining bitterly of his back. Radiographs showed complete collapse of the disc, and marked osteophytic changes round the vertebral bodies involved. The third case remained untreated except by physiotherapy for six months. This was because when he was first admitted to hospital there was no radiographic evidence of collapse. It was not until he was readmitted to hospital some six months later that the condition was discovered. He was then treated by a lumbosacral belt which made him very much more comfortable. The two main sequelae that are still present in all three cases are difficulty in lifting weights from the stooping position and stiffness of the back after sitting for any length of time.

I agree with Pease that the probable causation of the condition is a slow seepage of the nucleus pulposus through the annulus fibrosus. Whether this occurs directly through a tear caused by the lumbar puncture needle, or whether the needle sets up an inflammation in this structure which weakens it, as suggested by Milward and Grout, it seems impossible to say, but in none of our cases was there any evidence of prolapse of the nucleus pulposus, which is the condition one would have expected if the annulus fibrosus had become weakened by an inflammatory reaction. In some cases it seems certain that there is also present a mild low-grade infection which causes an osteomyelitis of the spine. Whatever is the actual mechanism, the changes in the bodies are obviously secondary to the disc changes.

CASE NOTES

(1) A soldier, aged 21. On 15.2.41 a diagnostic lumbar puncture was done, with some difficulty. A few days after this he began to complain of low back pain. This apparently got steadily worse during the next few weeks, being especially bad towards the end of the day, when he could hardly walk. Great pain on stooping and sitting down. On examination (end of March).—Temperature 99.4°, rigidity of lumbar spine with angulation, the spine of the 4th lumbar being prominent. X-rays showed elimination of intervertebral space between 3rd and 4th lumbar, with considerable erosion of adjacent

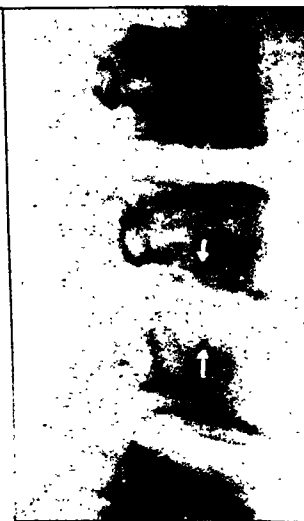


FIG. 1.

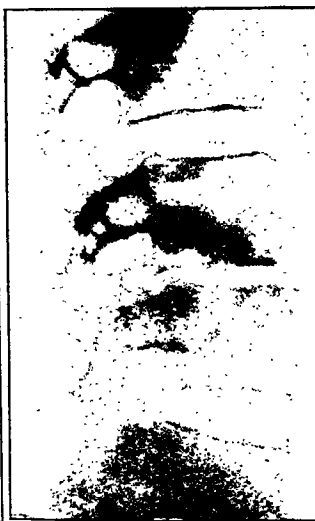


FIG. 2.



FIG. 3.

FIG. 1 (*Case 1*).—Taken within a few weeks of lumbar puncture, showing considerable collapse of intervertebral disc and erosion of the bodies of the vertebræ above and below.

FIG. 2. (*Case 1*).—Towards the end of three months on a plaster bed. Note eroded area considerably less in extent and slight forward displacement of L.4.

FIG. 3 (*Case 1*).—Taken four and a half months after beginning treatment. Patient now on remedial exercises. Note considerable diminution of joint space and slight sclerosis of vertebral bodies which are otherwise returning to normal.

aspects of the bodies of these vertebræ (*see fig. 1*). A blood sedimentation rate was done and showed a mean of 13 mm. for the two hours. A diagnosis of low-grade infection of the spine, ? cause, was made and the patient put in a plaster bed.

During the next three months patient became symptom-free. His pyrexia subsided in the first few days and his blood sedimentation rate came down to a mean of 5 mm. X-rays taken during this time showed a steady improvement of the eroded area (*see fig. 2*).

At end of June patient was allowed back into an ordinary bed, and at the end of July started to walk.

X-rays taken in August showed no sign of bony disease, but still evidence of disc collapse (*see fig. 3*). Patient was put on remedial exercises, and by the middle of September was doing full physical training. He still had some limitation of flexion and complained of difficulty in lifting weights from the stooping position. Apart from this he was symptom-free.

At end of September patient boarded back to the Army Category "C", with a recommendation for up-grading in a further three months.

(2) Sailor, aged 25. In October 1940 patient had two diagnostic lumbar punctures in one week. When first seen in November, six weeks after lumbar puncture, he was complaining of low back pain made worse by exercise.

On examination.—The lumbar spine was rigid, but there was no tenderness. X-rays showed a spina bifida, but otherwise nothing abnormal. A diagnosis of fibrositis was made, and physiotherapy given. In February 1941 lumbo-sacral belt fitted, pain in back less. In April 1941 pain improved but still pain on semiflexion and now some tenderness over 4th and 5th lumbar. X-rays showed narrowing of joint-space between 4th and 5th lumbar.

June 1941 patient discharged wearing his belt and fairly comfortable.
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FIG. 4 (*Case 3*).—Showing diminution of intervertebral joint space due to collapse of disc.

(3) Builder, aged 29. On 14.3.41 spinal anaesthesia for hernia operation. This presented some difficulty. The patient was discharged on 13.4.41, but returned as an out-patient on 21.5.41 complaining of aching pain in lower part of back and right buttock. This pain was made much worse by any flexion of the spine such as sitting down or lifting weights from the stooping position.

On examination.—Tenderness over 3rd and 4th lumbar spines and all movement of spine in this area limited. X-rays showed gross diminution of intervertebral space between border of the 3rd and 4th lumbar vertebrae (*see fig. 4*). The patient would not agree to a plaster bed or spinal jacket. He was kept fourteen days in bed after which he refused further treatment. He was kept in hospital for a further few weeks with modified rest. At the end of July X-rays showed no appreciable change and the patient was discharged.

In September he was seen again as an out-patient and was then complaining bitterly of his back. Could not lift weights and could not sit for any length of time. X-rays showed complete collapse of disc with considerable osteophytic outgrowths on anterior surface of bodies.

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Section of Orthopædics

President—C. LAMBRINUDI, F.R.C.S.

[November 22, 1941]

PRESIDENT'S ADDRESS

The Role of Orthopædics in Medical Education

By C. LAMBRINUDI, F.R.C.S.

It is tempting and certainly easier to choose as a subject for an Address, one of the many problems connected with war surgery, but, when everything is in the melting pot, all old habits subjected to scrutiny, "Planning" rather than "Wait and See" the trend of the day, I would like to use this privileged occasion to examine our system of medical education, with particular reference to the role that orthopædics is, I hope, destined to play in it in the future.

During the last war, it required all the persuasive genius of Sir Robert Jones, before orthopædic surgeons were given a fair trial; in this one there are not enough to go round.

After the last war, one teaching hospital after another appointed an orthopædic surgeon to its Staff, now most of them have two. Chairs of orthopædic surgery have been created in several universities.

In thinking about orthopædics and education, one must, of course, draw a distinction between the education of an orthopædic surgeon, and the place that orthopædics ought to take in the general training.

The question we ought to ask ourselves is, not how much orthopædics should a student be taught, but rather how may orthopædics be used to enhance the intellectual equipment of the budding general practitioner. Put in that form, emphasis is laid, as it should be, on education, and not specialization.

The medical educationalists of to-day are the general surgeons and physicians. It is they, in the main, who arrange the curriculum, and see to it that the student does not enter the mysteries of the special departments until very late in his career, when he is almost rushed through them.

Our medical and surgical colleagues have a profound distrust of specialists, and whenever a part of general medicine or surgery is nipped off and takes the form of a specialty, it is cast aside and put into the background so far as teaching is concerned, always on the plea that a student must have a general training before he can specialize.

This habit has become cemented by tradition. Priestley puts it aptly, when he says that: "Tradition is meant to be a Guide and not a Jailer." In the pre-clinical period the teachers and examiners are all specialists, in the clinical period the teachers and examiners are in the main general surgeons and physicians. Which is the right tradition? It is pertinent to ask: Are we becoming jail-birds?

In the pre-clinical period, the specialists are physicists, chemists, biologists, anatomists and the physiologists. They all work together for the benefit of the student, and as far as I know, not one of them abrogates to himself a special place of authority. The final product of that period is good, especially lately since anatomy is being taught with more imagination. When we first meet the student, after he has passed his Second M.B., he seems on the whole to be well trained, facts have been presented to him in an orderly manner, he does not appear too muddled and is ready for the next step forward in his career.

But when he enters the wards, that method of teaching through a series of graduated and well-balanced specialties, which worked well in the past, is abandoned. He is plunged into a new world, a new emotional state, has innumerable strange names, and a huge variety of strange pathological conditions flung at him, none of which appear to him to be connected one with the other, and still less with the mechanics, biology, anatomy and physiology which he has already learnt and been taught to believe were the foundations of his future activities.

His foundations, acquired with such labour, are suddenly taken from him, and a strange



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with a problem that had an obvious bearing on the only medical knowledge at their disposal, anatomy and physiology, they automatically applied it and gave the correct answer. In the manner of any healthy young animal when placed in its proper environment, they were educating themselves and opening up their association tracts. It is not so much that the senior men had forgotten the broad facts of anatomy and physiology, but they had never been placed in the suitable atmosphere at the right time in which to learn how to apply it. These association tracts had never been used. Yet, they too, responded to my question in a perfectly normal manner.

The difference between them was their mental reflexes, or training, not their knowledge or intelligence. For two years the young men had been trained to think in terms of anatomy, physiology and function, whilst for the last three years, the older men had concentrated on disease, deformity, and operations, the fundamental subjects lay dormant, unassociated, isolated. I do not think that I am guilty of over-emphasis, if I repeat that according to their respective lights, both these sets of students reacted to the stimulus of my question in a normal way. But, it is a reflection on a system to regard it as normal that junior men should recognize and interpret a case requiring a knowledge of fundamental subjects, more readily than their older and more erudite companions.

I do not want to imply that it is a calamity to fail to spot the details of a case of infantile paralysis, but it does seem that orthopædics offers an excellent opportunity to put into practice the knowledge gained in the pre-clinical period without introducing too much that is new and confusing. Indeed, in my experience, the junior men take to orthopædics and seem to profit from what it has to teach very much more than senior ones. What is needed, is, so to speak, a grammar of Clinical Medicine, something that will take the place of Latin and Greek in a general education, which aims not so much at instruction, but in training the mind. I believe, that we, together with some of our other specialist colleagues, could write it.

The subjects I would choose would be orthopædics including fractures, neurology including elementary psychology, and urology. All these are essentially anatomy and physiology, they are all to some extent connected one with the other, and they are neither wholly surgical nor wholly medical, but a combination of the two.

What better exercise in applied anatomy and physiology, than localizing a neurological lesion, or a bleeding point in the genito-urinary tract?

I would include urology, because of its precision, its nice use of instrumentation, and the comparatively simple nature of its physiological and biochemical tests; moreover, the mechanical effects on the viscera, due to obstruction, are beautifully and simply demonstrated by X-ray and, the secondary effects on the cardiovascular system are well within the competence of a student fresh from his Second M.B. to understand. In fact, in that compact subject he will see in miniature and in easily demonstrable form all the mechanical and physiological principles of visceral surgery.

Orthopædics too, with its emphasis on function rather than extirpation of disease, has a special educational value in the early clinical period. It is almost entirely applied mechanics, anatomy and physiology, with just enough pathology to be a stepping stone to profounder studies later. It gives an excellent training in observation and deduction, for apart from X-rays, it requires no aids to diagnosis, only the eyes, the fingers, and an accurate taking and evaluation of history are needed. Moreover, fractures offer a good opportunity for the first intimate contact between student and patient.

What better exercise in observation, than interpreting a limp, or in applied anatomy, than analysing a case of infantile paralysis, and planning a tendon transplantation or some other operation to improve function? I am not going to labour the value of fractures, their production, reduction, and treatment by adequate splintage as a lesson in applied mechanics, but a word or two about posture is not out of place. It shows a lack of sense of proportion, that a student should have to wait to the end of his training before the true significance of that huge group of minor ailments, attendant upon poor body mechanics, is revealed to him. The symptoms to which they give rise, local pains, referred pains, referred tenderness, low level of health, often so simulate organic disease, that they will frequently have seen such cases in the general ward subjected to routine examinations, even laparotomy, with negative results, before the true nature of the symptoms were appreciated. Posture, and maluse of the whole or part of the body, is a physiological problem. It is connected with muscle tone, reciprocal innervation of muscles, their grouping into agonists, antagonists and synergists, and requires for its

thing called pathology is prematurely put in their place. He is thrown into utter confusion.

We, who work in a department where the student population consists of final year men about to take their qualifying examination, see this confusion. We find that their powers of observation and deduction are disappointing, and that they have forgotten their anatomy and physiology; consequently when the more enterprising of them attempt to apply what they have learnt in the general wards, to what they see in our department, it almost invariably is in terms of pathology. More often, however, they make very little effort to apply their knowledge, and even fail to detect signs of inflammation. In other words, even at this late stage, when they enter a new sphere, they apparently cut themselves off from a lot that has gone before, and start afresh.

The fact is, that all students are "specialists", with that type of single-track mind which the general surgeons and physicians abominate so much, and try, so hard and unsuccessfully, to crush. This is a fact that we teachers must accept, and not fight against, for it is a normal process of intellectual development.

Whenever a crisis arises through poor examination results, the remedy is always the same, more or different registrars, and more classes. I have never heard the subject of medical education broadly discussed in Committee, or a doubt raised as to whether we were presenting the material to our students in a palatable and coherent form.

This tendency to "specialize", in its narrow sense, or, if you like, learning in a series of more or less watertight compartments, is a characteristic of the growing mind. A child "specializes" in crawling before standing, in standing before walking, in a tricycle before a bicycle, and so on all along the road to maturity. That as he grows he can "specialize" in an ever-increasing number of things, does not alter the fact, that his approach to each in turn is that of a single-minded "specialist". Multiplicity of "specialisms" does not of itself make for breadth of vision or wisdom, only the process of integration, which is the very essence of maturity, can do that.

Maturity may appear precociously, more often in the late twenties, too frequently never at all, and there are many in high places, wielding authority, whose intellectual development has not progressed beyond the "multi-specialist" stage. There is one thing certain, and that is that passing the Second M.B., or attaining a certain age is not the yardstick by which to measure it, or the signal to abandon a method of teaching suitable to immature minds. I doubt if anything can hasten maturity; confusion can most certainly delay it, perhaps even frustrate it, and I am convinced that, what must be to the student a jumble of "multi-specialties" presented in haphazard sequence, which goes by the treasured name of general training, is not the best introduction to clinical medicine.

Pari passu with this mania to "specialize", the developing mind shows a strong inclination for the association of ideas. Provided the association is obvious, it will seek to already knows. A child, for instance, when he first sees an aeroplane, will call it a bird. It is a kind of mental reflex, one which all wise teachers of the young appreciate and make use of, and must not be confused with the purely intellectual process of selecting material for comparison.

Our medical educationalists should not try to crush normal peculiarities of intellectual development. Instead, they should utilize them and so arrange the curriculum that subjects follow one another in logical sequence, choosing for the first, those which have an obvious bearing on mechanics, anatomy and physiology.

In this connexion I have had an illuminating experience. On account of the scattering of our school, my dressers now consist of final year men as before, and first year men fresh from the Second M.B. One day, at the beginning of the war, a patient appeared at Out-patients with a paralytic equinovarus, walking with a marked limp due to paralysis of the glutei muscles. I asked the students if they thought the limp would be improved if I corrected the deformity of the foot. The senior men said "Yes", and talked learnedly about a subastragaloid arthrodesis, the junior men said "No, because he is walking with a tilted pelvis". In all my experience of teaching senior students, I have never had that answer given me so spontaneously. These raw young men had spotted for themselves the Trendelenburg limp, and there was no difficulty whatever in explaining to them its diagnostic significance.

Naturally, my first impression was that they were exceptionally intelligent. But, on further reflection, I realized that they were merely displaying normal reactions. Faced

with a problem that had an obvious bearing on the only medical knowledge at their disposal, anatomy and physiology, they automatically applied it and gave the correct answer. In the manner of any healthy young animal when placed in its proper environment, they were educating themselves and opening up their association tracts. It is not so much that the senior men had forgotten the broad facts of anatomy and physiology, but they had never been placed in the suitable atmosphere at the right time in which to learn how to apply it. These association tracts had never been used. Yet, they too, responded to my question in a perfectly normal manner.

The difference between them was their mental reflexes, or training, not their knowledge or intelligence. For two years the young men had been trained to think in terms of anatomy, physiology and function, whilst for the last three years, the older men had concentrated on disease, deformity, and operations, the fundamental subjects lay dormant, unassociated, isolated. I do not think that I am guilty of over-emphasis, if I repeat that according to their respective lights, both these sets of students reacted to the stimulus of my question in a normal way. But, it is a reflection on a system to regard it as normal that junior men should recognize and interpret a case requiring a knowledge of fundamental subjects, more readily than their older and more erudite companions.

I do not want to imply that it is a calamity to fail to spot the details of a case of infantile paralysis, but it does seem that orthopaedics offers an excellent opportunity to put into practice the knowledge gained in the pre-clinical period without introducing too much that is new and confusing. Indeed, in my experience, the junior men take to orthopaedics and seem to profit from what it has to teach very much more than senior ones. What is needed, is, so to speak, a grammar of Clinical Medicine, something that will take the place of Latin and Greek in a general education, which aims not so much at instruction, but in training the mind. I believe, that we, together with some of our other specialist colleagues, could write it.

The subjects I would choose would be orthopaedics including fractures, neurology including elementary psychology, and urology. All these are essentially anatomy and physiology, they are all to some extent connected one with the other, and they are neither wholly surgical nor wholly medical, but a combination of the two.

What better exercise in applied anatomy and physiology, than localizing a neurological lesion, or a bleeding point in the genito-urinary tract?

I would include urology, because of its precision, its nice use of instrumentation, and the comparatively simple nature of its physiological and biochemical tests; moreover, the mechanical effects on the viscera, due to obstruction, are beautifully and simply demonstrated by X-ray and, the secondary effects on the cardiovascular system are well within the competence of a student fresh from his Second M.B. to understand. In fact, in that compact subject he will see in miniature and in easily demonstrable form all the mechanical and physiological principles of visceral surgery.

Orthopaedics too, with its emphasis on function rather than extirpation of disease, has a special educational value in the early clinical period. It is almost entirely applied mechanics, anatomy and physiology, with just enough pathology to be a stepping stone to profounder studies later. It gives an excellent training in observation and deduction, for apart from X-rays, it requires no aids to diagnosis, only the eyes, the fingers, and an accurate taking and evaluation of history are needed. Moreover, fractures offer a good opportunity for the first intimate contact between student and patient.

What better exercise in observation, than interpreting a limp, or in applied anatomy, than analysing a case of infantile paralysis, and planning a tendon transplantation or some other operation to improve function? I am not going to labour the value of fractures, their production, reduction, and treatment by adequate splintage as a lesson in applied mechanics, but a word or two about posture is not out of place. It shows a lack of sense of proportion, that a student should have to wait to the end of his training before the true significance of that huge group of minor ailments, attendant upon poor body mechanics, is revealed to him. The symptoms to which they give rise, local pains, referred pains, referred tenderness, low level of health, often so simulate organic disease, that they will frequently have seen such cases in the general ward subjected to routine examinations, even laparotomy, with negative results, before the true nature of the symptoms were appreciated. Posture, and maluse of the whole or part of the body, is a physiological problem. It is connected with muscle tone, reciprocal innervation of muscles, their grouping into agonists, antagonists and synergists, and requires for its

understanding a fairly intimate knowledge of the physiology of the neuromuscular system. The right time to impress its most important bearing on clinical medicine, how to detect its abnormalities, the reasons why such abnormalities give rise to symptoms, the variety of factors which influence it, is at the beginning of the clinical period, when physiology is still fresh in the mind of the student.

Before leaving the subject of orthopaedics, there is one other aspect of it which I think requires emphasis on account of its great general educational value, and that is its close association with the Social Services of the hospital. A student should be given the opportunity of appreciating that we are as interested in the environment and soil from which Diseases and Dysfunction arise, as in their cure. Most cases of ill-health consequent upon poor muscle tone are social problems; maladjustments, malnutrition, overcrowding, poverty, fatigue, &c., and without the assistance of the Lady Almoner we are quite unable to help our patients. At present the student only sees this contact late in his career when clerking in the Departments of Psychology, Tuberculosis and Orthopaedics, and even then its extreme importance is insufficiently stressed.

My theme throughout this paper has been that learning, like any other activity of the central nervous system, is a reflex action, a matter of external stimulus and response. The ultimate aim of education is to awaken the higher intellectual centres and assist maturity. But, in the young, it is the lower mental centres which require our immediate attention, maturity can safely be left to look after itself. The best way to assist development is to avoid confusion by placing the student in an environment in which he can step by step educate himself, and I believe that, in the early forcing period, it is in the terrain of some of the specialties where the most suitable soil is to be found and the appropriate atmosphere most easily created.

If we have decided that certain subjects are fundamental to the study of medicine and are prepared to devote two or three years to them, it is folly not to make sure that these subjects become conditioned in the student's mind. This can be achieved without prolonging the training simply by rearranging the curriculum.

After the student has spent the first six months of the clinical period in departments which continually stress the important association of mechanics, anatomy and physiology to clinical medicine, he will be well grounded. By dint of constant repetition, from different angles and by different teachers, the reflexes will become conditioned, the fundamental subjects less likely to atrophy from disuse, then, he will be ready to broaden his foundation to include pathology and proceed to the study of disease.

Before we can persuade our general medical and surgical colleagues to allow us to take our proper place in the training of medical students, we must first try to persuade them to overcome their prejudice against us specialists. I cannot believe that they really object to specialism, and assume, that because a man studies a subject profoundly, he necessarily becomes narrow-minded; the reverse is the case, the more an intelligent person knows of one thing, the more he perceives its ramifications, the wider his horizon inevitably grows.

It has no doubt often happened, that on account of personal popularity, or other reasons, men, who failed to get on to the General Staff of their Hospital, because they did not quite come up to the standard, have been appointed in charge of a special department. Consequently, I believe, the idea has grown that specialists are intellectually second raters. How strong this factor is, it is hard to say, but, I suspect that subconsciously it plays a bigger part than either we or they realize. I personally have no objection to their holding such opinions. I am not arguing for myself or for you, only for specialism. If they feel their objections to us are valid, the onus lies with them to appoint specialists whom they can trust, and not on account of personal prejudice deny to specialism its logical place in the scheme of education.

On our side we too are to blame. We have not yet lived down the exuberance of youth. When struggling for advancement, there is a definite technique, and that is, to persuade the other fellow that you have something very special to sell. Our particular ware which we hawk around, is "orthopaedic principles". I have studied orthopaedics for twenty-five years, and have failed to perceive a single principle upon which our work is based, that was not taught to me by the Surgical Registrar, when I was a ward clerk. In reality we only employ general principles applicable to every branch of medicine and surgery. How can we ask to take a share in the general training of a student, when we talk of very special principles of our own? -What we can claim, and with very good reason, is that we teach

general principles, and that we practise them on a larger scale in meticulous detail, and with amazing perseverance. That, I maintain, is our strongest claim for taking our proper place, which is at the beginning, and not at the end, of the clinical period.

If we are to play our part in any such reconstruction, we, particularly those of us on teaching hospitals, will have to reorientate ourselves. It will require a considerable amount of mental readjustment to change our position from that of a more or less post-graduate teacher to a muddled audience, already harassed by the prospect of approaching examinations, to one of a team of teachers of elementary surgery to eager young students. For the purposes of undergraduate teaching, we must seek to simplify our subject, we must get out of the "interesting case" complex, we must learn to extract interest from the commonplace, always to think of the student's intellectual development, always try to bridge the gap between what he already knows and what he is going to meet with in the immediate future. Moreover, I think we would do well to follow the tradition of the teachers of the pre-clinical period, where the heads of the departments themselves undertake the teaching of the fundamentals of their subject, and do not leave it to comparatively inexperienced assistants or registrars as occurs at present in the clinical period.

I trust the day will come, when all of us teachers will have a round-table conference to rearrange the curriculum upon more logical lines, and try to give it a more definite form, so that the student will the more readily perceive that it is all one continuous tale and not a cumbersome tome of short disconnected stories, some interesting and some dull.

I appeal to our general medical and surgical colleagues to elevate us from the status of short-story tellers, and allow us to contribute a few of the earlier chapters in that most thrilling of all narratives, "The Marvels of the Human Body".

The Conservative Treatment of Osteomyelitis

By K. C. McKEOWN, M.Ch., F.R.C.S.Ed.

RECORDS of the use of sulphathiazole in cases of acute osteomyelitis are comparatively scarce. Those of Melton, G. (sulphathiazole in treatment of staphylococcal infections, *Lancet* (i), 274, 1941), Smith, A. L. (*Medical Times, New York*, 68, 268, 1940), and Phemister (*Bull. New York Acad. of Med.*), may be mentioned. These papers, however, deal mainly with its use in the reduction of the primary mortality of the disease.

The object of this paper is to give a preliminary report of a short consecutive series of unselected cases treated with the drug.

The surgical treatment in all cases has been minimal, consisting of incision of the periosteum and drilling of the bone in the acute stage; and sequestrectomy, if required, at a later date.

The dosage of the drug was estimated as 1 g. of sulphathiazole per 20 lb. body-weight.

The cases fall into two groups.—In the first three cases to be described administration of the drug was commenced at the onset of the disease and was continued for eight days; a repeat course being given after an interval of three weeks.

In the other three cases administration of sulphathiazole began twelve to forty-eight days after the onset of symptoms.

GROUP I

CASE I.—Boy, aged 10 years. Complained of pain over the lower end of the left femur for a day before admission to hospital. On examination the patient looked toxic. The temperature was 103° F. and the pulse 130. There was marked tenderness over the lower end of the femur and swelling of the knee.

Sulphathiazole was given at once in doses of 6 g. in the twenty-four hours and continued according to the plan already mentioned.

Operation was performed at the end of the second day. The lower end of the femur was exposed, the periosteum, which was thickened, was incised, and five drill holes were made in the metaphysis. Pus exuded from the lower four holes. A vaseline pack was inserted and plaster applied. The condition rapidly improved during the following five days.

Examination of the pus showed *Staphylococcus aureus* and a profuse growth was cultured.

Estimation of the blood sulphathiazole showed the concentration of the drug to be 7 mg. %.

X-ray at the end of one month showed commencing sequestration of a small area of the originally drilled cortex. The bone changes were minimal in extent.

Sequestrectomy was carried out after five months.

In this case the duration of treatment was six and a half months. On discharge the patient walked well with no limp. Flexion of the knee was almost full. The scar was soundly healed and has remained so.

CASE II.—Girl, aged 11 years. Complained of pain over the lower end of the left femur of two days' duration. Acute osteomyelitis of the lower end of the femur was diagnosed. A course of sulphathiazole was given and operation was carried out after six days. The periosteum was incised, and pus lying below it was evacuated. A single large drill hole was made in the shaft of the bone. The wound was packed with vaseline gauze and plaster applied.

Examination of the pus showed Gram-positive cocci, and culture yielded a scanty growth of *Staphylococcus aureus*.

Blood culture was sterile after six days' incubation.

X-ray examination after two months showed a small sequestrum, but otherwise bone change was minimal.

A small sequestrum was removed from the sinus after three months.

The patient was discharged after four and a half months' treatment. At this time the wound was soundly healed and movements of the knee-joint were only slightly limited (130°).

CASE III.—Boy, aged 9 years. Complained that, following an injury, he had pain in the region of the right arm. He was given a course of sulphathiazole and operation was carried out after ten days. A large abscess over the lower end of the right humerus was incised. The bone was not drilled. Subsequent treatment was carried out by the closed plaster technique.

X-rays taken after three weeks and further examination at eight weeks showed no bone change. The sinus was soundly healed at the end of four months' treatment and the boy has subsequently had no further trouble.

Group II

CASE IV.—Girl, aged 14 years. Complained of pain over the upper end of the left tibia and swelling over the knee-joint. The duration of symptoms before admission to hospital was two days.

Operation was carried out at the end of the second day. The periosteum was incised over the upper end of the tibia and four drill holes were made in the metaphysis. Pus exuded from all of these.

Culture of pus grew *Staphylococcus aureus*.

Sulphathiazole was not given until twelve days after the onset of the disease.

X-ray at the end of four weeks showed osteomyelitic changes in the metaphysis of the tibia. A sinus was still discharging over the upper end of the tibia after eight months but the wound appeared soundly healed three months later. At the present time there are arthritic changes in the knee-joint and flexion of the knee is limited to 90 degrees. The duration of treatment in this case was one year.

CASE V.—Girl, aged 11 years. History of a fall a week before symptoms began. She then complained of pain in the right knee and was unable to walk for a week. She was admitted to hospital and operated on two days later; that is, ten days after the commencement of the symptoms. The lower end of the femur was exposed and pus evacuated from the metaphysis by a single large drill hole. A course of sulphathiazole was not started until the twelfth day of the disease and a repeat course was given in six weeks.

X-ray at the end of three months showed extensive osteomyelitic changes in the lower third of the femur; and a further X-ray four months later showed that sequestration had taken place.

Sequestrectomy was carried out at the end of the eighth month, and a pedicle muscle graft inserted into the bone cavity. The wound was closed, and sound healing was obtained. The duration of treatment in this case was ten months.

CASE VI.—Boy, aged 10 years. He stated that he fell over a railing and injured his right thigh. Three days later he had very severe pain in the right thigh but he was not admitted to hospital until just under two weeks from the onset of the pain.

Operation was carried out and two large drill holes were made over the lower end of the shaft of the femur.

In this case sulphathiazole was not given until six weeks after the onset of the disease. Examination of the pus showed that the infection was staphylococcal.

X-ray showed osteomyelitis of the lower half of the shaft of the femur. A further X-ray at the end of three months showed the formation of a sequestrum and the bone changes were more marked than in the previous cases, though still confined to the lower half of the femur. Sequestrectomy was performed at the end of eight months. At the time of discharge the wound was soundly healed; the range of movement at the knee-joint was considerably limited. The duration of treatment in this, the last case of the series, was fourteen months.

CONCLUSION

In none of the six cases described was there spreading and gross bone involvement, but rather the destructive changes remained localized to a relatively small area.

The bone involvement in cases in Group I in which early administration of the drug was carried out was minimal, while in cases in Group II in which the drug was given at a later date the bone change was much more marked.

The duration of treatment averaged five months when the drug was given early, and eleven months when its administration was delayed.

In comparing the duration of treatment of this series of cases with similar cases treated at Queen Mary's Hospital during the past five years, before the advent of sulphathiazole, it was found that the duration of the disease was approximately halved.

I wish to thank Dr. W. Allen Daley for his kind permission to use the clinical material available at Queen Mary's Hospital, Carshalton.

Physiotherapy and the Soldier's Foot

By E. J. CRISP, M.B.

At every E.M.S. Orthopædic Centre a considerable problem is presented by the large numbers of soldiers with painful feet. Of these, only a minority have glaring defects, such as gross hallux valgus or rigidus, pes cavus with clawing of the toes, fixed valgus deformity secondary to spasmodic valgus or other similar conditions. These men have been placed in too high a medical category, and the only treatment advisable in the majority of cases is regrading or discharge from the Army, while of the remainder some may be improved by the orthopædic surgeon and others helped by the orthopædic bootmaker. Physiotherapy plays but a passive role; it alleviates, but cannot cure.

Fortunately, the type of case just described represents but a small percentage of those coming for treatment. The real problem is presented by the apparently normal foot which has broken down during military training. This type of case we see in ever-increasing numbers, and the absenteeism for which it is responsible must be causing the Army medical authorities much anxiety.

I am going to divide the cases into two groups, namely, Group A: feet which gave no trouble prior to joining the Army, and Group B: feet which, though more or less normal to look at, were already giving trouble in civil life. The majority of cases coming for treatment will be in Group B.

(A) THE FOOT WHICH WAS TROUBLE-FREE PRIOR TO JOINING THE ARMY

Only a foot which is mechanically sound in every respect will stand up to the Army's vigorous system of training. Many a foot which in peace-time did all that was asked of it without complaint, nevertheless had certain inherent weaknesses which only came to light when it was subjected to greatly increased activity. The following types of foot are likely to breakdown:

(1) *The weak foot*.—The foot which we associate with a feeble physique, the foot with a deficient musculature due to a sedentary life, adolescence, overweight in early middle age, or following acute illness.

(2) *The stiff foot*.—The foot which is stiff as the result of inactivity, following a sprain, or as the result of assuming an abnormal posture on account of a painful focus. For example, the recruit with a plantar wart may march with his foot in varus in order to avoid pain. His foot will, as a result, ultimately become stiff.

(3) *The inco-ordinated foot*.—The foot in which individual muscle control is lacking, and which the owner uses not as a foot, but rather as a cow uses its hoof.

A recruit with feet subject to one or other of these defects is bound to crack up at a certain stage of his training, the contributing factor being ill-judged progression of his activities.

The Army boot.—I have not apportioned any blame to the Army boot, which is admirable for the normal foot. It is only the frankly defective foot which will be aggravated by its use. Soldiers with sound feet, without exception, all tell me that the Army boot is exceedingly comfortable. Moreover, when on leave they have found their civilian shoes rather uncomfortable and have been glad to return to their Army boots. Though soldiers with painful feet blame Army boots, what is really at fault is the Army's system of training.

The weak foot.—Acute fatigue, the direct result of making weak muscles work under

Sequestrectomy was carried out after five months.

In this case the duration of treatment was six and a half months. On discharge the patient walked well with no limp. Flexion of the knee was almost full. The scar was soundly healed and has remained so.

CASE II.—Girl, aged 11 years. Complained of pain over the lower end of the left femur of two days' duration. Acute osteomyelitis of the lower end of the femur was diagnosed. A course of sulphathiazole was given and operation was carried out after six days. The periosteum was incised, and pus lying below it was evacuated. A single large drill hole was made in the shaft of the bone. The wound was packed with vaseline gauze and plaster applied.

Examination of the pus showed Gram-positive cocci, and culture yielded a scanty growth of *Staphylococcus aureus*.

Blood culture was sterile after six days' incubation.

X-ray examination after two months showed a small sequestrum, but otherwise bone change was minimal.

A small sequestrum was removed from the sinus after three months.

The patient was discharged after four and a half months' treatment. At this time the wound was soundly healed and movements of the knee-joint were only slightly limited (130°).

CASE III.—Boy, aged 9 years. Complained that, following an injury, he had pain in the region of the right arm. He was given a course of sulphathiazole and operation was carried out after ten days. A large abscess over the lower end of the right humerus was incised. The bone was not drilled. Subsequent treatment was carried out by the closed plaster technique.

X-rays taken after three weeks and further examination at eight weeks showed no bone change. The sinus was soundly healed at the end of four months' treatment and the boy has subsequently had no further trouble.

GROUP II

CASE IV.—Girl, aged 14 years. Complained of pain over the upper end of the left tibia and swelling over the knee-joint. The duration of symptoms before admission to hospital was two days.

Operation was carried out at the end of the second day. The periosteum was incised over the upper end of the tibia and four drill holes were made in the metaphysis. Pus exuded from all of these.

Culture of pus grew *Staphylococcus aureus*.

Sulphathiazole was not given until twelve days after the onset of the disease.

X-ray at the end of four weeks showed osteomyelitic changes in the metaphysis of the tibia. A sinus was still discharging over the upper end of the tibia after eight months but the wound appeared soundly healed three months later. At the present time there are arthritic changes in the knee-joint and flexion of the knee is limited to 90 degrees. The duration of treatment in this case was one year.

CASE V.—Girl, aged 11 years. History of a fall a week before symptoms began. She then complained of pain in the right knee and was unable to walk for a week. She was admitted to hospital and operated on two days later; that is, ten days after the commencement of the symptoms. The lower end of the femur was exposed and pus evacuated from the metaphysis by a single large drill hole. A course of sulphathiazole was not started until the twelfth day of the disease and a repeat course was given in six weeks.

X-ray at the end of three months showed extensive osteomyelitic changes in the lower third of the femur; and a further X-ray four months later showed that sequestration had taken place.

Sequestrectomy was carried out at the end of the eighth month, and a pedicle muscle graft inserted into the bone cavity. The wound was closed, and sound healing was obtained. The duration of treatment in this case was ten months.

CASE VI.—Boy, aged 10 years. He stated that he fell over a railing and injured his right thigh. Three days later he had very severe pain in the right thigh but he was not admitted to hospital until just under two weeks from the onset of the pain.

Operation was carried out and two large drill holes were made over the lower end of the shaft of the femur.

In this case sulphathiazole was not given until six weeks after the onset of the disease. Examination of the pus showed that the infection was staphylococcal.

X-ray showed osteomyelitis of the lower half of the shaft of the femur. A further X-ray at the end of three months showed the formation of a sequestrum and the bone changes were more marked than in the previous cases, though still confined to the lower half of the femur. Sequestrectomy was performed at the end of eight months. At the time of discharge the wound was soundly healed; the range of movement at the knee-joint was considerably limited. The duration of treatment in this, the last case of the series, was fourteen months.

A deformity occurring in so short a time, and blamed the recruiting boards for faulty training. However, I am now quite satisfied that military training is responsible for the deformity, but that it only occurs in feet which are inherently deficient in individual muscle strength. Every foot which has broken down from this defect shows one constant feature, namely, its inability to dissociate the tibialis anticus from the long extensors of the toes. These two muscles, in addition to their other functions, act synergically in dorsiflexing the foot, but in every single case we shall find the long extensor of the toes greatly overacting at the expense of the tibialis anticus, whose action is either partly or completely suppressed. Presumably, fatigue plays a part in the production of this state of affairs, and it is worth pointing out that the extensor longus digitorum is at a mechanical advantage when the foot is turned out, and the tibialis anticus when the foot is turned in. In addition, pain in the transverse arch may provoke a "pain with drawal" phenomenon, in which the toes are powerfully hyperextended by the unimpeded reflex contraction of the long extensors.

Once we understand the mechanism by which this very interesting type of deformity is produced, it is comparatively easy to reverse the procedure and to re-educate the foot to normality.

After a preliminary rest in bed, the keynote of our treatment is to teach dissociation of the tibialis anticus and the extensor digitorum longus. This we do by training the patient to plantar-flex his toes while dorsiflexing his foot, and vice versa to extend his toes while plantar-flexing his foot. This I may say needs endless patience from the manœuvre, full co-operation from the patient, and, of course, strictly individual treatment. As an additional measure we apply surging faradism to the tibialis anticus and the intrinsic, and, if contractions are present, passive stretchings of the toes will be necessary.

I have recently devised an improved method of muscle stimulation by which several individual muscles, in this case the tibialis anticus and the intrinsic of the foot, may be made to contract simultaneously and perform a definite action. The machine used is an "Indolor" and each muscle or group of muscles is supplied through a separate potentiometer so that each may receive an equal stimulus. The foot is attached to a footpiece which is hinged at the level of the metatarsophalangeal joints, the toes being tied down separately. The rear of the footpiece rotates on a swivel, the front of the footpiece slides to and fro in a horizontal slot, the result being that dorsiflexion of the foot produces plantar-flexion of the toes.

Two electrodes each are applied to the tibialis anticus and to the intrinsic. On switching the current, contraction of the tibialis anticus produces dorsiflexion of the foot, and by the attachment of the foot to the footpiece, plantar-flexion of the toes. Simultaneously, the stimulus to the intrinsic produces full lumbrical action. This is the precise movement we have been endeavouring to teach the patient and helps in re-education. At the same time our electrical stimulation strengthens the two muscles which are weak and, in addition, powerfully stretches the long extensor tendons and metatarsophalangeal joints, rendering any manipulation quite unnecessary.

By these various means this type of foot is re-educated, muscular tone and co-ordination restored, and deformities corrected. Then, and only then, does the recruit start class work and in due course P.T. After a stay in hospital of upwards of six weeks he will be fit to return to his unit, wearing his Army boots, and in his original category.

In theory, almost 100% of cases of the types which I have just described, should recover completely. In practice, our results are now approaching this figure. We have only achieved this by rigorous observance of the principles of preliminary rest, individual treatment, and a sufficiently long stay in hospital.

(B) THE FOOT WHICH WAS ALREADY GIVING TROUBLE IN CIVIL LIFE

The majority of people who suffered with their feet in days of peace, brought the trouble on themselves, a sedentary life, faulty posture, muscular inco-ordination or unsuitable shoes being mainly responsible. In other words, with the addition of faulty footwear, the same factors were in operation as those causing the pain-free foot to break down after joining the Army. In this case, however, the weak foot was already painful, the inco-ordinated foot already tending towards deformity, and military life has aggravated the condition.

The problem with which we are faced is thus very similar to that of the normal foot which has broken down during training. As, however, the condition will often have been

a full load, will cause many breakdowns. For example, a recruit who has never marched more than five miles is suddenly sent out for a twenty mile route march. His feet are completely exhausted, but before they have a chance to recover they are called upon for further activities the very next day. The consequence is that his feet never recover. In a case like this acute foot strain results, the feet will be valgus and exceedingly tender, there will probably be spasm of the intrinsic muscles and there may be some swelling. In addition, the condition of the feet will provide a suitable soil for the fibro-sitic module. The patient will complain of aching and throbbing even when at rest. There is only one thing to do; he is utterly exhausted; put him to bed until all pain, spasm and swelling have gone, the only treatment during this period being heat and massage. Strengthening and corrective exercises are then given for several days, the patient still remaining in bed. After this he is allowed to get up, strictly individual treatment is continued, and he is taught to stand and walk correctly. Only when all pain has disappeared and his feet are reasonably strong, is he promoted to the foot class. Later, when he has sufficiently recovered, he is sent for light P.T. and in due course he progresses to full P.T. and strengthening walks. He continues to attend the foot class throughout his stay in hospital, the physiotherapy department looking after his feet, and the sergeant-instructor keeping the rest of his body fit. This course of treatment will take upwards of six to eight weeks, but it is the only certain way of assuring that the recruit will return to his unit with his feet and body sound.

The stiff foot.—After a short time in the Army a stiff foot will become a painful foot. The foot will be called upon to work as it never did in peace-time, and, as it fatigues, adhesions and contractions will prevent the long arch flattening. Crippling pain will result and the soldier will be compelled to go sick. The treatment is eminently satisfactory. After a preliminary rest in bed, manipulation by the orthopaedic surgeon is followed by heat, massage and exercises, still in bed, until bruising or reaction has subsided. Thereafter the soldier gets up and receives individual corrective, mobilizing and strengthening exercises from the masseuse, not being sent on to the foot class until his mechanics and mobility are normal. Subsequently the scheme is identical with that for the weak foot, but the time spent in hospital will be only four to six weeks. The final result will be excellent. Should the foot have become stiff in consequence of assuming an abnormal posture to relieve a painful area, in addition to restoring mobility, it will, of course, be necessary to deal with the primary focus.

The inco-ordinated foot. The foot which is deficient in control of individual muscles, which is unable to dissociate synergists or to synchronize contraction and relaxation of opposing muscles will, under the stress of military training, become still more inco-ordinated, and this may ultimately result in the development of deformity. This will present a fairly constant picture; the foot will be in valgus, the transverse arch dropped, the toes clawed, and there may be some pronation of the fore-foot. At first glance the condition may be suggestive of spasmodic valgus, but examination will show that this is not the case, as peroneal spasm is totally absent. Pain will first be complained of under the metatarsal heads, later in the mid-tarsal joints and under the external malleolus.

We meet this type of deformity at every stage in its development, depending upon when the pain became sufficiently severe to cause the soldier to report sick. In an early case we notice that when the patient either stands or walks, his toes stick out straight ahead and may point upwards, because of excessive action of the long extensors, and that he is often unable to plantar-grade them. In fact, the appearance may give the impression of an hysterical foot. This state of affairs, if left untreated, will progress to clawing of the toes, which will occur when the lumbricals give up the unequal struggle. Ultimately, contractions in the long extensor tendons will complete the picture. The result will be best described as "pes valgus with claw toes." The great toe almost always escapes.

If we examine the recruit's foot in the recumbent position, we note the shape and the inability of the foot to relax. The patient would appear to be actively resisting, but experience suggests that this is not the case, the rigidity of the foot being symptomatic of its inco-ordinated condition. With patience, total relaxation can be obtained and the foot passively restored to its normal shape.

One sees a large number of soldiers with varying degrees of this deformity, and many of them tell you that their feet were normal prior to joining the Army. It took me a long time to realize that they were speaking the truth; I could not imagine such a degree

of his pain was such that it was decided to immobilize him on a plaster bed. The pain grew worse, however, and at the end of September he suffered severe attacks coming on in spasms, lasting about ten minutes, of such acuteness that his pulse became feeble and his complexion paled. During the worst of these attacks, his abdominal muscles were rigidly in spasm.

X-rays taken on October 10 showed collapse of the intervertebral disc between the 4th and 5th lumbar vertebrae with absorption of the adjacent portions of the vertebral bodies.



FIG. 1.—Antero-posterior view showing diminution of joint space between L.4 and L.5.



FIG. 2.—Lateral view showing (a) diminution of joint space; (b) herniation of disc substance into the vertebral body of L.4; and (c) a large osteophyte growing from the margin of the body of L.4.

Since the end of September his condition has steadily improved. During his stay in hospital he has been afebrile, his blood sedimentation rate is 8 mm. in one hour, and his blood-count normal. The process appears to be aseptic degeneration.

I am grateful to Mr. Stamm and to Dr. Campbell, the Medical Superintendent of the hospital, for permission to publish this case.

The Technique of Arthroplasty

By T. T. STAMM, F.R.C.S.

It is not the purpose of this paper to suggest any new method in the technique of arthroplasty, but to emphasize what the older surgeons clearly realized, namely, that the essential structures of the new joint—articular surfaces, synovial membrane, &c.—are formed, not at the time of the operation, but during the subsequent period of treatment. Arthroplasty is not an operation, it is a mode of treatment, the first stage of which consists in an operation for arthralgia.

Most surgeons have had the opportunity of exploring a false joint which has formed

of fairly long standing, additional measures may be necessary, and in the presence of deformities surgical intervention will be called for. With these exceptions, the scheme of treatment is identical with that for the foot which was trouble-free before joining the Army, and the results should be equally good.

As regards deformities, after the orthopaedic surgeon has corrected the hammer-toe or over-riding little toe, after he has dealt with claw toes by tenotomy or, in selected cases, by arthrodesis of the phalanges, after the chiropodist has excised all corns and callosities, then the stage is set for intelligent physiotherapy to play its part in returning the recruit to duty, his feet in better shape than ever before. This type of case offers plenty of scope for collaboration between the orthopaedic surgeon and the physiotherapist, and, if sufficient time is allowed, the results can be brilliant.

Early hallux rigidus, in which bony changes are absent, can often be improved by treatment with hot wax, gently increased passive stretchings, and gently progressed resisted and active exercises, while any degree of elevatus of the first metatarsal is corrected by training the peroneus longus to plantar-grade the foot.

Fibrositis, which usually occurs in the plantar fascia, should be treated by infra-red and massage to localize the nodule, after which the injection of 1% procaine usually effects a cure.

The majority of cases of the types which I have just described should, after a full course of physiotherapy, be able to resume their military training in their original grade. Of course, we are bound to meet with disappointments, and among these will be the valgus foot secondary to a short tendo Achillis. If this shortening is considerable, neither raising the heel nor concentrated physiotherapy will avail, and regrading to a lower category will be necessary.

SUMMARY

There is no place in the Army for the soldier with painful feet, and our task is to return him to his unit in the shortest possible time with his feet so sound that there is no likelihood of their breaking down again. This can only be achieved by strict attention to the following points:

(1) The soldier with painful feet is suffering from a major disability which must be treated as such, and he must therefore be admitted to hospital. Out-patient treatment is unsatisfactory.

(2) In most cases preliminary rest in bed will be essential.

(3) Individual treatment is absolutely necessary until all mechanical errors are corrected.

(4) Sufficient time must be allowed in hospital for the soldier's feet to recover. This will take four to eight weeks. Allowing insufficient time will only result in failure.

(5) Full and intelligent co-operation from the patient is required.

(6) When up and about, Army boots should be worn whenever possible. "Stopping about" in plimsolls is harmful.

(7) The regimental M.O. should never regard a painful foot as a trivial complaint. He must realize that the sooner a case receives treatment, the greater the prospect of a satisfactory result.

A Case of Degeneration of the Intervertebral Disc Following Lumbar Puncture

By P. G. Epps, M.B.

THIS case is very similar to those described at the last meeting by Mr. Everett (see Sect. Orthop., p. 208 this issue). The patient is a soldier, aged 27 years, who in May 1941 developed meningitis. During the course of treatment he had five lumbar punctures, the last of which was about July 25; this one took thirty-five minutes, and the patient describes pains shooting down each leg as repeated attempts were made to pierce the theca. At the beginning of August he was discharged, and went on leave; he had no pain at all then, but after fourteen days he developed, quite suddenly, a severe lower back pain, which he said caused him to collapse in about three days, and he was taken to hospital.

X-rays taken then, on August 25, showed no abnormality of the spine. The severity

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released, and the patient is encouraged to assist the movements actively. The new joint must not, however, be permitted to bear any load or strain at all. In this way the movements of the two joint surfaces on one another will mould the layer of young fibrous tissue on each articular surface before it has become too firm, so that two smooth and accurately fitting surfaces are formed. Traction is, however, still maintained in the intervening periods, to prevent the fibrous capsule, which is forming around the joint, from contracting.

After about six to eight weeks it may be assumed that this fibrous tissue is sufficiently mature to show little further tendency to contract, beyond the amount that can be prevented by gradually increasing periods of active, assisted exercises. The traction may now be discontinued tentatively, but if the range of movement should show any tendency to diminish, it must be recommenced at once.

The condition of the joint should now be as indicated in fig. 1, iv. The bone-ends are covered by a layer of smooth dense white fibrous tissue, closely simulating articular cartilage. The fibrous capsule is lax, permitting free movements, and the whole cavity of the joint is lined by synovial membrane secreting a glairy lubricating fluid, similar to that found in an adventitious bursa.

The joint is now fully formed, but is not fit to bear full body-weight or to take any great strain for at least another three months, since the internal structure of the bones themselves has to be remodelled to be able to withstand the altered stresses.

This method of after-treatment may be summarized and tabulated as shown in fig. 2.

| | | | |
|---------------|---|-------------------------------------------------------|------------------------------------------------------|
| First month | { | 1- 2 days. | Immobilization without traction |
| | | 2- 7 days. | Immobilization. Traction gradually applied |
| | | 7-14 days. | Immobilization with full traction |
| | | 14-28 days. | Daily movements commenced, traction being maintained |
| Second month: | | Traction released daily for active assisted movements | |
| Third month: | | Traction discarded. Daily active assisted exercises | |

NOTE.—Times suggested apply to the larger joints. In small joints these times may be considerably reduced.

FIG. 2.—After-treatment for arthroplasty.

The optimum time to be allowed for each step in treatment should, of course, correspond with the average times which it takes for the process of organization of blood into fibrous tissue to reach its various stages. The times given here are those which seem to be in keeping with what we know of this process, and they work out satisfactorily in practice. In the case of small joints where the amount of tissue to be organized is less, the times may be reduced without ill-effect. Once the initial reaction to the operation has subsided, the remainder of the treatment should, in most cases, be painless. Any complaint of pain by a reasonable patient is usually a sign that something is wrong.

A successful result depends entirely upon the most careful attention to detail and every step must be carried out either by the surgeon himself or by an assistant who understands the factors involved.

If after-treatment on these lines is to be followed, the operation itself should be as simple as possible. It would be irrational to interpose flaps of fascia or fatty tissues between the bone-ends, as is sometimes advocated. A layer of devitalized tissue of this nature could not possibly be expected to form a good weight-bearing surface; on the other hand it would be certain to interfere seriously with the proper development of the joint. The idea that it prevents the new joint from seizing up again is quite erroneous. With the technique of arthroplasty already described there is no difficulty whatever in preventing reunion occurring between the articular surfaces themselves. Failure to secure a reasonable range of movement is almost invariably due to fibrosis occurring around the joint. The interposition of fascial flaps cannot prevent this occurring—in fact, the reaction to its presence by the surrounding tissues might rather be expected to increase this tendency.

The operation itself should therefore consist in a simple arthrolysis, the bone-ends being carefully reshaped, and in the removal of any surrounding scar tissue or contracted ligaments that might interfere with movement.

The use of metal cups is, I believe, equally fallacious, if they are employed with the

spontaneously following a fracture. It is found that the bone-ends are covered by a smooth glistening layer of dense white fibrous tissue, which forms an excellent bearing surface, while the surrounding fibrous capsule is lined by a very close imitation of synovial membrane, which does in fact secrete a glairy lubricating fluid. These structures can only have been formed by the organization of the blood-clot which lay between the broken bone-ends. The aim of the after-treatment in a case of arthroplasty should therefore be to endeavour to reproduce the conditions under which the blood-clot will tend to be organized in this way.

In the absence of direct confirmation by experiments on animals, the following scheme appears to me to be rational, and has in practice given good results.

The first essential is to limit the amount of blood-clot that is allowed to collect between the new joint surfaces and in the rest of the wound area. At the end of the operation, therefore, a firm dressing is applied, and the limb is immobilized without applying any traction. I believe this is a most important point. If there is much oozing from the bone-ends, a small drain may be left in for twelve hours to allow the blood to escape.

After forty-eight hours, traction is applied gradually, the maximum pull being achieved in three to four days. In this way the joint surfaces are slowly drawn apart without any fresh bleeding being caused. A layer of blood-clot may be expected to adhere to each of the rough bone surfaces, and as the gap between the bones widens, a space will appear between the two portions of the clot. This space will fill up with tissue fluid as it forms, provided that no fresh bleeding has been caused. The condition of the parts is now as depicted in fig. 1, n.

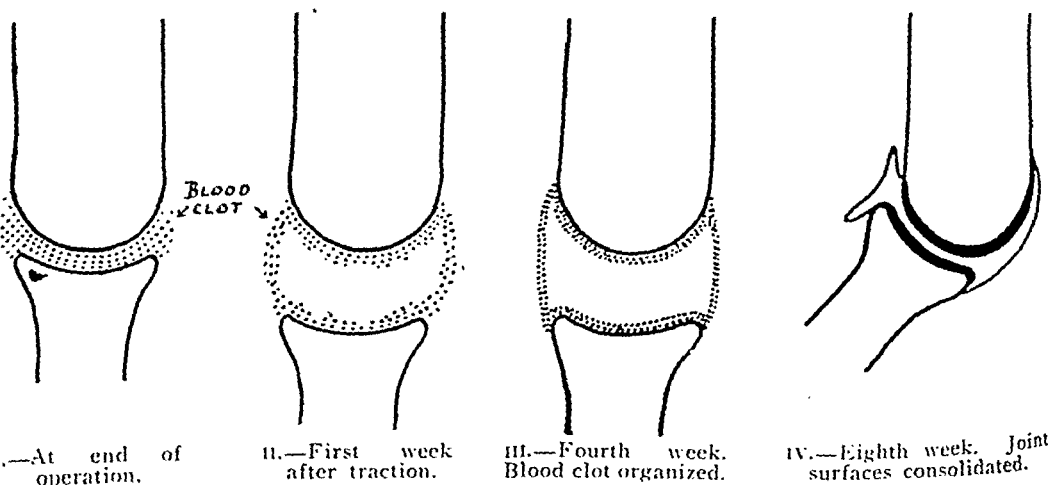


FIG. 1.

If traction is applied immediately after the conclusion of the operation, or is applied too rapidly, the whole of the space between the bone-ends will become filled with blood-clot, and its organization will be likely to lead to a fibrous, if not a bony, ankylosis.

Full traction is maintained for fourteen days before any movements are permitted. By the end of this period the clot will have become firmly adherent and will be commencing to be organized on its deep or bone surface. One movement daily in each direction may now be given, while traction is maintained manually, since there is now little risk of causing fresh bleeding, provided that the two surfaces are kept apart. These movements help to prevent the surrounding structures from becoming adherent. With this method of after-treatment, the full range of movement can often be attained easily and without pain on the first occasion that the joint is moved, though usually it is not wise to attempt so much.

After a further fourteen days it may be assumed that the clot has been organized into young fibrous tissue. A few movements daily may now be given with the traction

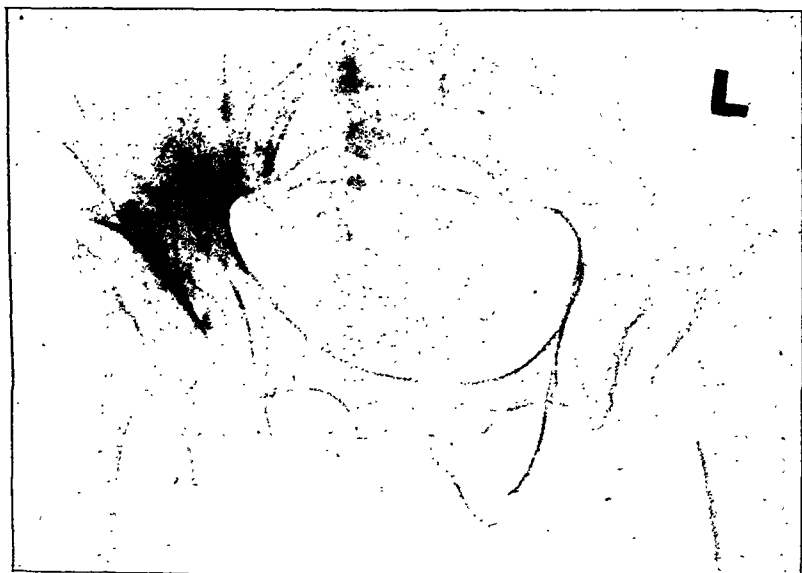


FIG. 1.



FIG. 2.



FIG. 3.

CASE OF ANKYLOSING SPONDYLITIS.

Fig. 1 is a recent X-ray photograph of both hips, and figs. 2 and 3 show the patient sitting and standing.

object of preventing reunion of the bone-ends, for, again, this can be prevented without their employment, while they certainly do nothing to minimize the surrounding fibrosis.

I am not suggesting, however, that they may not prove to serve a useful function in certain arthroplasties. It appears to me that their function is twofold. First, to increase the area of the weight-bearing surface of the joint, when the bones themselves do not provide sufficient material for the purpose, and second, to distribute the weight borne by the joint more evenly over the whole articular surface, and so prevent excessive pressure being borne by any one part of it. I have tried to illustrate this in fig. 3.

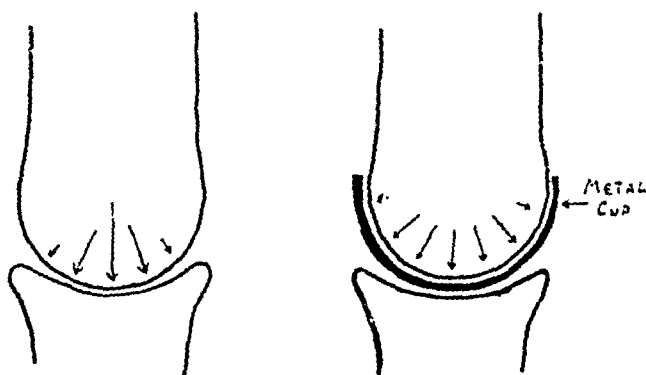


FIG. 3.—To show better distribution of pressure over articular surface afforded by use of metal cup.

I believe that treatment on the lines I have indicated would give a satisfactory result in every properly selected case if it were possible to control the extent of the surrounding fibrosis. While this method of treatment does, I think, tend to minimize the amount of fibrosis, this still remains a definite problem. It is proposed to experiment now with the use of deep X-ray therapy in small doses, on the analogy of its action on keloid scars, but an extended trial will, of course, be necessary before any conclusions can be drawn.

Excision of Both Femoral Heads in a Case of Ankylosing Spondylitis.—J. S. BATCHELOR. F.R.C.S.

S. H., male, aged 35 years.

This patient was first seen in 1938. His condition was unenviable, for both hips, the sacro-iliac joints and the lumbo-dorsal spine were ankylosed by bone as a result of ankylosing spondylitis which had commenced some years previously.

In March 1938 the head and neck of the right femur were freely removed. After-treatment consisted of strapping extension, with the leg at first in a Thomas's knee splint. At the end of the first fortnight hip movements were commenced; after eight weeks the splint was removed, and hip and knee movements were performed with the assistance of a sling beneath the knee. The patient left hospital at the end of fourteen weeks wearing a weight-relieving caliper with a moulded bucket, which was worn continuously for four months and then discarded during the next two months.

Although his condition was much improved and he could walk moderately well, the ankylosis of the left hip and spine made sitting impossible. Accordingly, in October 1938 the left femoral head and neck were excised, the after-treatment being the same as with the first operation.

He now has 90 degrees flexion, 25 to 30 degrees abduction, and a few degrees of rotation in each hip. All movements are painless. He gets about well with two sticks, sits comfortably in a chair and goes by car every day to work at an office. He has completely discarded the calipers, and, although there is obviously no real stability at the hips, there has been no appreciable increase in shortening during the past two years.

made to the thigh sling, and the weight gradually reduced as complete reduction is obtained.

Daily quadriceps contractions are begun, but it is important that these should be given carefully during the first three or four weeks and until there is evidence of callus union.

At the end of six to eight weeks, if union is taking place satisfactorily, it is my practice to remove the pin or wire and apply strapping extension to the leg as far as the knee. Active knee movements over a limited range may now be begun. When the leg is taken out of the Thomas's splint, at the end of ten to fourteen weeks, active knee movements are practised in bed; in the majority of cases flexion to a right angle is obtained in three to four weeks.

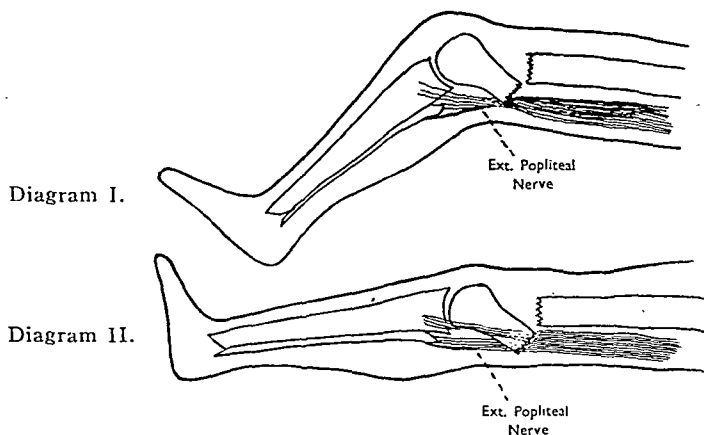
SUPRA-CONDYLAR FRACTURES

These fractures, which are notoriously difficult to treat, form a separate problem. The characteristic deformity of the distal fragment is generally assumed to be due to the pull of the gastrocnemius muscle, and it is customary to treat these cases with the knee flexed. My limited experience has not convinced me that this position facilitates reduction.

I endeavour to lift the flexed lower fragment forward with a sling stretched between the bars of a Thomas's splint. There are two reasons why the flexed position of the knee hinders, rather than assists, this manœuvre.

Firstly, the length of the distal fragment available for leverage purposes is reduced by the backward gliding of the head of the tibia on the femur; and secondly, the depth of soft tissue below the lower fragment is increased by the relaxation of the knee flexors. Both these factors make it difficult to get a satisfactory purchase with a sling beneath the distal fragment.

Further, flexion of the knee brings the external popliteal nerve into a relatively more superficial position and makes it vulnerable to pressure by the sling. In a small number of cases treated with the knee flexed I have found it extremely difficult to get a satisfactory position, whilst pressure sores beneath the lower fragment, and damage to the external popliteal nerve, have occurred too frequently (Diagram I).



I now treat all these cases in the same position as that used for the routine treatment of fractures of the femoral shaft. When the leg is splinted with the knee extended I have not found that the pull of the gastrocnemius presents any obstacle to reduction, whilst it is easier to get a satisfactory lift below the lower fragment, with less risk of pressure sores and damage to the external popliteal nerve (Diagram II). There is also the additional advantage that the knee is ready for a caliper immediately union has taken place. In a small number of cases treated by this method satisfactory results have been obtained.

Treatment of Fractures of the Shaft of the Femur.—J. S. BATHFLOP, F.R.C.S.

During recent years it has become a common practice to treat fractures of the shaft of the femur by means of skeletal traction through the lower end of the femur or upper end of the tibia, with the limb in a position of flexion at the knee.

I believe that this method is unsound and is responsible for many indifferent results.

There is no better splint for the routine treatment of fractures of the shaft of the femur than the Thomas's knee bed splint. This splint, when properly used with the leg straight, not only provides excellent immobilization, but also assists in the correction of deformities of alignment. With the knee flexed, these advantages are largely lost. The splint now acts merely as a cradle for the limb and its action in correcting axial deformities is much reduced.

Skeletal traction around the knee is open to many serious objections. A pin or wire through the lower end of the femur or upper end of the tibia passes through bone largely cancellous in nature, well covered with soft parts, and in close proximity to the synovial reflections of the knee.

These factors increase the risks of infection, and I have seen septic arthritis of the knee following traction in both situations, whilst delay in regaining movement at the knee and frequently permanent limitation of flexion are not uncommon complications of supracondylar skeletal traction. I think it is fair to say that in a straightforward case of fracture of the shaft of the femur skeletal traction through the lower end of the femur is completely unjustifiable.

When these fractures are treated with the leg straight it is possible to employ traction through the lower end of the tibia, and, in my opinion, this is a relatively safe area. Here the bone is largely compact and thinly covered with soft parts, and the synovial reflections of the ankle-joint are not involved.

I have encountered other minor difficulties when treating these fractures with the knee flexed. The pull on the shaft of the femur from a pin through the upper end of the tibia is indirect, and part of the force acts on the head of the tibia, pulling it forwards in relation to the lower end of the femur. If heavy or prolonged traction is used, this action may damage the ligaments of the knee and is a potent cause of knee stiffness.

With traction through the femur it is not uncommon for the lower fragment to be angled forwards; when this occurs it may be difficult to correct the deformity without transferring traction to a lower level.

In the majority of simple fractures of the shaft of the femur there is good callus union at the end of ten to fourteen weeks and rapid consolidation may now be produced by getting the patient up in a walking caliper. When the leg has been treated with the knee flexed, there may be some difficulty in getting full extension at the knee, and this delays the fitting of the caliper. This is a considerable disadvantage, for, quite apart from lengthening the period spent in bed, the stiff flexed knee throws a considerable strain on the fracture; and in the interval between taking the knee out of the Thomas's splint and fitting the caliper, whilst the patient is lying in bed exercising the leg, re-fracture may take place. This happened in two of my cases.

These difficulties with the flexed leg and with skeletal traction around the knee have led me to abandon this method of treatment, and I now treat all fractures of the shaft of the femur with the leg straight, with traction through the lower end of the tibia.

To get the best results it is essential for the surgeon himself to supervise the details of treatment. The initial reduction should be performed in the operating theatre. After insertion of the pin or wire through the lower end of the tibia, about three fingerbreadths above the tip of the internal malleolus, a Thomas's splint with a well-fitting ring is slipped over the leg, and manual traction is applied until approximately full length is obtained. The leg is then held in fixed extension by tying the stirrup to the end of the splint. A sling is placed below the site of fracture and, having been pulled tight so as to maintain the anterior bowing of the femur, fixed with two pins. This is the only sling that should be placed below the thigh. The leg below the knee is supported by slings fixed with clips, the knee being slightly flexed. The foot is held at a right angle with a footpiece attached to the splint. In the ward the splint may be slung from a Balkan beam, and a weight of 20 to 25 lb. is attached to the end of the splint over a pulley.

X-ray examination is carried out on the following day: any necessary adjustments are

made to the thigh sling, and the weight gradually reduced as complete reduction is obtained.

Daily quadriceps contractions are begun, but it is important that these should be given carefully during the first three or four weeks and until there is evidence of callus union.

At the end of six to eight weeks, if union is taking place satisfactorily, it is my practice to remove the pin or wire and apply strapping extension to the leg as far as the knee. Active knee movements over a limited range may now be begun. When the leg is taken out of the Thomas's splint, at the end of ten to fourteen weeks, active knee movements are practised in bed; in the majority of cases flexion to a right angle is obtained in three to four weeks.

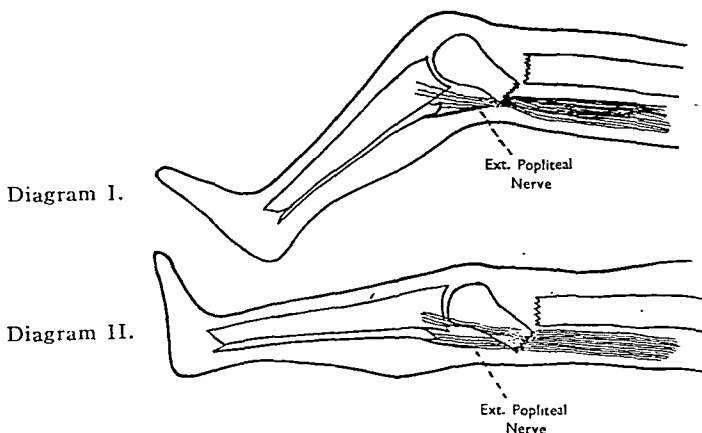
SUPRA-CONDYLAR FRACTURES

These fractures, which are notoriously difficult to treat, form a separate problem. The characteristic deformity of the distal fragment is generally assumed to be due to the pull of the gastrocnemius muscle, and it is customary to treat these cases with the knee flexed. My limited experience has not convinced me that this position facilitates reduction.

I endeavour to lift the flexed lower fragment forward with a sling stretched between the bars of a Thomas's splint. There are two reasons why the flexed position of the knee hinders, rather than assists, this manœuvre.

Firstly, the length of the distal fragment available for leverage purposes is reduced by the backward gliding of the head of the tibia on the femur; and secondly, the depth of soft tissue below the lower fragment is increased by the relaxation of the knee flexors. Both these factors make it difficult to get a satisfactory purchase with a sling beneath the distal fragment.

Further, flexion of the knee brings the external popliteal nerve into a relatively more superficial position and makes it vulnerable to pressure by the sling. In a small number of cases treated with the knee flexed I have found it extremely difficult to get a satisfactory position, whilst pressure sores beneath the lower fragment, and damage to the external popliteal nerve, have occurred too frequently (Diagram I).



I now treat all these cases in the same position as that used for the routine treatment of fractures of the femoral shaft. When the leg is splinted with the knee extended I have not found that the pull of the gastrocnemius presents any obstacle to reduction, whilst it is easier to get a satisfactory lift below the lower fragment, with less risk of pressure sores and damage to the external popliteal nerve (Diagram II). There is also the additional advantage that the knee is ready for a caliper immediately union has been obtained.

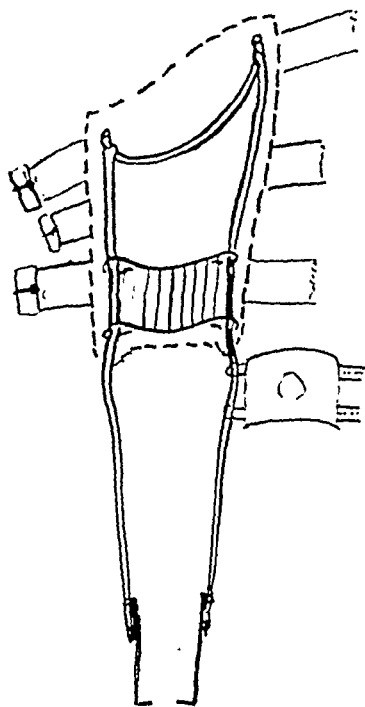
A New Walking Caliper

By W. H. GERVIS, F.R.C.S.

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A wide plaster slab is then moulded on the posterior surface of the patient's thigh and the tuber ischii. The caliper is then placed in position over this slab and incorporated into it with several turns of plaster bandages. When set the plaster over the front of the thigh is cut away, the edges trimmed and 2-in. webbing straps incorporated.



The drawing shows the caliper with front half of ring removed, Cramer's wire in position, usual knee straps retained. Dotted line represents plaster of Paris moulded to fit posterior surface of thigh, with straps in position.

The caliper fits into the heel of the boot in the ordinary way, and is kept in position by the straps. It is weight-relieving and, in addition, supports the shaft of the femur when the patient is sitting. It can, of course, be removed for knee exercises.

Section of Ophthalmology

President—A. J. BALLANTYNE, M.D.

[September 26, 1941]

A Study of Mustard Gas Lesions of the Eyes of Rabbits and Men By IDA MANN, D.Sc., F.R.C.S., and B. D. PULLINGER, M.D.

MUSTARD GAS (dichlorodiethylsulphide) was first used by the Germans at Ypres in 1917. At the time, the large number of casualties, the urgency of the situation and the absence of facilities for the minute examination of the eyes combined to account for the relatively rare and incomplete studies of the ocular lesion published during and just after the war of 1914-18. Interest in the subject dropped though occasional case-histories and speculations as to after-effects appeared between 1920 and 1935, but after that period interest again revived as it became clear that slightly mysterious cases of recurrent corneal ulceration were occurring among men subjected to relatively heavy doses of mustard gas ten to twenty years previously. The attention of one of us was first called to these cases of delayed mustard gas keratitis by Mr. T. J. Phillips, who collected a large number (70) at Moorfields with carefully recorded case-histories and detailed clinical and slit-lamp investigations (*Proc. Roy. Soc. Med.*, 1940, 33, 229 (Sect. Ophthal., 5)). It was then apparent that a large gap existed in our knowledge of the progress of the condition. No records were known of the state of the eyes between the initial stage at the time of exposure and the subsequent onset of recurrent ulceration years after. The patients stated that they had been practically symptom-free during the interval, had continued in ordinary work and apparently suffered no visual disability. From the onset of the delayed keratitis, however, they experienced rapid deterioration of sight.

Since the subject of prophylaxis and treatment of mustard gas lesions of the eyes had again assumed great importance and since improved methods of examination (e.g. introduction of the slit-lamp) and facilities for experiment were available we decided to undertake an inquiry into the clinical pathology of the lesion based in the first place on animal experiment and secondly on laboratory accident cases.

The work falls into two sections, firstly, experiments on rabbits carried out at the Imperial Cancer Research Fund Laboratory by the kindness of the Council and the Director, Dr. W. E. Gye, and secondly, the correlation of the results of animal experiments with recent and late accident and war cases studied by one of us at the Royal London Ophthalmic (Moorfields) Hospital, and at various experimental laboratories where mustard gas is being handled at the present time.

PART I

THE PATHOLOGICAL EFFECTS OF MUSTARD GAS ON THE EYES OF RABBITS

A strain of crossed Dutch rabbits with blue irides and a ring of pigmented conjunctival epithelium at the limbus is used for preference. Brown-eyed and albino rabbits are less suitable for observations of vascular changes in the iris and of epithelial healing.

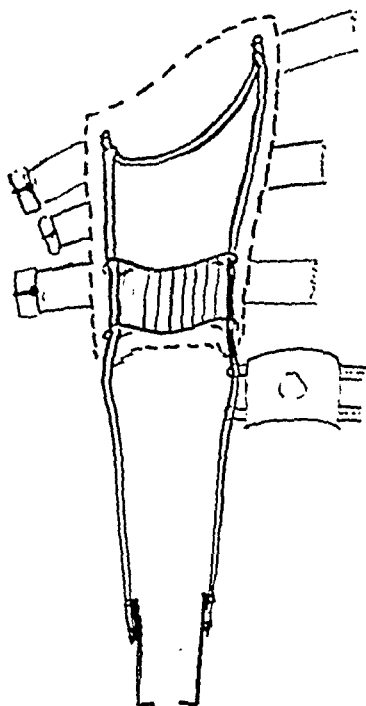
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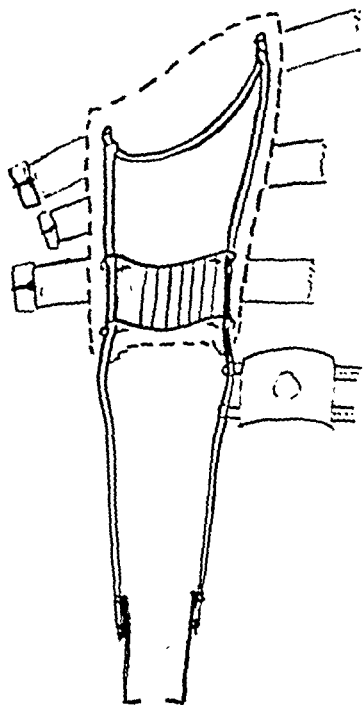
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Observations.—*Day of application:* The liquid mustard tends to slide unless the centre of the cornea is maintained in an upward facing position for four minutes. After four minutes there is practically no tendency to slide. The small droplet is at first hemispherical and projects from the surface of the cornea. After about a minute it often changes its shape, puts out pseudopodia and begins to slide. From observations on sliding droplets it is possible to say that contact for two to three seconds is sufficient to destroy the epithelium. If a droplet remains in contact for longer than this it begins to penetrate the substantia propria. It reaches Descemet's membrane and endothelium in two minutes. In 6.5 minutes (average) it has passed through the cornea and the aqueous and has reached the iris where it produces localized hyperæmia. In most cases the hyperæmia gradually spreads to involve the whole iris. In four minutes none of the droplet remains on the surface. If droplets be placed over a sector of the iris and not in the centre of the cornea the small radial vessels running from the major circle towards the pupil will be seen to flush first (fig. 1, Plate I). After a few minutes more the larger vessels running peripherally from the major circle dilate also. This flushing of the iris may persist as an iritis lasting some days accompanied by aqueous flare and occasionally there is a true exudate in the anterior chamber.

As the droplets soak into the cornea they appear to dig little pits in the epithelium into which they gradually sink. Abundant clear or milky tears¹ are poured out immediately.

If fluorescein is dropped into the eye within a few minutes of the application of the mustard there will be no staining. In a quarter to half an hour the points of application are stained, but nothing else. As the effect of the mustard spreads the stained areas increase so that at twenty-four hours there is a large central stain embracing all the spots of the application. This may increase further up to forty-eight hours, though it usually begins to heal at about thirty hours.

There is no immediate change in transparency of the cornea and in unanæsthetized rabbits the application does not appear to be at all painful. After some hours the eyes look a little watery and there is some hyperæmia of the conjunctiva. There is no visible change in the substantia propria on the first day. Slit-lamp examination reveals damage to the endothelium which shows a "beaten silver reflex".

After twenty-four hours: The conjunctival hyperæmia has increased slightly, but there is no discharge, beyond occasionally a little dried mucus at the inner canthus. The lids show no abnormality.

The area of staining in the centre of the cornea is circular and includes all the spots of application. Occasionally the exact sites of the droplets can be seen as small greyish dots within the stained area. The stain penetrates the whole thickness of the cornea more rapidly than normal and diffuses into the aqueous in from three to five minutes.² The greyish areas (i.e. the worst affected) often take up the stain less well than the region just surrounding them (fig. 2, Plate I). The area involved therefore shows a central region over which the epithelium is destroyed and within which there are denser grey spots marking the sites of the droplets. The large area of destruction of the epithelium is due probably in part to spread of the mustard by soaking into the tissue in all directions and partly to a vapour effect from evaporation of the droplets during the time in which they are on the surface.

The epithelium surrounding the staining area is œdematous (epithelial bedewing) and if the diameter of the stained area is two-thirds that of the cornea this bedewing will extend to the limbus. Otherwise the edges of the cornea will be normal.

The substantia propria of the cornea is now no longer transparent, but shows a greyish haze in the central region. This is due to œdema which separates the fibres and gives a very characteristic slit-lamp appearance of dark (optically empty) spaces outlined by irregular fluffy whitish partitions (fig. 3, Plate I). This appearance is most conspicuous in the superficial third of the substantia propria. The cornea may be swollen to twice its normal thickness or more in severe cases.

¹ The milky secretion appears to well out from under the third eyelid and is probably Harderian.

² If the epithelium is mechanically removed from a rabbit's cornea and fluorescein dropped in, the stroma is stained half-way through in five minutes, all through in ten minutes and in twelve minutes the endothelium is stained and the colour begins to appear in the aqueous.

Rabbits are suitable since they reproduce lesions almost identical with those seen in man, though they require a larger dose to do so. Their pathological processes are speeded up in proportion to their shorter expectation of life, and since the life of a rabbit is roughly one-tenth that of a man, a result which would take ten years to develop in a man may be obtained in a rabbit within a year.¹ It has thus been possible to study the genesis of delayed keratitis in eighteen months' work.

Rabbits show certain anatomical and physiological differences from man which are useful also from an experimental point of view. Their cornea are relatively insensitive and their blinking reflex is often delayed for twenty minutes. It is therefore possible to keep the eyes open during long periods of observation without introducing complications due to abnormal drying of the cornea. The normal rabbit cornea usually shows a faint punctate staining with fluorescein from minute injuries with foreign bodies and from drying, but the epithelium is resistant and spontaneous ulceration or even conjunctivitis is rare. The possibility of infection is therefore not great and the induced lesions can be studied in their uncomplicated state. The greater area of the cornea relative to the sclerotic in rabbits makes them rather more suitable than is man for the study of corneal lesions and less so of combined conjunctival and corneal lesions. The fact that the vessels corresponding to the major circle of the iris are in rabbits visible through the cornea is also an advantage when observing early stages of iritis, while the palpebral aperture is sufficiently wide to allow of the examination of the limbus all round, which cannot be done in the smaller experimental animals such as guinea-pigs and rats.

We found by experiment that the sequence of events following the application of mustard gas to the eye varies very considerably with the technique of the application and that these variations are dependent on the anatomical site at which the mustard comes in contact with the eye, on the time it remains in contact and therefore on the size of the dose. It was therefore decided that a detailed study of the local lesion produced by a constant dose confined in turn to (a) the cornea, (b) the limbus, and (c) the lids and conjunctiva, would give more information than the application of varying concentrations to the whole eye and adnexa at once, although this also was studied when severe effects were required.

If every precaution is taken to ensure similarity of dose and accuracy of application the results obtained are of great constancy and suggest an inevitable chemical reaction rather than a biological response subject to individual variation. These results will be considered first with reference to anatomical site.

(1) THE UNCOMPLICATED CORNEAL LESION

Observations were made on 33 eyes in this group.

The rabbit is anesthetized with ether and laid on its side. This is necessary on account of the peculiar oily consistency of liquid mustard, which tends to slide about on the eye under the action of gravity and which unless carefully controlled, may not exert its action at the apparent site of application. To see the liquid mustard more easily and thus control it, a small quantity of sudan black is added. This makes it visible on a blue eye and does not modify its action in any way. In the experiments which follow, the mustard gas used was an impure sample of the liquid. The lesions are thus likely to be comparable with those expected in man. The application is made with a glass rod having a small rounded end 0.25 mm. in diameter. The tip is dipped in the liquid mustard gas and applied lightly to the cornea without abrading the epithelium. From three to nine spots of mustard are used, arranged in a circle in the central area. The diameter of this circle should be less than two-thirds the diameter of the whole cornea; otherwise the limbus may become involved by spreading. The lids should be held open for fifteen minutes, either by hand or with a speculum. At the end of fifteen minutes the liquid mustard disappears, leaving a thin film of sudan black on the surface of each spot. This may be removed by touching lightly with filter paper or may be left. The eye is then allowed to close and the rabbit to recover. In some cases the rabbit was not anesthetized and the application did not appear to be painful. The sequence of events was then studied in detail.

¹ This calculation is based on observations made on spontaneous cancer in man and animals and on the induction of cancer by means of carcinogenic agents. It can be shown also that this rough proportionality exists in healing lesions. It has been borne out in the present paper.

of a V with the ends of the two limbs on or near the limbus. The treated eyes were kept open for fifteen minutes after the application, in all cases except those in which the speculum accidentally fell out. In this way a certain variety of lesions was produced; in some cases all the nine spots were effective, in others, some of the spots were not as large as others and in some the third eyelid brushed over the cornea before fifteen minutes, or the lids partially closed. Thus it was possible to study a series of lesions, varying from a pure corneal and corneoscleral lesion to one of moderate severity involving the third lid or the margin of the upper lid as well. A further series of experiments to be dealt with later, was done, using a single measured drop placed on the cornea, the eye being allowed to close immediately, so that the drop spread to the cornea, limbus, ocular and palpebral conjunctiva and the lid margins.

(i) *Sectorial lesion of moderate severity.*—51 eyes were observed in this group. Not all the rabbits were anesthetized. The spots (five to seven) were placed in a V-shaped area at the upper part of the eye, the apex of the V at the centre of the cornea and the two limbs on the corneoscleral junction (see fig. 1, Plate I).

Observations.—At the time of application the eyes water slightly but there does not appear to be pain in the unanesthetized animals. In about six minutes the section of the iris underlying the V becomes hyperæmic, the radial vessels running towards the pupil first and then the larger vessels peripheral to the major circle (fig. 1, Plate I). In ten minutes the conjunctiva at the limbus becomes slightly swollen. In two to three hours there is a small fleck of mucopus at the inner canthus, the conjunctiva in the sector of application is more swollen and the spots stain individually. The hyperæmia of the iris vessels spreads so that the whole iris is engorged in five hours. In six hours there appears to be slight photophobia and the eyes are kept half shut. In seven hours the slit-lamp shows an aqueous flare (=increased albumin content of aqueous). The corneal changes are as described above.

In one day the eyes are open with slightly drooping lids, conjunctivæ are hyperæmic and flakes of mucopus are found at the inner canthi. If this discharge dries on the fur of the lower lid it may produce a mechanical ectropion which disappears on washing the lid. The whole of the sector of application, not only the spots, now stains with fluorescein and also the œdematous conjunctiva at the limbus opposite it. The stain on the conjunctiva is orange, that on the cornea green and shows unusually rapid penetration, apparently due to damage to the stroma (*substantia propria*) allowing the dye to soak through. If the epithelium is mechanically removed from the rabbit's cornea and fluorescein dropped on, the colour begins to appear in the aqueous in twelve minutes, but in the mustard gas injury the fluorescein passes into the aqueous in from three to five minutes.

There is destruction of epithelium in the staining area and bedewing over a wide area but not over the whole cornea. The *substantia propria* in the sector is œdematous (primary œdema) and the spots of application may look denser than the rest as in the central lesion described above. There is the endothelial disturbance previously described or destruction. The pupil may be sluggish and there is hyperæmia of the iris and an aqueous flare. In some cases a gelatinous exudate can be seen coming from the sector of the iris corresponding to the sector of application.

In two days there is still slight mucopurulent discharge and where it sticks to the fur this tends to fall out, but the lids are not glued together, though they may be slightly swollen. The epithelium is beginning to cover the denuded area by sliding from the healthy parts. This can be seen in brown and blue eyed rabbits as the pigmented conjunctiva at the limbus slides over the cornea carrying the pigment with it (fig. 5, Plate II). This pigment slide begins in about forty hours and continues for three days, i.e. until the epithelium can no longer be stained. The new areas of epithelium show bedewing. The *substantia propria* is more œdematous in the sector and just beyond, but the penetration of fluorescein is not quite so rapid. The iritis is subsiding.

The conjunctiva at and between the points of application at the limbus is œdematous, quite white and no blood-vessels can be seen in it. There is usually a line of hæmorrhage at the limbus and sometimes above the pale area. The surrounding conjunctiva is hyperæmic.

In three days the staining area is less and the discharge is clearing up, unless the lids have been accidentally touched when it persists longer. The corneal œdema has increased

The endothelium may be completely destroyed in the centre of the lesion or may show a disturbed reflex like beaten silver.

At two days, the staining area is usually smaller, but the endothelial disturbance and the œdema of the substantia propria are worse. There is a zone of epithelial bedewing around the staining area, and beyond that single hydropic cells can be seen here and there in the epithelium some distance away. These cells, seen as single, bright, shining points are also characteristic of recent lesions of moderate severity in man. There is no discharge.

At three days there is still slight conjunctival hyperœmia, but no discharge and no abnormality of the lids. There may be a small central staining area, but in the smaller lesions this has usually disappeared, and the epithelium is intact though still bedewed. The œdema of the substantia propria is lessening and as it subsides the cornea returns to its normal thickness but not to its normal texture. Instead of its usual homogeneous and slightly speckled appearance it now shows a series of thin shiny curved lines, running in all directions and looking rather like fibrils of floss silk scattered throughout the thickness of the cornea (fig. 4, Plate I). This "silkeness" is best seen in the broad beam of the slit-lamp. In the narrow beam the cross sections of the silky lines show up as scattered bright dots and short, shiny, stratified lines. Silkeness is first seen on the third day in a zone, round the centre of the lesion, where the œdema is subsiding. As this subsidence continues the silkeness increases until the whole of the central area shows it. The endothelial disturbance is still present, but the area of destruction is lessening.

At four days there is no staining of the epithelium and the bedewing and œdema are less. The lesion can be resolved into three concentric zones. The centre shows some œdema of the substantia propria. Surrounding this is an area of "silkeness" which corresponds to the area of endothelial disturbance and outside this again is the outer edge of the area of epithelial bedewing. Thus the largest circle represents the area of damage to the epithelium, with a smaller zone within it in which the substantia propria and the endothelium are altered. The endothelium can be seen regenerating at the edges of the patch of destruction.

The lesion is therefore practically quiescent at four days. The œdema continues to subside and its place is taken by the silky appearance.

At seven days there is no œdema and only slight epithelial bedewing in the centre. The silky change is well developed and the endothelium still shows a disturbed reflex. The eye looks perfectly quiet and normal to macroscopic examination. The silky change if excessive may show as a very faint nebula in certain lights, but is difficult to see without a slit-lamp. It appears to be more or less permanent though in the course of months it becomes less marked. The endothelial disturbance disappears completely in time and is usually gone at eleven days. The epithelial bedewing lasts the longest and may persist in the centre for several weeks (up to five in a nine-spot lesion). In man it lasts much longer and there is some evidence that it may be permanent though very slight. Isolated hydropic cells can be detected for a long time in both man and rabbit, after the eyes look absolutely normal to superficial examination.

The appearance and course of this central lesion affords a striking example of some of the physical and chemical properties of mustard gas. That the liquid penetrates with remarkable rapidity is shown by the fact that it passes through the cornea and aqueous in six and a half minutes. It is, as it were, soaked up by the cornea and spreads widely within it and throughout the deeper structures also until it has become sufficiently diluted or broken down to cease to act. On its passage through the cornea it changes the fibres, and kills epithelium, corneal corpuscles and endothelium. The epithelium and endothelium are replaced by cells from beyond the lesion, but the substantia propria, though permanently altered, is not cast off as a slough, and after a brief period of œdema, returns to its normal thickness though it still shows a pathological change which may be permanent. Since the cornea is not vascular we can thus see the action of the mustard on a tissue uncomplicated by coincident disturbance of circulation, except at the limbus where there is slight hyperœmia. It is interesting to note that even in the absence of capillaries, œdema is a prominent feature of the lesion.

(2) THE LESION INVOLVING THE CORNEOSCLERAL JUNCTION

The lids of some rabbits were held open with a speculum. Other rabbits were anaesthetized. Five to nine droplets were applied to a sector of the cornea in the shape

more than a year in a few rabbits. The hæmolyzed blood disappears as do many of the vessels. Those remaining become fine and regular in arrangement and are often empty. They can be made to fill up temporarily by gently rubbing the eye through the closed lids. Slight changes such as endothelial disturbance, faint pigment speckling and faint epithelial bedewing may persist for three months. Indeed slight epithelial bedewing and Hassl-Henle bodies on the endothelium may remain almost indefinitely. Such an eye, however, does not seem in danger of relapse.

(ii) *Sectorial lesion of greater severity with slight lid involvement.*—Thirty-nine eyes were observed in this group. If nine spots are applied and the limbus and conjunctiva slightly more injured than in the preceding series, as well as possibly a small area of the lid margin or the third lid, the course of the process is lengthened and various further changes occur. These can be classified as: (a) Increased discharge. (b) Recurrent thromboses. (c) Formation of blood islands. (d) Deposition of cholesterin and fat. (e) Persistent œdema, formation of fibrous tissue and cellular infiltrations.

(a) and (b). *Increased discharge and recurrent thromboses.*—The lesion is at first similar to one of moderate severity except that the discharge persists longer, especially if the third lid has been injured. The œdema of the substantia propria, both primary and secondary, involves not only the sector of application, but the whole cornea, and the newly formed vessels are more exuberant. The clearing up of the primary œdema at the end of the first week is not so complete. The condition begins to clear up at about sixty days as before, but instead of steadily subsiding it shows exacerbations about every fortnight after that, with fresh ingrowth of vessels, increase of œdema and thromboses and intracorneal hæmorrhages which begin to clear up again and then recur. These events may continue for six or nine months or more, and then finally cease, the vessels narrowing and the œdema subsiding but probably relapses are always possible since many vessels remain in the cornea.

(c) *Blood islands* (fig. 8, Plate II).—In some lesions similar to those described above the intracorneal hæmorrhages take the form of large blister-like collections of blood, usually in the most superficial layers of the substantia propria. The vessels of supply constrict and disappear but the "blood islands" remain for many months. There appears to be no great stimulus for their removal, such as exists in connective tissues.

(d) *Deposition of cholesterin and fat.*—In some instances of severe vascularization especially if there is one large vessel of supply arborizing freely towards the centre of the cornea, the regression of the vessels at about three to four months may be accompanied by the deposition of cholesterin crystals, first outside the vessel walls and later over the whole area previously occupied by them. As the vessels retrogress the cholesterin is left behind often in the form of a semilunar glistening opacity with its concavity towards the main supply vessel (fig. 9, Plate IV). Fatty degeneration visible with the slit-lamp as opaque, flocculent, white masses may follow the cholesterin and both may be present together. The eye may be fairly quiet after six to seven months. If it be watched, however, it will be seen to break down again in about seven to eight months, the cholesterin apparently working up to the surface, causing ulceration of the epithelium and being cast off with loss of substance so that a small pitted scar is left. The fatty degeneration also may lead to recurrent secondary ulceration. These eyes continually heal and break down, each small ulcer staining for about four days. In some cases, however, the cholesterin is deposited in the deeper layers near Descemet's membrane where it does not cause secondary ulceration and remains unchanged; in others it disappears without ulceration.

(e) *Persistent œdema and formation of fibrous tissue.*—When the eye and lids have been more severely injured and the discharge has been greater and more prolonged, there may be no tendency for the œdema to clear up (fig. 10, Plate IV). The primary œdema may merge with the secondary œdema and the whole cornea may become completely waterlogged and enormously swollen up to twenty times its normal thickness. This may persist, with repeated thromboses and exuberant vascularization for months. Large blister-like collections of fluid form among the superficial fibres of the substantia propria and occasionally though not often between the epithelium and Bowman's membrane. The surface becomes irregular, corrugated and thrown up into ridges and circular blebs. In these the corneal structure is entirely replaced by very vascular fibrous tissue which may invade the larger part of the cornea. These œdematous thickened fibrous corneæ never clear and show a great tendency to secondary ulceration and to surface keratinization. Large

slightly, and the migration of pigmented epithelium has progressed. The œdema is mostly of the superficial third of the cornea.

In four days: As a rule the staining has almost or completely disappeared (as in the central lesion) and the pigment is scattering over a wider area and disappearing. The œdema of the substantia propria is less and is confined to the sector. In the conjunctiva there is increased capillary activity at the margins of the pale area and the limbal loops on either side of it are engorged. The eye on the whole looks better.

In five days there is no stain, practically no discharge and the œdema is much better, though still present in the sector just at the limbus. The conjunctiva is not hyperæmic except just at the limbus at the edges of the pale patch where the limbal loops are active. The iritis has disappeared.

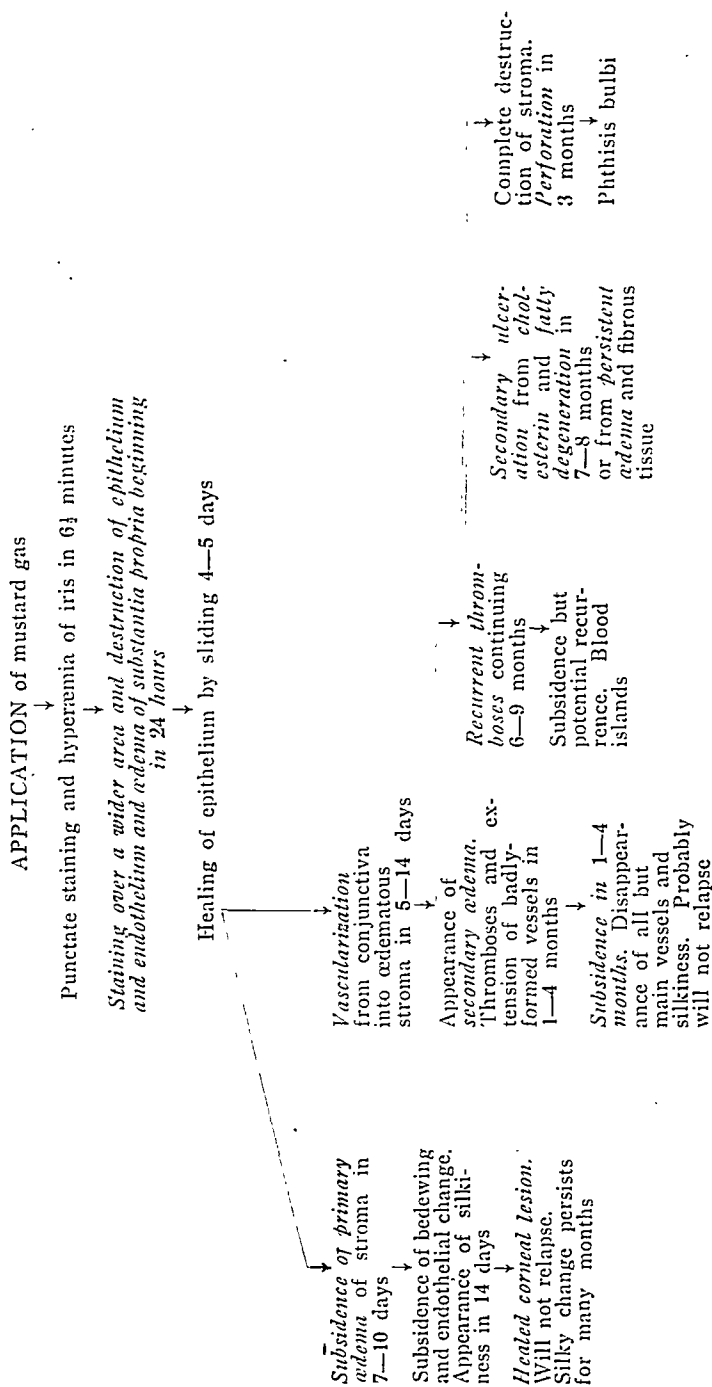
In six days the eye looks macroscopically quite quiet except for a very faint haze above. The œdema (primary œdema) has practically disappeared but the slit-lamp shows great activity of the conjunctival vessels at the edges of the pale area (fig. 6, Plate II). If the eye is observed only until this stage it would be passed as practically cured, and so far the course is that of the pure corneal lesion except for the damage to the vessels at the limbus.

In nine days, however, a marked change for the worse is apparent. The conjunctival vessels are beginning to invade the cornea at the edges of the sector, not only superficially, but also at all levels, most of them being about one-third the way through. These invading vessels are accompanied by a considerable increase in the œdema of the substantia propria (secondary œdema) which just precedes them and spreads through the sector as the vessels advance. The rest of the conjunctiva is not hyperæmic and there is no fresh discharge but the cornea begins to look worse.

In eleven days the invading vessels are well established and the secondary œdema is considerable. The appearance is often like the typical "salmon patch" of interstitial keratitis in man with the difference that although the vessels in the cornea are at varying depths they all originate from the superficial conjunctival vessels and not from scleral branches. The advancing capillaries are peculiar and their distribution characteristic. They grow in as fine straight vessels with tapering ends which do not yet curve round to make a return channel. Close observation with a slit-lamp magnifying 25 times fails to reveal any movement of the blood in these tips which are packed close with corpuscles. The ends often branch at right angles, or right-angled branches occur nearer the source of the vessels. The blood appears to clot in many of the tapering ends; intracorneal hæmorrhages spread out from them; hæmolysed blood can sometimes be seen seeping along the corneal fibres. More often the tapering ends of the vessels become detached. This is usually followed by the formation of a channel for a return supply at the proximal end. Some of the invading vessels have bulbous ends and assume fantastic shapes, detached points, branches and islands of blood being constantly formed, disappearing and reappearing rapidly, the whole pattern varying from day to day, always extending farther into the cornea, and always accompanied by secondary œdema (fig. 7, Plate III). The patterns assumed by these invading vessels suggest that they are growing unchecked into spaces formed by splitting apart by œdema of altered corneal fibres. Their arrangement is not unlike that of the so-called Bowman's canals, an artefact produced by splitting a cornea by forcing air into it, which show the same bulbous ends and right-angled branchings. On the other hand their arrangement often differs entirely from that of blood-vessels grown experimentally in the transparent Sandison-Clark chambers in the rabbit's ear. The vascularization spreads and increases from the eleventh to the thirtieth day or longer. The pale area of conjunctiva (which has been killed) is invaded by regenerating vessels which in turn spread to the cornea, but the first and greatest invasion is from the edges of the sector, through the uninjured limbus. Most of the vessels lie one-third the way through the cornea. After thirty to thirty-five days the supply vessels begin to narrow, the circulation slows, the tapering ends disappear and the remaining vessels narrow down and gradually become more regular. The œdema subsides simultaneously. The pigment line which is broken at the limbus may partially reform. The pigment scattered on the surface of the cornea disappears and the eye begins to improve again. As the corneal œdema subsides the silky appearance mentioned in the pure corneal lesion becomes visible throughout the greater part of the sector.

By about the sixtieth day or less the secondary œdema has all cleared up leaving only the silkiness, which is more or less permanent: that is to say, it has disappeared after

TABLE I.—COURSE OF MUSTARD GAS LESIONS OF RABBIT CORNEA COMPILED FROM OBSERVATIONS ON 138 EYES.



superficial ulcers may occur after about seven months (fig. 11, Plate V). They may take a week or longer to heal and then break down again. They are much larger and more persistent than the small ulcers associated with cholesterol noted above. Some of these cornæ have been observed for nine or ten months and show no tendency to stabilize or resolve.

In some cases the blisters and blebs become filled with pus and indeed in all these severely injured eyes there is persistent discharge. How great a part infection plays in keeping up the tridema is not yet clear, but it seems likely to be of more importance in cases where the lids are injured severely as well as the cornea and limbus.

(3) SEVERE LESION OF THE WHOLE EYE AND LIDS

Fifteen eyes were used in this group. If a large measured drop (approximately 0.0005 c.c.) be placed on the cornea and the eye allowed to close, a generalized lesion of great severity is produced. The reaction is too great to study the early stages in detail. For the first nine days the eyelids and ocular and palpebral conjunctiva are so swollen and oedematous that it is impossible to see the cornea. There is copious discharge which glues the lids together and mats the fur and causes it to fall out over a wide area around the eye. As the swelling decreases and it becomes possible to see the eye, the lids become notched or "ruffled" and everted so that the lower lid especially is not properly in contact with the globe and is constantly covered with discharge.

The cornæ are richly vascularized and excessively oedematous. Necrosis of the cornea may be so extensive that in two and a half to three months the substantia propria will appear to deliquesce and disappear over a large or small area, giving rise to a descemetocoele which eventually ruptures, with collapse of the eye, infection, panophthalmitis and finally phthisis bulbi with a shrunken globe, deformed lids and chronic discharge.

COMMENT

These possibilities can be tabulated (*see Table I*). They correspond very closely, though much condensed in time, with the lesions seen in man, including the "delayed keratitis" still affecting men exposed to mustard gas in the last war. Oedema, persistent epithelial bedewing, nebulae and silkiness, vascularization, recurrent thromboses and intra-corneal hæmorrhages, curiously shaped vessels, blood islands, cholesterol, fat, recurrent ulceration, perforation and phthisis bulbi have all been observed in man so that there seems good ground for supposing that the experimental lesion in rabbits produced by droplets is comparable to the effect of fairly severe exposure to vapour in man.

The majority of these pathological changes may be produced by agents other than mustard gas. The most characteristic signs are the blood islands and the peculiar varicosities seen after subsidence of the acute stage, also the ulceration of the superficial cholesterol deposits in the absence of fat. So far these signs have not been produced in rabbits by any other means, nor observed in man in conditions other than mustard gas keratitis. The diagnosis rests on a combination of the non-specific lesions with the typical vascular and degenerative changes in a definite time relationship and at a definite anatomical site. The lesions in man are described in the next section.

In conclusion we wish to thank Group Captain P. C. Livingston, Flight Lieutenant H. M. Walker, and Messrs. Glaxo for their collaboration in handing over to us their collection of rabbits for a study of the late lesion in their series as well as our own.

PART II

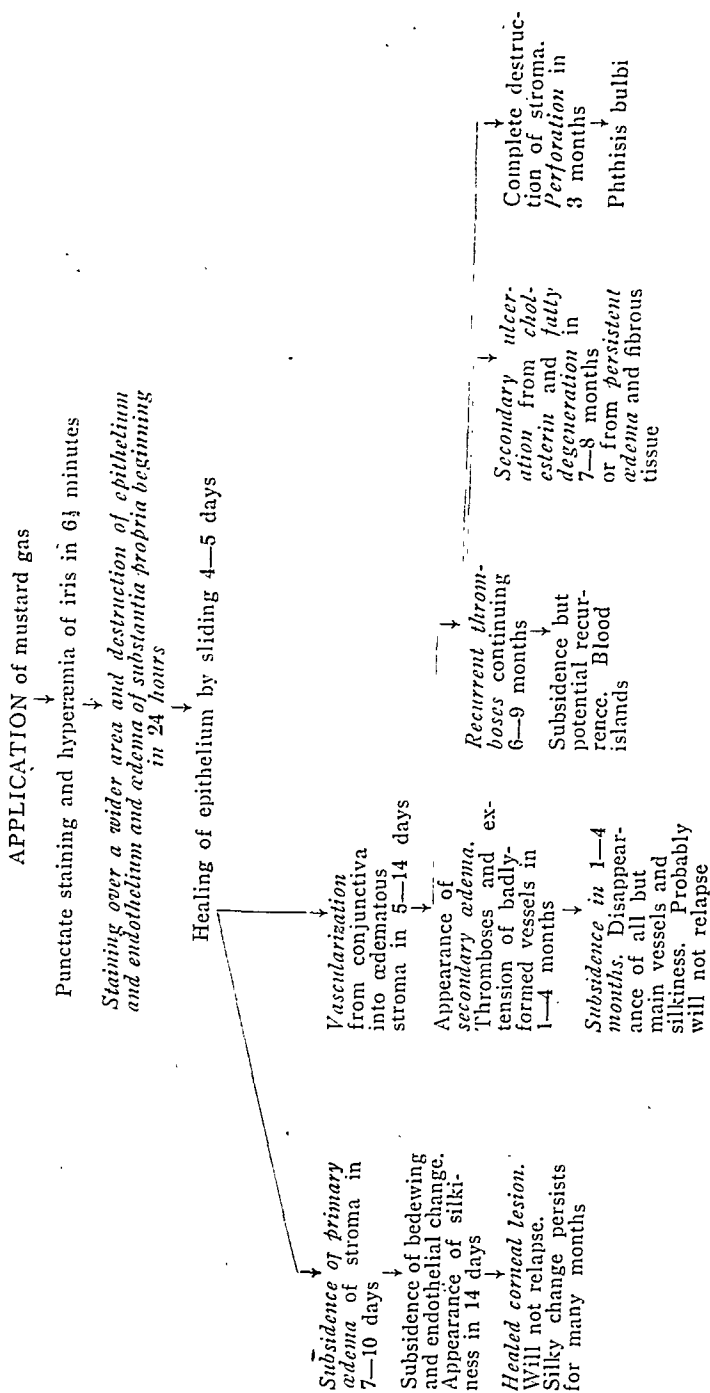
STUDY OF THE CLINICAL PATHOLOGY OF MUSTARD GAS INJURIES TO THE EYES IN MAN AND ITS CORRELATION WITH THAT SEEN IN RABBITS.

By IDA MANN, F.R.C.S.

A sufficient number of cases of mustard gas injuries to the eye in man were available for the correlation of the changes with those in the rabbit. Allowing for slight anatomical differences which modify the appearance somewhat in rabbits, the similarity is remarkably close.

I am deeply indebted to Drs. MacLaren-Ferrie and Chiesman for their valuable help and close co-operation in collecting and observing the recent cases, to the late Mr. Bernard

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Chavasse for allowing me access to his cases, and to Mr. T. J. Phillips for permission to use his cases and detailed unpublished work, on the late stages of the lesion, which has been necessary to complete the picture.

Although observations on the recent accident cases are still continuing and certain gaps exist in the human series, it is possible to say that:

(1) The pathological process is similar in man and the rabbit, the time relationships of the various stages being comparable, taking the accepted standard that a rabbit's life is one-tenth that of a man and its reactions therefore ten times as fast.

(2) That the reaction (acquired sensitivity apart) in man as in the rabbit is dependent on the size of the dose and the anatomical situation.

(3) That the explanation of the "late keratitis" seen in man up to twenty years after exposure has been attained by observation of the lesion in rabbits.

The human cases can be divided into five groups depending on the severity of the lesion.

GROUP 1.—EXTREMELY MILD CASES. CONJUNCTIVA ALONE AFFECTED

Ten cases fell into this group. They were examined at varying times after the accident from nine days to two years. Most of them were wearing either eye shields, chemical goggles or respirator at the time of the accident, which in some cases was a direct splash, in others exposure to vapour. The amount of protection afforded even by the Army eye shield appears to be considerable.

Three of the cases were unaware that they had been exposed until the onset of symptoms. The usual history, however, was that the eyes became slightly bloodshot and the lids slightly swollen a few hours (usually not more than six hours) after known exposure. Complaint was made of a feeling of irritation, but there was very little mention of photophobia and the eyes were never closed. The feeling of soreness lasted usually four to seven days, but the patient did not stop working. In some cases the irritation was said to have lasted some weeks, but nothing could be seen to account for it.

In none of the cases examined in this group was there any corneal abnormality due to mustard gas.

The earliest case seen was at five days. The patient had also had an erythema of the neck which was still just visible. The eyes showed hyperemia of the conjunctivæ but no hæmorrhages or anatomical disturbance of vessels. This was all that was ever seen in cases in this group.

Two of the cases are instructive. They both had a history of a splash and in each case the eye was washed out very efficiently and copiously (in one for twenty minutes) within less than a minute. One used bicarbonate of soda lotion first and then tap water, the other tap water only. In one case the fingers of the man who washed out the eye were burnt as were the patient's lids. In both the lids were very swollen and the eye closed the next day, suggesting a severe lesion, but when the eyes could be opened, in four days, it was found in both that the cornea had escaped completely, although there was intense conjunctival hyperemia. On the thirteenth day, in one patient, the skin of the lids showed hard dry sloughs, but the eye was open and the cornea absolutely normal, in spite of the fact that the lower fornix and lower half of the ocular conjunctiva were intensely hyperemic, making it certain that some mustard (either vapour or liquid) had actually entered the eye. The other case had occurred two years previously and at present the cornea shows no scarring and there is no disturbance of the limbal blood-vessels. In this case also the eyes were closed for four days and the patient was off work for three weeks.

GROUP 2.—MILD CASES. CONJUNCTIVA HYPEREMIC AND CORNEAL EPITHELIUM INJURED

Nine cases fell into this group. The earliest seen was three days after the injury, which was caused by vapour. The first thing noticed was a feeling of soreness in the eyes and throat on waking in the night nine hours after exposure. The left eye was slightly worse than the right. The voice was hoarse. The eyes could be opened but there was photophobia and lacrimation (which were still present at examination). The conjunctivæ were hyperemic and the corneal epithelium in the palpebral aperture was oedematous (epithelial bedewing) and faintly grey and slightly hazy. In the left eye a few minute punctate staining areas could be seen in the oedematous area. (In one case this was still visible at

nine days.) In the right eye a few fine, shining lines could be seen in the centre of Descemet's endothelium and the outer side of the iris was hyperemic. This seemed to show that there was some penetration deep to the epithelium, but there was no œdema of the substantia propria and the condition cleared up completely in both eyes. Three months later both corneæ were normal, with slight conjunctival hyperæmia. The triangular white patches of conjunctiva often described at the limbus are not seen in either Group 1 or 2. They occur only in more severe lesions. The lesion in this patient resembles very mild epithelial damage in the rabbit.

The epithelial bedewing subsides, but before complete restoration to normal there is a stage at which a linear group of isolated shining dots (single hydropic epithelial cells) can be seen with the slit-lamp in the line of the palpebral aperture. In mild cases this is all that may be seen by the fourth day. In slightly more severe cases they may remain visible for from four to six weeks. These may be present even in patients who have not made much complaint. In one case in which the eyes were completely closed for one day the patient was able to return to work in forty-eight hours (though the lids were sore for three days more), but hydropic cells were present in fairly large numbers for three weeks and could just be detected for six weeks.

One case in this group was unable to open the eyes for four to five days, one for one day, while the rest stated that though the lids were swollen the eyes could just be opened.

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The third case was seen eighteen months after the injury. There were no symptoms. The corneæ were normal, but in both eyes there were faint, pale, triangular areas at the limbus (their apices pointing to the inner and outer canthi) in which the limbal capillaries were absent and their place taken by a sparse and irregular arrangement of larger vessels. This appearance is sometimes called "marbling".

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sisting of washing his skin and eyes with bicarbonate of soda lotion (no copious irrigation of the eyes was given) immediately after which liquid paraffin was dropped into the eyes. In five hours the eyes and lids began to burn. Three hours after this, at eight hours, he was admitted to hospital with both lids extremely swollen and the eyes quite closed. The conjunctivae were oedematous and the left was even then obviously worse than the right. In three days the lids could just be opened but the sight was misty in both eyes according to the patient. There was no blistering of the lids and no purulent discharge, only clear tears. Cocaine 2% was given whenever pain became excessive and gave great relief. It did not appear to have any harmful action. The right eye improved quicker than the left.

I saw the patient on the eighth day.

The right eye was open though the lids were swollen and the conjunctiva hyperæmic. There was no stain. There was diffuse epithelial bedewing across the lower part of the cornea with scattered minute white dots, probably single, hydropic, epithelial cells. The limbal blood-vessels were engorged especially below. The endothelium was normal. This lesion belongs to Group 2 and when seen at three weeks was still hyperæmic. At six weeks it was completely normal again.

The left eye on the eighth day was extremely hyperæmic and the conjunctiva in the palpebral aperture and also to a slighter extent in the fornices was oedematous, gelatinous looking and slightly raised up so that it formed a rim round the cornea which appeared depressed below its level. The vessels were violently congested and towards the outer canthus showed some irregularity of calibre. There was punctate staining of the cornea which was greyish and hazy especially in its central and lower part, presenting the characteristic *peau d'orange* appearance of the early severe lesion in man. The corneal epithelium was oedematous, the underlying *substantia propria* showed an altered, more shiny and brighter zone in its anterior one-third (early oedema) with endothelial disturbance and ruckling of Descemet's membrane (fig. 12, Plate V). As the iris was brown, hyperemia could not be made out. At the limbus on either side in the palpebral aperture were several somewhat circular opaque whitish patches, looking a little like small ulcers or sloughs but not staining with fluorescein. They occupied the position of the triangular patches usually described but were different in shape and extended on to the cornea. They probably correspond to the pale area at the limbus in the sector of application in the rabbit experiment. Their appearance thus suggested those seen in rabbits with a lesion involving corneoscleral junction and lids, at twenty-four hours. This is to be expected if ten days for a man is roughly equivalent to one day for a rabbit.

I next saw the patient at three weeks. He was unable to work. The right eye had recovered except for hyperamia but he stated that the left had at first improved greatly (probably corresponding to the healing of the epithelium and subsidence of primary oedema) and five days before had relapsed and he now wanted to keep it shut as there was a feeling of a foreign body all the time, especially under the upper lid.

The conjunctiva was not so swollen but conspicuous changes had occurred in it (Coloured Plate, fig. 2). The opaque areas in the limbal region were now present as areas of disturbance and partial destruction of the limbal capillaries. Above and below them were large subconjunctival hæmorrhages and amongst these were many dilated vessels with thromboses.

The cornea was clearer but was covered with a very large number of minute whitish specks, probably closely set hydropic cells (many of them less shiny and whiter than in the milder cases). In the lower part of the cornea there was oedema of the *substantia propria*, with engorgement of the limbal blood-vessels. With the slit-lamp it could be seen that the texture of the *substantia propria* was altered in its superficial layers (fig. 13, Plate V). On everting the upper lid an oval patch could be seen on the tarsal plate, white, opaque, with no visible blood-vessels and resembling an infarct. A similar patch was present at the limbus on the inner side. Both were surrounded by engorged vessels. They were similar to conjunctival and lid lesions in rabbits.

I next saw the patient at five and a half weeks. He was unable to work and still had the feeling of a foreign body under the upper lid in the region of the necrotic patch,

which remained dead white. The white patch on the ocular conjunctiva was surrounded by hæmorrhages and extended on to the palpebral conjunctiva below (Coloured Plate, fig. 3). The cornea was much clearer. The œdema of the substantia propria had subsided, though there was still bedewing of the epithelium. The stroma showed an altered texture, best described as a faint dappling, in its superficial layers (figs. 14 and 15, Plate V).

This eye appears to be in the condition of a rabbit at four to five days, when the primary œdema is subsiding, the thromboses and hæmorrhages are still present at the limbus, no vessels having yet grown in, and the eye is rapidly quietening. The dappled appearance in man seems to correspond with the silky change in rabbits. The difference is probably due to anatomical differences in arrangement of cells and fibrils.

At eight weeks the patient was back at work, and felt much better. The white patch under the upper lid was smaller and was partially vascularized. Although the man complained of very little the eye appeared rather worse. There was intense engorgement of the ocular conjunctiva around and in the previously seen white patch below and also to the inner side. There was a reappearance of the corneal œdema below and the limbal blood-vessels on either side of the necrotic area were beginning to invade the cornea. There were thromboses at their growing tips and intracorneal hæmorrhages in the superficial layers of the substantia propria (Coloured Plate, fig. 4). The secondary œdema and vascularization were beginning and the condition corresponded to the rabbit at five to seven days. The patient was next seen seven months after the injury. He stated that he had recovered and worked until two weeks previously. At that time the feeling of a "lump under the lid" returned. He was off work. He showed minute hæmorrhages around the necrotic patch under the upper lid. The necrotic patch was completely revascularized and contained globules suggestive of fat. There was also some granulation tissue around it. The cornea was still bedewed and in the area of limbal involvement there were some new hæmorrhages from the extending capillary loops in the cornea and also deep in the conjunctiva (Coloured Plate, fig. 4). This case therefore would appear to be in the stage of recurrent hæmorrhages and probable thrombosis which begins in the rabbit after the first month. This history brings out very well the extreme slowness of the changes in man and the likelihood of mistaking a clinical cure for a true cure since a good deal of the process in the more serious lesions is practically symptomless.

Cases such as this in which vascularization and secondary œdema occur may subside and give rise to no further symptoms (cf. the subsidence in rabbits after one to four months). Three such cases from the last war were examined and although they considered that their eyes had recovered completely they all showed typical changes.

The mildest case seen was gassed in 1917 and recovered after six months. One eye remained symptomless, but the other has had severe ulceration following an injury with sand in 1934. The uninjured eye showed destruction of limbal loops and a few varicosities of the conjunctival vessels in the palpebral aperture. The cornea showed epithelial bedewing, a faint dappling in the superficial layers of the substantia propria, and a disturbance of the endothelium (fig. 16a and b, Plate V).

Two other cases of greater severity but also symptomless were seen. They both showed destruction of limbal capillaries in the white triangular area, varicosities of conjunctival vessels, epithelial bedewing, shiny dots and lines in the substantia propria (fig. 17, Plate V) and also a peculiar appearance, probably diagnostic when it occurs and not so far seen in rabbits. This is usually referred to clinically as "black lines" or "tubes" (fig. 18, Plate V). It is an appearance of branching clefts or spaces running through the substantia propria at all depths and more or less perpendicular to the surface. It does not alter the transparency of the cornea but definitely indicates a deep penetration by mustard gas in the past. It affects only the part of the cornea exposed and not necessarily all of that. It is not present in every case. Sometimes grey lines are seen instead of, or as well as, the "black" clefts. They are not so far explained and probably have no connexion with them. These cases correspond to the healed rabbits with silkiness and the remains of vessels of peculiar shape, examined at nine months.

GROUP 4. VERY SEVERE CASES SHOWING LATE SECONDARY ULCERATION (SO-CALLED DELAYED KERATITIS)

Since we know that the lesion in rabbits continues to show changes for at least nine months or a year and that in the severe cases secondary ulceration (from chlolesterin

deposition and fatty degeneration or persistent edema) may begin in eight to nine months, we should expect to find ulceration in man in about eight to ten years when the slow degenerative changes will have reached the requisite stage. The interim period is symptomless since there is no loss of epithelium and the changes are confined to the substantia propria of the scarred area. There is decreasing vascularization as the vessels recede, leaving behind them the deposit of cholesterol, so that clinically the eye appears to be getting better all the time. A study has been made of 39 cases and case-histories of patients at present attending Moorfields Eye Hospital or the Ministry of Pensions for delayed war gas keratitis, and this, together with Mr. T. J. Phillips' examination of 70 cases and my own observations on the course of the recurrent attacks, completes the picture. Coloured Plate, figs. 5 and 6 show a typical case.

Typical Case of Delayed Keratitis

A patient aged 50 was examined. He gave a history of exposure to gas from a gas shell barrage in July 1917. He put on his gas mask, left the lorry he was driving and went into a dugout for some hours. He was in hospital in France for a month and in England for six months during the whole of which time his eyes were kept bandaged. He had burns on the face and his chest was affected. His eyes gradually recovered except for slight di-like of strong light, but his chest has given trouble ever since.

In 1927 he attended an eye hospital for corneal ulceration, especially in the right eye. The individual ulcers healed quickly, often in a few days, but the irregularity of the surface of the cornea increased after each attack (cf. depressed area in rabbit's eye where cholesterol has been cast off, fig. 9, Plate IV) as there seems to be actual loss of substance when the degenerated area ulcerates. His sight therefore steadily deteriorated and in 1931 he was awarded a pension for his eyes. The recurrent ulceration continued and this year he was seen by Mr. Phillips and fitted with Dallos contact lenses. His visual acuity is R. eye = $\frac{1}{2}$, L. eye = $\frac{1}{2}$ not improved by spectacles. With contact lenses which are well tolerated he is improved to R. eye = $\frac{2}{2}$ partly, L. eye = $\frac{1}{2}$, and so far has had no recurrence of ulceration. He shows in the right eye (which has had most ulcers) cholesterol crystals, fatty degeneration, silkiness (seen partly as dappling and partly as fine short lines, slightly different in texture from that in the rabbit, and probably depending on the slightly different shape and size of the cells and fibres in man) and irregular vessels in process of disappearing together with deposition of cholesterol (cf. rabbit, eight months or more). In the left there is less cholesterol, grey and black lines and alteration of the substantia propria.

The astonishing improvement in visual acuity with contact lenses which he shows is seen in all cases and is due to the substitution of the smooth glass for the irregular and uneven corneal surface as the main refracting interface. The good tolerance of all delayed gas keratitis cases for individually fitted contact lenses is associated with a certain amount of corneal anaesthesia from previous destruction of nerve fibres and the effect of the lenses in preventing recurrent ulceration is probably explained by the protection of the partially insensitive cornea from multiple minute injuries. The same beneficial effect is seen from a tarsorrhaphy, but the contact lens has these great advantages, that it is permanent, transparent and improves visual acuity.

All the cases of delayed ulceration (which should not, in my opinion, be called keratitis as it is not an inflammation but a degenerative ulceration) show these changes. The typical picture is of an eye in which the upper part of the cornea and the limbal loops are normal (fig. 19, Plate VI). Across the centre and lower part runs a band of opacity stretching between the white areas on the conjunctiva in which the limbal loops are absent and the few vessels there are, are badly formed. The scar shows great irregularity of surface. At its edges and in its base are cholesterol crystals. It is vascularized by peculiarly contorted and varicose vessels which enter through the normal limbus above and below the worst scars (fig. 20A and B, Plate VI). There may be narrow constrictions, blood islands, intracorneal haemorrhages and thromboses. The scar shows in addition to the cholesterol, white flocculent masses of fatty degeneration, patches of a fibrillar silky texture and black and grey lines of a peculiar distribution. All these changes except the black lines, have been reproduced in rabbits. The pathology therefore appears as a slow degenerative process. It is initiated by direct chemical action which produces injury and intense edema. These two factors combined allow invasion of the cornea by newly formed blood-vessels which are extremely abnormal in form. They are derived from both normal and injured conjunctival vessels. As the corneal vessels slowly retrogress in the course of years, blood may be left in the substantia propria. Vascular regression is followed by

cholesterin and fatty degeneration which are responsible, alone or combined, for the "delayed keratitis" (see Table II). Although there is said to be a completely quiescent interval of six to sixteen years the eyes show definite diagnostic signs the whole time, and are merely symptomless.

GROUP 5. EXTREMELY SEVERE CASES SHOWING CORNEAL PERFORATION

These cases are very rare in man. I have only seen one which is undoubtedly and have heard of two others. The patient I examined is now aged 55. He stated that he was in a "pill box" in 1917 and a gas shell entered it and exploded within 6 ft. of him, splashing him with the contained liquid mustard. He felt a splash in his face and eyes. He had a burning sensation for a short time but could open his eyes at once and could see for about ten hours afterwards. Intense discomfort and much swelling of the lids then came on and he was unable to open his eyes for ten weeks. When the lids opened he could at first see practically nothing but in the following nine months his sight improved so that he was able to read with glasses. He worked as a gardener but had frequent attacks of discomfort and burning. For the last fifteen years he has had severe attacks of ulceration and a perforation occurred in the left eye.

At the present time the right eye shows a typical cholesterin scarring which is more severe than usual as the whole cornea is involved and not only the exposed portion. The left eye, however, shows a perforation which has healed with inclusion of iris. This is above the centre of the pupil, is small, almost circular and resembles those seen in rabbits except that it has healed, while in the rabbits complete phthisis bulbi usually resulted. The whole of the rest of the cornea is scarred, vascular and irregular. This patient cannot tolerate contact lenses though they improve his visual acuity from counting fingers to $\frac{5}{80}$ in his better eye.

This case completes the series and corresponds with the rabbits in Group 3. It is probable that only a direct splash in man would cause perforation, though, as we have seen, prolonged exposure to vapour will produce nearly as severe a lesion, the difference between the effects of vapour and the effects of a splash of liquid being merely one of degree and situation. It is thus apparent that the difference between the effects of vapour and the effects of a splash of liquid mustard gas is merely one of degree and situation.

All stages linking the two have been seen and of the severe but symptomless cases described in Group 3 (page 11) one was due to prolonged exposure to vapour in France in the last war and the other to a splash in a laboratory. To-day it is not possible to say which lesion is due to the splash and which to vapour. The vapour burn is, if anything, the worse of the two.

Twenty of the very severe cases from the last war were questioned as to circumstances and length of time in hospital. 15 out of 20 were confined in an enclosed space for some hours in vapour (dugout, trench, cellar, &c.), 8 stated that it was not raining at the time and some gave their opinion that gas was not troublesome during rain. Three were not wearing a gas mask. One wore it all the time, but was several hours in vapour. Ten only wore the mask part of the time. All of them were seriously ill, vomited, and several were unconscious soon after. The average stay in hospital was six months and the average time the eyes were closed was seven weeks. Many stated that their eyes were kept bandaged and this may have lengthened the time. It is probably better not to bandage.

CONCLUSIONS

(1) The clinical pathology of the lesion involving the limbus is similar in man and the rabbit (see Tables I and II).

(2) The reaction in man and the rabbit is dependent on the size of the dose and the anatomical situation. The effects of vapour and liquid are different only when the actual amount of mustard gas which soaks into the tissues is different, i.e. the difference is quantitative only.

(3) The late keratitis in man is seen as a degenerative ulceration depending on the initial damage sustained by the limbus and cornea, and not on any continued action of mustard gas or any of its breakdown products.

(4) From 3 it follows that in the present state of our knowledge prophylaxis is likely to be much more important than treatment. It is very difficult to assess the value of treatment in man since the dose of mustard gas received can never be known, and no two cases will be comparable. Two eyes of the same patient may be comparable though by no means always, even when the lesions are due to vapour.

deposition and fatty degeneration or persistent oedema) may begin in eight to nine months we should expect to find ulceration in man in about eight to ten years when the slow degenerative changes will have reached the requisite stage. The interim period is symptomless since there is no loss of epithelium and the changes are confined to the substantia propria of the scarred area. There is decreasing vascularization as the vessels recede, leaving behind them the deposit of cholesterol, so that clinically the eye appears to be getting better all the time. A study has been made of 39 cases and case-histories of patients at present attending Moorfields Eye Hospital or the Ministry of Pensions for delayed war gas keratitis, and this, together with Mr. T. J. Phillips' examination of 70 cases and my own observations on the course of the recurrent attacks, completes the picture. Coloured Plate, figs. 5 and 6 show a typical case.

Typical Case of Delayed Keratitis

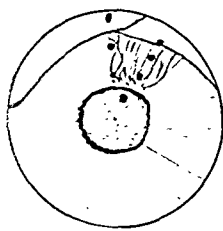
A patient aged 50 was examined. He gave a history of exposure to gas from a gas-shell barrage in July 1917. He put on his gas mask, left the lorry he was driving and went into a dugout for some hours. He was in hospital in France for a month and in England for six months during the whole of which time his eyes were kept bandaged. He had burns on the face and his chest was affected. His eyes gradually recovered except for slight dislike of strong light, but his chest has given trouble ever since.

In 1927 he attended an eye hospital for corneal ulceration, especially in the right eye. The individual ulcers healed quickly, often in a few days, but the irregularity of the surface of the cornea increased after each attack (cf. depressed area in rabbit's eye where cholesterol has been cast off, fig. 6, Plate IV) as there seems to be actual loss of substance when the degenerated area ulcerates. His sight therefore steadily deteriorated and in 1931 he was awarded a pension for his eyes. The recurrent ulceration continued and this year he was seen by Mr. Phillips and fitted with Dallos contact lenses. His visual acuity is R. eye = $\frac{1}{2}$, L. eye = $\frac{1}{2}$ not improved by spectacles. With contact lenses which are well tolerated he is improved to R. eye = 2 partly, L. eye = $\frac{1}{2}$, and so far has had no recurrence of ulceration. He shows in the right eye (which has had most ulcerated cholesterol crystals, fatty degeneration, silkiness (seen partly as dappling and partly as fine short lines, slightly different in texture from that in the rabbit, and probably depending on the slightly different shape and size of the cells and fibres in man) and irregular vessels in process of disappearing together with deposition of cholesterol (cf. rabbit, eight months or more). In the left there is less cholesterol, grey and black lines and alteration of the substantia propria.

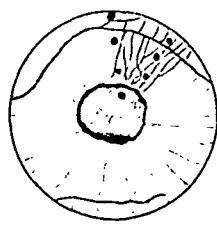
The astonishing improvement in visual acuity with contact lenses which he shows is seen in all cases and is due to the substitution of the smooth glass for the irregular and uneven corneal surface as the main refracting interface. The good tolerance of all delayed gas keratitis cases for individually fitted contact lenses is associated with a certain amount of corneal anaesthesia from previous destruction of nerve fibres and the effect of the lenses in preventing recurrent ulceration is probably explained by the protection of the partially insensitive cornea from multiple minute injuries. The same beneficial effect is seen from a tarsorrhaphy, but the contact lens has these great advantages, that it is permanent, transparent and improves visual acuity.

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PLATE I.



A



B

Fig. 1.—A. The dilatations of the smaller radial vessels in one sector of the iris 6.5 minutes after application of liquid mustard gas to the spots indicated in black. B. The same eye at ten minutes. The peripheral vessels are now dilated as well.



Fig. 2.—Appearance of cornea in narrow beam of slit-lamp in the region of two spots of liquid mustard gas (after twenty-four hours). These are shown by darker shading and are thinner than the surrounding paler area which stains more deeply than they do.

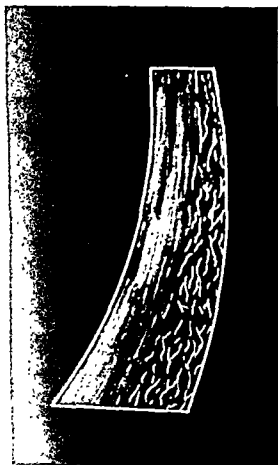


Fig. 3.—Appearance of corneal edema in the narrow beam of the slit-lamp.

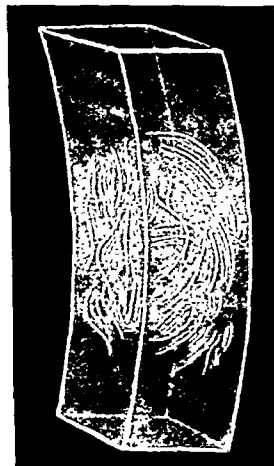


Fig. 4.—Appearance of "silki-ness" in the broad beam of the slit-lamp.

TABLE II.—COURSE OF MUSTARD GAS LESIONS IN MAN, COMPILED FROM OBSERVATIONS ON 65 CASES.

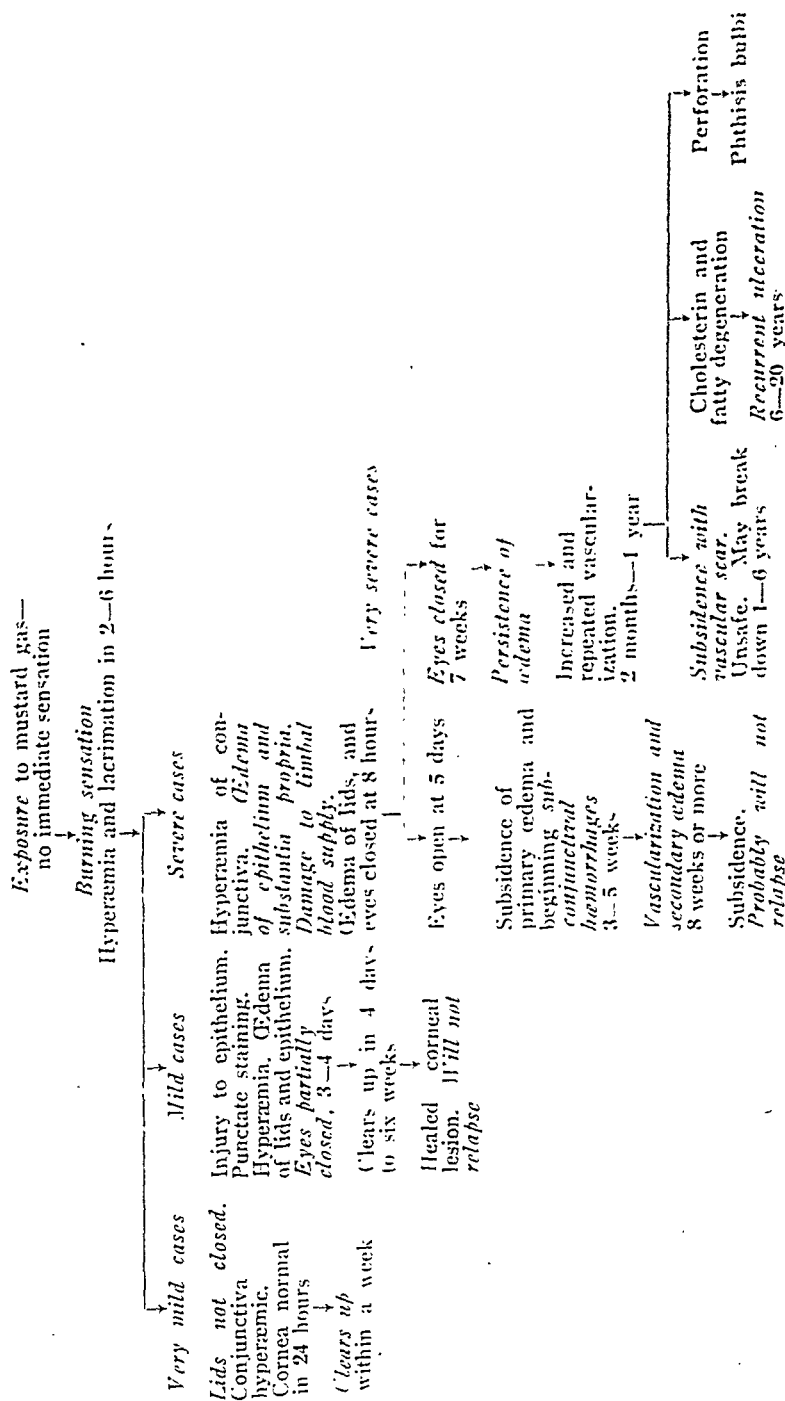
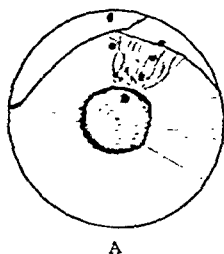
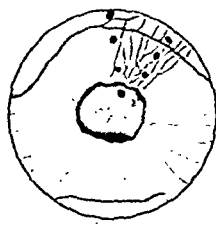


PLATE I.



A



B

Fig. 1.—A. The dilations of the smaller radial vessels in one sector of the iris 0.5 minutes after application of liquid mustard gas to the spots indicated in black. B. The same eye at ten minutes. The peripheral vessels are now dilated as well.



Fig. 2.—Appearance of cornea in narrow beam of slit-lamp in the region of two spots of liquid mustard gas (after twenty-four hours). These are shown by darker shading and are thinner than the surrounding paler area which stains more deeply than they do.



Fig. 3.—Appearance of corneal oedema in the narrow beam of the slit-lamp.

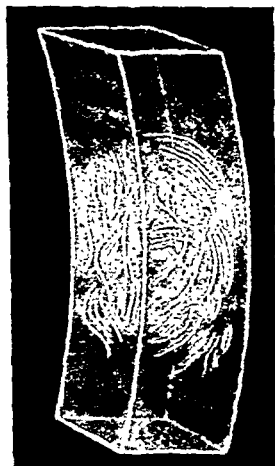


Fig. 4.—Appearance of "silki-ness" in the broad beam of the slit-lamp.

PLATE II.

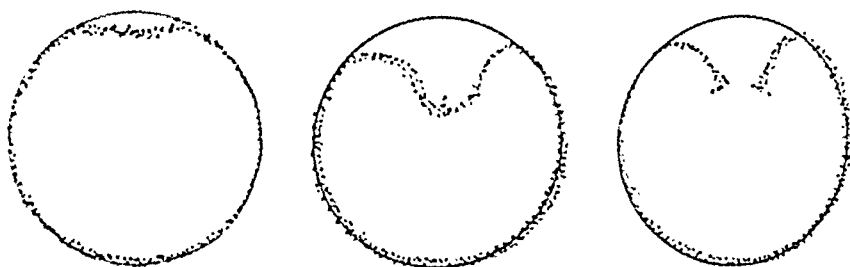


Fig. 5.—Some varieties of pigment slide formed during healing of the sectorial lesion.

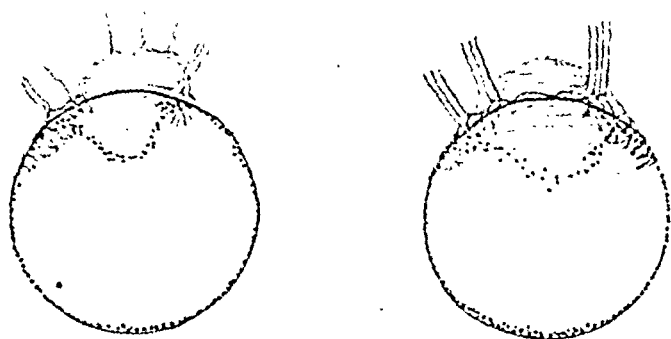


Fig. 6.—Diagram of a cornea showing progress of invading vessels. The injury is a sector in the upper part. The pigment slide is shown and the shaded portion is cedematous. The vessels enter first through the undamaged limbus.

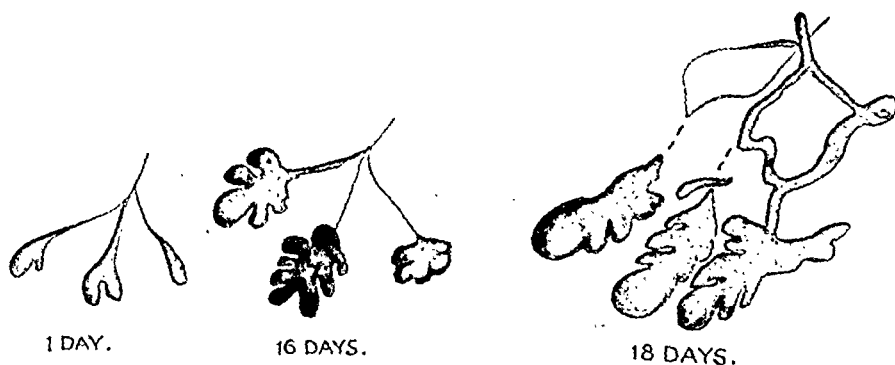


Fig. 8.—The genesis of a group of blood islands, with constriction of their feeding vessels.

PLATE III.

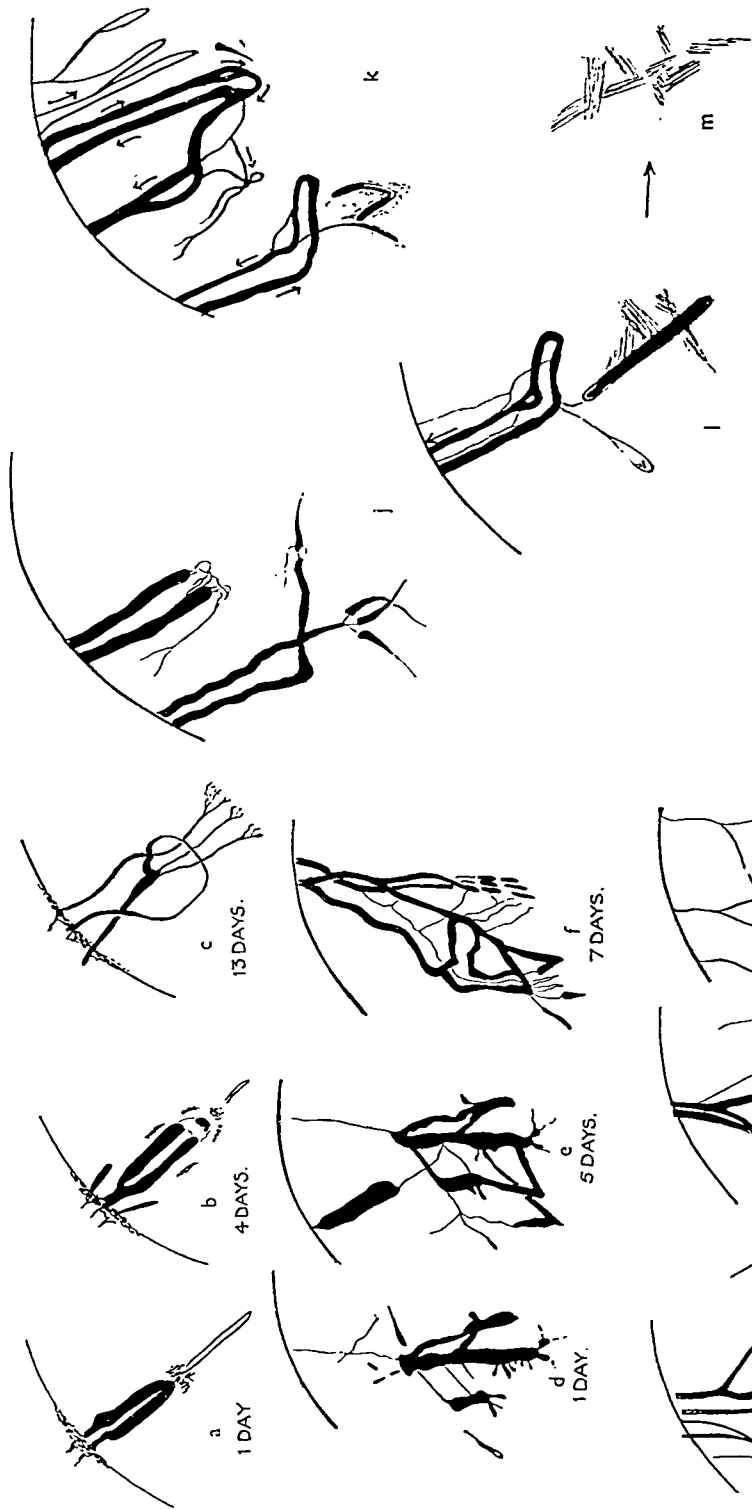


Fig. 7.—Varieties of vessels seen in corneal vascularization. a. Single loop with constriction and narrow advancing point. Hemorrhage at point of constriction. (First day of observation, not first day of experiment.) b. The same loop showing thrombosis, hemorrhage and isolation of the point. c. The system healed by replacement by narrow well-formed vessels. d, e, f, g, h, i. Six stages (fifty-four days of observation) of vascularization and healing in another rabbit. j, k. Two stages of advancing vascularization. l, m. Stoppage of circulation in terminal vessel and hemolysis, which is finally all that can be seen.

PLATE IV.

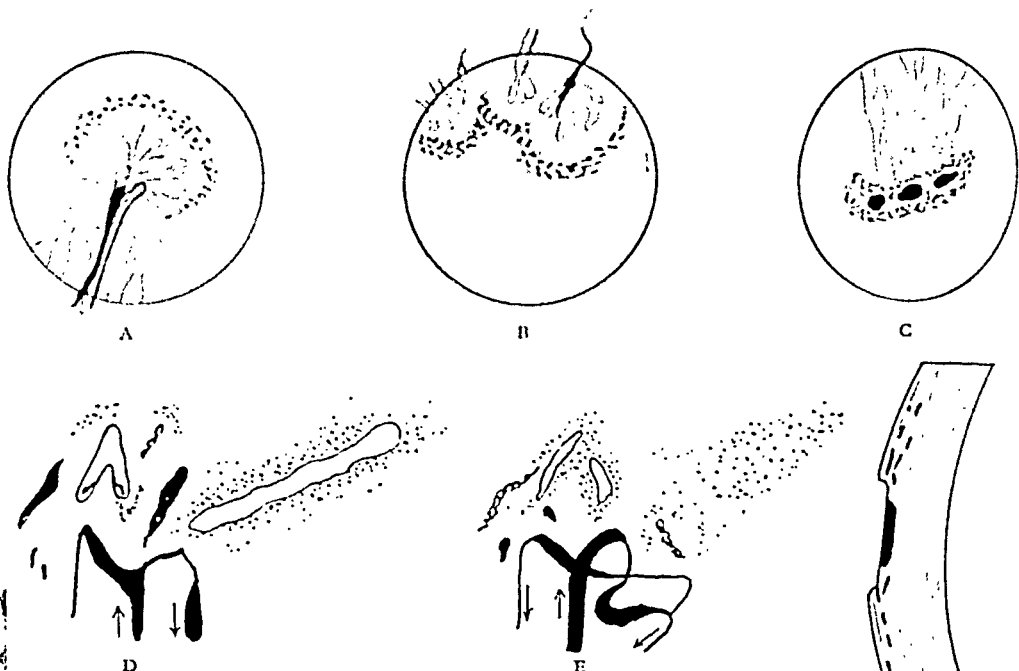


Fig. 9.—Deposition of cholesterol in scars and recurrent ulceration. A. Semicircular cholesterol deposit round arborizing vessel. B. Circinate cholesterol scar. C. Recurrent ulcers. The black spots represent staining areas. D. Empty vessel surrounded by cholesterol crystals, and other detached ends or vessels partly empty. E. Disappearance of many of these. F. Position of cholesterol in C at edge of ulcer seen in narrow beam.

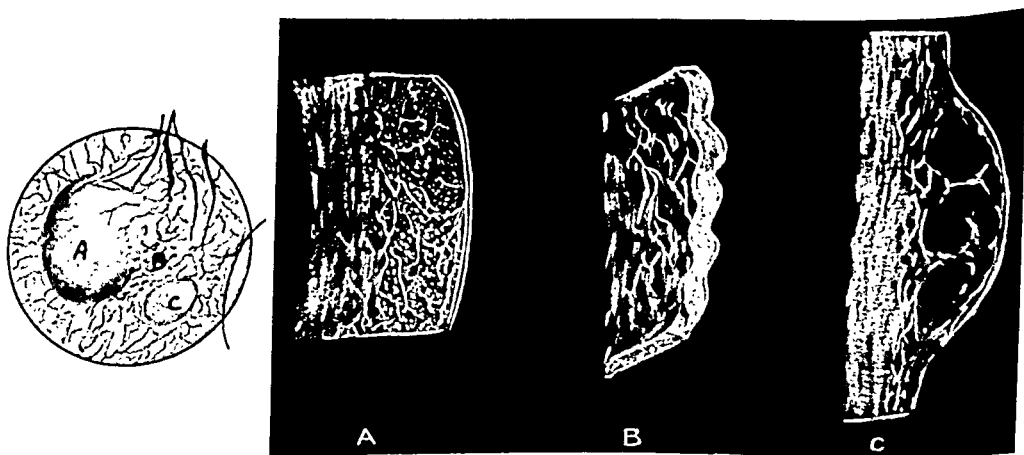
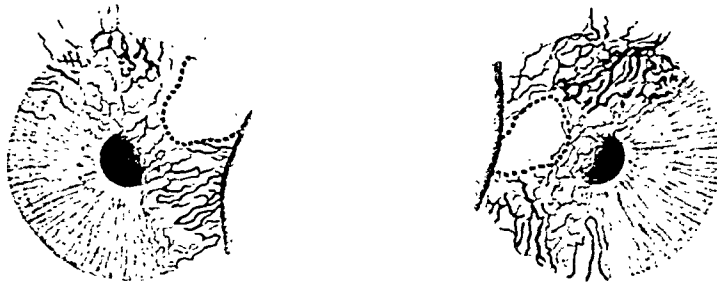


Fig. 10.—Appearance of cornea of badly injured eye. A. Section (narrow beam) through the blister-like protuberance at A. This shows fibrils in a jelly-like matrix filled with minute bright spots, the blood-vessels (black) running deep to this. B. A broader beam section through B, showing formation of fibrous tissue with blood-vessels running among the new fibrils. C. Section (narrow beam) through the bleb at C. The fluid is in spaces in the superficial layers of the stroma and not truly subepithelial. There is granulation tissue in the angles, invading the bleb, which will in time become completely converted into solid vascular connective tissue.

PLATE V.



(I. M., copied
by Hamblin.)

Fig. 11.—Two eyes of rabbit with recurrent ulceration from persistent oedema and fibrous tissue formation. The areas surrounded by the dotted lines stain with fluorescein.

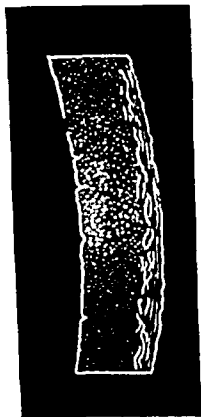


Fig. 12.—Appearance of cornea in Case 4 (Group 3) in the narrow beam of slit-lamp at one week. The epithelial surface and oedema of the substantia propria are to the right, the endothelial irregularity to the left.

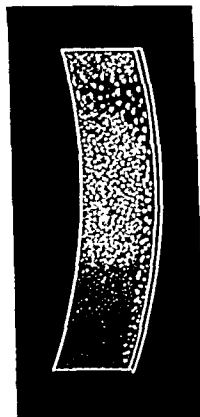


Fig. 13.—Appearance of cornea in Case 4 (Group 3) in the narrow beam of the slit-lamp at three weeks, showing shiny dots in the substantia propria (cf. fig. 12).

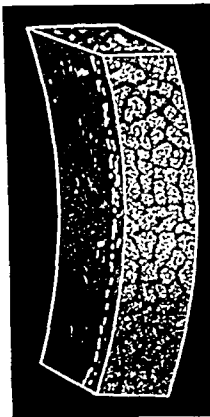


Fig. 14.—Appearance of superficial dapping in Case 4 (Group 3) seen in broad beam at five and a half weeks.

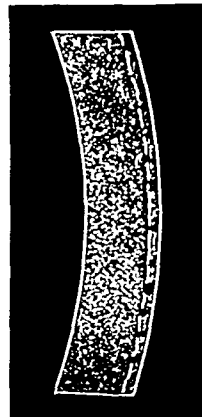


Fig. 15.—Case 4 (Group 3) seen in narrow beam at five and a half weeks.

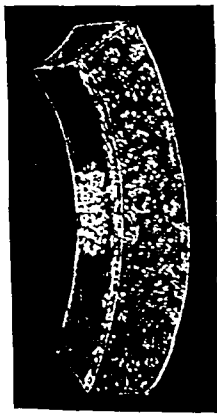


Fig. 16.—The mildest Group 3 case. Appearance of dapping and endothelial change seen in a, the broad beam, and b, the narrow beam of the slit-lamp, twenty-three years after the injury.

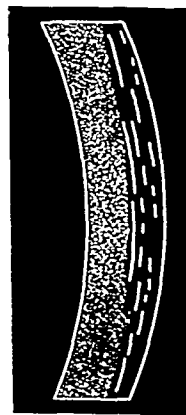


Fig. 17.—Shiny dots and lines in the substantia propria many years after exposure.

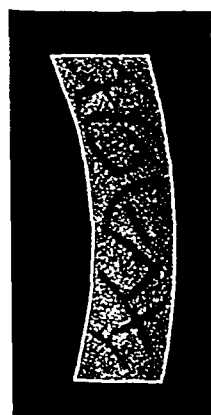


Fig. 18.—"Black-lines" or "tubes" seen in narrow beam of slit-lamp many years after exposure.

PLATE VI.

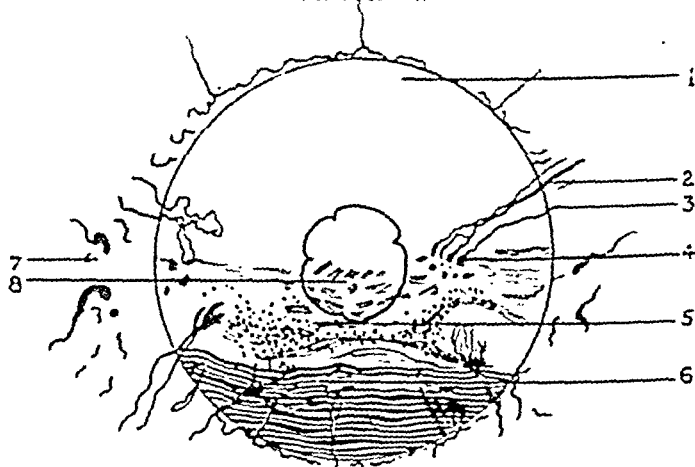


Fig. 19.—Scheme showing characteristics of scar ten to twenty years after severe gassing. 1. Normal cornea and normal limbus, protected by upper lid. 2. Vessels entering cornea from conjunctiva at unscarred limbus and running down to scar. 3. Area of destruction of limbal loop. 4. Vessels in cornea showing thrombosis and hemorrhages. 5. Cholesterin and fatty degeneration. 6. Depressed scars of late ulcers. 7. Pale area and varicose conjunctival vessels. 8. Outline of pupil showing posterior synechiae. Fatty degeneration of overlying cornea.

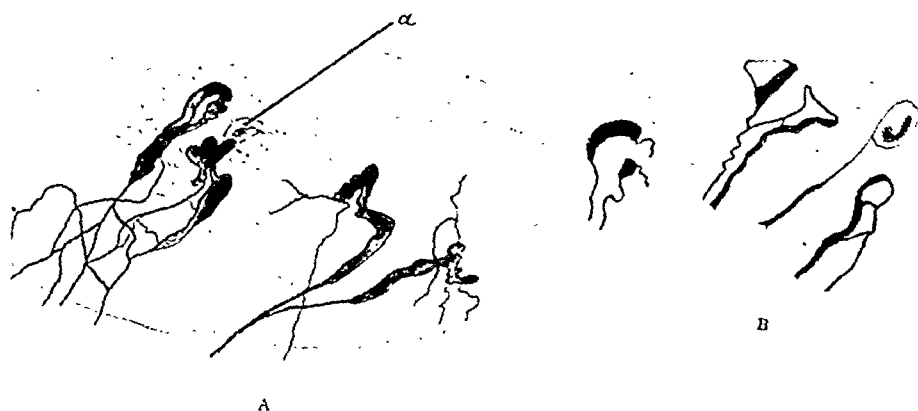
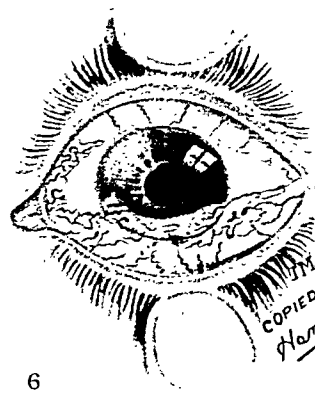
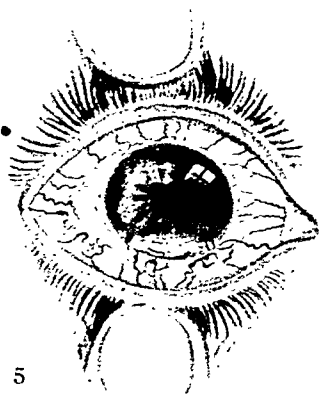
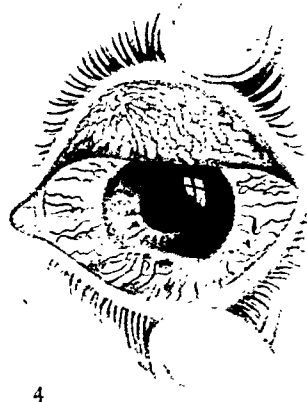
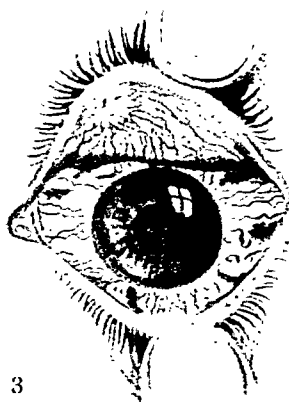
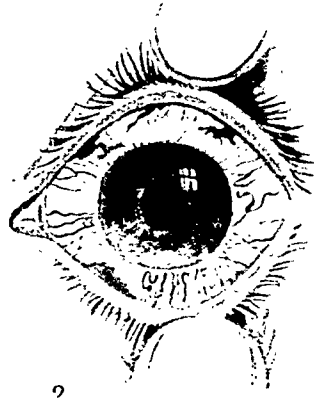
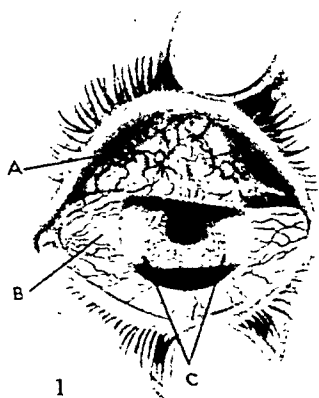


Fig. 20.—A. Detail of scar in Group 4 case subject to recurrent ulceration. Note abnormal vessels, cholesterin and fatty degeneration, and silky fibrillar appearance. At (a) the endothelial wall of a disappearing vessel can be seen surrounded by cholesterin. B. Further details of abnormal vessels.

COLOURED PLATE

FIGS. 1 TO 6.



R.E.

L.E.

Fig. 1.—Left eye of Case 2 in Group 3. A. Patch of hyperæmia. B. Triangular white patch. C. Corneal haze. Fig. 2.—Left eye of Case 4 in Group 3 seen at three weeks. Fig. 3.—Left eye of Case 4 in Group 3 seen at five and a half weeks. Fig. 4.—Left eye of Case 4 in Group 3 seen at eight weeks. Figs. 5 and 6.—Group 4 case, twenty-three years after exposure to mustard vapour and thirteen years after the first attack of delayed ulceration. R. E. shows plaque of fibrous tissue at limbus on outer side, abnormally arranged and varicose conjunctival vessels and gross scarring of lower part of cornea and limbus. A few posterior synechiæ. L. E. shows triangular pale areas at limbus, abnormal conjunctival vessels, dense corneal scarring and posterior synechiæ. The varicosities are slightly exaggerated in the drawing.

IDA MANN and B. D. PULLINGER:

A Study of Mustard Gas Lesions of the Eyes of Rabbits and Men.

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PLATE VI.

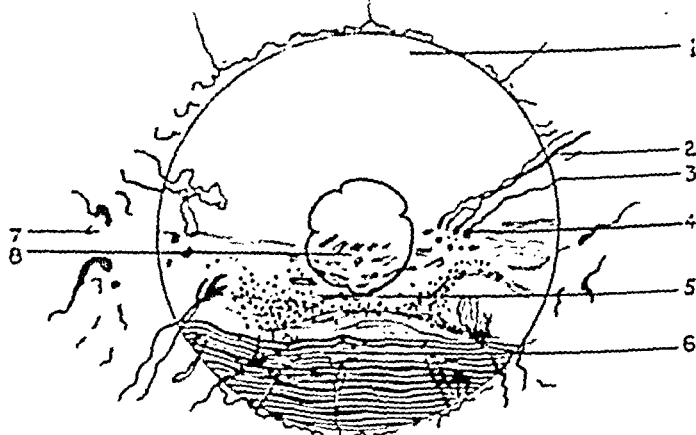


Fig. 19.—Scheme showing characteristics of scar ten to twenty years after severe gassing. 1. Normal cornea and normal limbus, protected by upper lid. 2. Vessels entering cornea from conjunctiva at unscarred limbus and running down to scar. 3. Area of destruction of limbal loops. 4. Vessels in cornea showing thrombosis and hemorrhages. 5. Cholesterol and fatty degeneration. 6. Depressed scars of late ulcers. 7. Pale area and varicose conjunctival vessels. 8. Outline of pupil showing posterior synechiae. Fatty degeneration of overlying cornea.

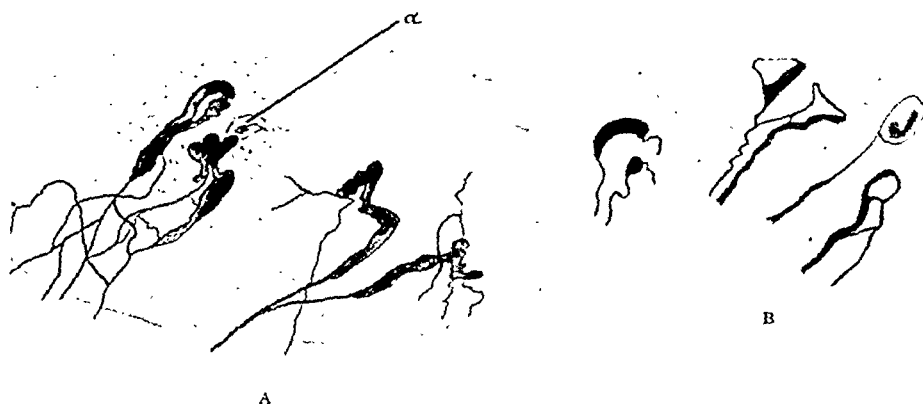


Fig. 20.—A. Detail of scar in Group 4 case subject to recurrent ulceration. Note abnormal vessels, cholesterol and fatty degeneration, and silky fibrillar appearance. At (α) the endothelial wall of a disappearing vessel can be seen surrounded by cholesterol. B. Further details of abnormal vessels.

JOINT DISCUSSION No. 1

Sections of Otology and Laryngology

[December 5, 1941]

Chairman—F. W. WATKYN-THOMAS, F.R.C.S.

(President of the Section of Otology)

DISCUSSION ON THE EFFECTS OF FLYING ON THE NOSE AND EAR

A GENERAL SURVEY OF OTORHINOLOGICAL CONSIDERATIONS IN SERVICE AVIATION

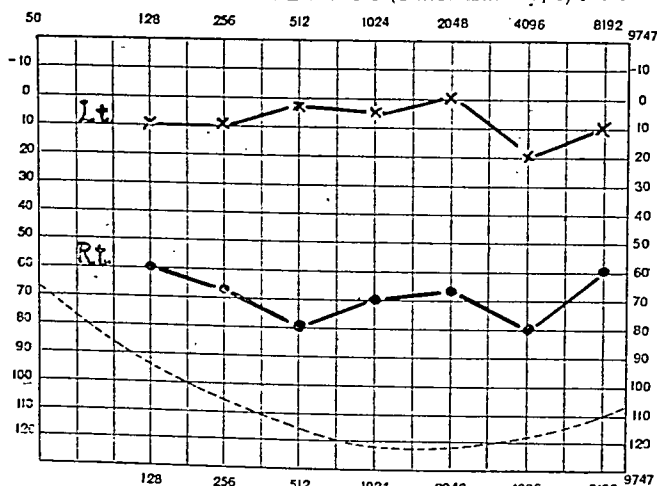
Wing Commander J. F. Simpson, Senior Ear, Nose and Throat Specialist, Royal Air Force: The first deleterious effect of aviation ever to be recorded was earache, and this was experienced during one of the earliest balloon flights ever made—to be exact, in 1783. To-day the organ which is perhaps most commonly affected by flying is still the ear. The nose, and in particular its accessory sinuses, can also be affected by flight, but less frequently than the ear.

During the last war many of the problems which face us now were well recognized; but advances in aviation and especially in aerial warfare since that time have intensified some, lessened others, and added new ones. These advances are represented by the great increase in the speed and manœuvrability of modern machines, by the greater altitudes now attained, and by the vastly increased duration of operational flying. Much of this improvement has been accomplished at the expense of greater engine power, and therefore greater noise. The noise of modern aircraft, especially multi-engined 'planes is immense and is responsible for the problem of aviation noise deafness. Recognition of its progressive and permanent character has led to investigation into the nature of the noise, into its mode of effect and to measures of protection against it. Group Captain Dickson has been personally responsible for much of this work. The part played by vibration is not fully understood, but no doubt our comrades in the tanks will be able to throw more light on that subject.

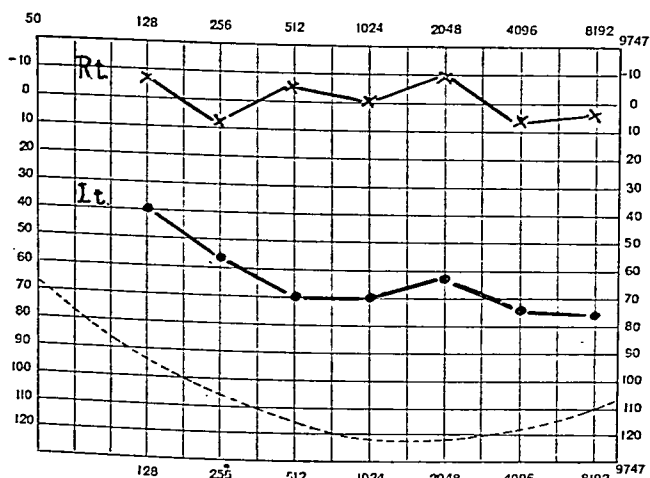
Yet another important change in modern Service aviation as contrasted with that of the last war is the greatly extended use and vital importance of all forms of telecommunication. Through wireless telephony and telegraphy 'planes maintain contact with ground stations and with each other, while members of the crew communicate with each other by means of an internal telephone system. The importance of good hearing in flying personnel is obvious, especially when it is remembered that they must be able to hear efficiently in the most difficult circumstances. Messages may be weak from various reasons and external noise excessive. Under such conditions subnormal hearing is likely to lead to a mistake in the reception of a message with possibly disastrous results. It follows that in the selection of aircrew personnel examination of hearing is of the utmost importance. This in itself presents a separate problem.

The usual hearing test used cannot be regarded as being entirely satisfactory and the results are not comparable. The estimation arrived at by the forced whisper test varies greatly, as it is carried out in different rooms where background noises are variable and is conducted by examiners whose forced whispers are of different intensity and tone, and often of accent. Pure tone audiometry is too lengthy for routine purposes, and in any

Audiograms of Two Cases of Permanent Deafness (Inner Ear Type) following Aero-otitis.



CASE I.—Power dive from 14,000 ft. Has been deaf in left ear ever since. No real pain during the dive.



CASE II.—Power dive from 30,000 ft. to 10,000 ft. Both ears became deafened, the left one recovered in twenty-four hours, but the right one has remained deaf. No pain experienced during the dive.

Both cases had normal tympanic membranes when seen some weeks after the onset of deafness caused by rapid descent. The tympanic membranes bulged on Valsalva's test. The Rinne test was positive and the Weber test lateralized to the unaffected ear in both cases.

not form a practical issue in aviation. The uncontrolled and widespread responses of a somewhat hypersensitive labyrinth are not considered evidence of disease. In practice the routine rotation test has been found to give little reliable help in the discrimination between men who will or will not develop intractable air-sickness and has been given up. Squadron Leader Winfield will be speaking about the problem of air-sickness.

Disease of the ear, nose and throat will impair the efficiency of an aviator, especially under conditions of modern aerial warfare, usually by deafness or by predisposition to

case does not give any clue to the quickness-of-interpretation factor of hearing. These difficulties have led to an attempt to find a practical but accurate, rapid and comparable method for grading the ability to hear speech. Squadron Leader Fry, from the Department of Phonetics at University College, London, is now working with us upon this problem.

It is by changes in altitude that the commonest and most dramatic disturbances of the ear and nose are brought about. These disturbances depend upon barometric pressure changes and not upon oxygen lack. Until the use of pressure suits or sealed cabins in aircraft is universal the ear and nose will continue to be subjected to pressure changes during climb and descent. With changes of altitude air will flow in or out of the middle-ear cleft and sinuses, so that the pressure within them is equalized with that of the external atmosphere. Obstruction to this equalizing mechanism gives rise to one of the commonest and most painful complaints met with in aviation. In the case of the sinuses the pain usually starts in the region of the frontal sinuses, spreads up towards the vertex, radiates between and behind the eyes, and may even involve the upper jaws. The decompression chamber has been largely used in observing this as it is a convenient method of reproducing the effect of large and rapid changes of altitude. I may remark here that no very gross or obvious changes have been seen in the nasal mucosa during these changes of pressure. In the case of the ear, besides pain there is deafness which is often accompanied by certain changes in the drumhead and middle ear, and occasionally though rarely, vertigo. This condition was described in detail by Armstrong in America who called it *aero-otitis*. Squadron Leader McGibbon will deal with this condition in greater detail.

From a physical point of view the middle-ear cleft and sinuses should be regarded as being essentially the same, both being rigid bony cavities lined by mucous membrane and having small openings to the external air. In the case of the middle-ear cleft, however, there is a small elastic window in one of the walls, the drumhead, and this bulges inwards or outwards according to the difference of pressure on either side of it. Because descent is usually more rapid than climb, and because there appears to be some one-way mechanism that lets air out of more easily than into the middle ear and sinuses, pain is usually, but not invariably, complained of during descent. The pain in the ears and sinuses may be mild, or so severe as to cause an airman to break off a dog-fight and even to make him faint. I have personally suffered such agonizing sinus pain as to drench myself with sweat.

Experiments seem to show that the main factor of the pain in the ear lies not in the bulging of the drumhead, but (as in the case of the sinuses where no drumhead exists) probably in the mucosa or bony walls of the cleft. It is doubtful if the pain can be explained merely on the basis of passive congestion; other and more complex factors probably take part. Congestion can occur as is evidenced by cases of *hematotympanum* and *hematocoele* of the antra. A sinister but occasional complication of *aero-otitis* is a severe and permanent form of deafness. When seen after an interval of some days these cases show no abnormal otoscopic appearances and the Eustachian tubes are clinically patent, but a high degree of deafness which is of the classical "nerve or inner ear" type remains, though in the several cases seen the vestibular reactions were not impaired (see audiograms).

Besides alteration in altitude the ear is affected by changes in speed, direction and position in aviation. During the last war much attention was paid to the vestibular reactions towards these changes, especially by Mr. Sydney Scott in this country. It was realized how important a part vision played in correcting the false and illusionary impressions derived from the labyrinth during flight. Without adequate visual reference to the earth or instruments a pilot eventually and inevitably goes into circular motion, develops vertigo, goes into a spin and crashes. The development of the technique of blind flying has overcome this. Essentially this consists in replacing the unreliable vestibular and other sensations by the reading of unfailing instruments which record speed, height, direction, climb, turn and bank. For success the blind flyer must have complete faith in his instruments and must disregard his own sensations. This, however, does not deny the existence of what is termed "flying sense", which is acquired by the experienced pilot who feels himself to be a part of the aeroplane. This "flying sense" will not suffice for blind flying, but is necessary for acrobatics and landing, and amongst other things involves the use of the labyrinth. True pathology of the labyrinth does

known to be injurious to the ear, but my interest has been mainly confined to those arising from exposure to aeroplane noise. The intensity level of such noises is in the region of 120-130 phons. In connexion with flying it arises chiefly from three sources, namely, the engine, the propeller (mainly from the tips of the blades where the velocity is greatest) and the wind (slipstream).

Observations extending over a prolonged period have shown that in a healthy person without history or evidence of aural disease and whose history does not suggest damage by drugs or severe constitutional disease or familial deafness but reveals exposure to noise of an intensity such as I mentioned, a high tone loss must be suspected to represent the effects of acoustic trauma.

It is now generally accepted that the first evidence of such trauma manifests itself as a dip localized to 4,096 cycles per second both for air and bone conduction without involvement of frequency below or above this level, when a 6A Western Electric Audiometer is used. This drop may be restricted at first for some time at this frequency, but repeated and continuous exposure to noise may involve frequencies below this level. The individual may be unaware of any auditory defect and probably notices no disability so long as this loss is restricted to 4,096 cycles per second and is not very marked. Involvement, however, of lower frequencies such as 2,048 or 1,024 cycles per second will constitute a definite impairment of hearing. Results of hearing tests for speech confirm audiometric findings. Mistakes in hearing the spoken word often occur when accurate interpretation depends upon the ability to recognize consonants whose essential characteristics are highness in frequency and weakness in intensity. For example with a 50-60 decibel loss at 4,096 cycles per second, the candidate has difficulty in hearing words like sister, solicitor, &c., from a distance of 20 ft. with a forced whisper whereas he can hear low-pitched words quite easily.

The factors which influence the degree of hearing loss from acoustic trauma are:

(1) The total time of exposure. Individual ears respond in different ways. I have seen a dip at 4,096 cycles after one hundred hours' exposure to aeroplane noise and again have seen only a slight localized drop after exposure for some years.

(2) The length of exposure at each period. The ears can recover with a rest at first but become permanently damaged if exposure continues.

(3) Loud sounds only cause damage.

(4) The character of the sound stimulus, whether continuous or interrupted.

(5) The type of protection adopted.

(6) The surroundings, whether in enclosed or open spaces. A smooth concrete wall will reflect 96% of the acoustic energy striking it.

(7) Previous disease. Previously damaged ears are more susceptible to acoustic trauma than normal ones.

The relation of the frequency to the resultant trauma is not clearly understood. Exposure of the ear to noise of pure tones of 512, 1,024, 2,048, 4,096 cycles per second show a maximum dip an octave above the frequency of the fatiguing note and not at the frequency of the fatiguing note. The common threshold curve with a 4,096 dip was not obtained for a series of tones of an approximate uniform intensity used to produce acoustic trauma. Subjects exposed to a fatiguing note of 2,048 cycles per second showed a localized dip at 4,096. Sounds of low frequency (64-256) produce less acoustic trauma, and certainly no dip at 4,096, than those of high frequency and of approximately equal intensity.

On the other hand analysis of aeroplane noise by means at our disposal has shown that the frequencies of the highest intensity 110-115 db. are at the low end of the acoustic spectrum. Thus it would suggest that a low tone was causing a high tone deafness. Explanation is not clear and I believe that when dealing with a complex noise of high overall intensity, damage is inflicted by the excessive sound pressure on the most susceptible part of the cochlea, that is the basal coil. This by virtue of its situation may probably bear the brunt of the insult.

It is also possible that sudden instantaneous peaks of high intensity, which are not recorded by instruments at our disposal, may occur at 2,048 cycles per second at frequent intervals and thus be responsible for the dip at 4,096 cycles per second. Lastly the external auditory meatus which, as Littler has shown, has in its normal condition a characteristic natural period, may act as a resonator intensifying a frequency in a complex sound in the region of 3,000 cycles per second and thus cause a dip at 4,096 cycles per second.

pain. The importance of such affections must be assessed from a Service point of view, which will include that of the Administration and the Treasury. Due allowance must always be made for a generous margin of safety and for the possibility of recurrences. Different views may be taken of the same disability according to whether it occurs in personnel undergoing training, or experienced aircrew. Active suppuration of the ear, on general lines, regarded as a contra-indication to flying, especially operational flying, if only from the point of view that hearing is affected and quite apart from the fact that treatment is required. It is realized that a suppurating and deaf ear will not actually render a man incapable of flying a machine, but he cannot be considered 100% efficient. This leads to the difficult question of the assessment of a monaural hearing defect as compared with a binaural one. It hinges upon the amount of hearing present in either ear and upon the margin of safety required for the particular duty to be performed.

On similar lines a dry perforation usually means deficient hearing and a chance of recurrence of otorrhoea under Service conditions, but experienced airmen so affected are sometimes used in non-operational work. Provided there is no appreciable hearing loss and that the Eustachian tube is easily opened, a moderate degree of scarring of the drum does not debar from flying, though it is possible that one adherent to the inner wall might give rise to discomfort. Any evidence of past suppuration in the middle-ear cleft should make one suspect that the Eustachian tube might be left with some residual impairment of function. This is shown by the fairly high incidence of cases which give a past history of otitis media amongst those who suffer from aero-otitis. Judging the ease with which a Eustachian tube can be opened is not as simple as one might believe, but it is desirable when examining candidates to have some measurement of this potential patency. Various methods have been employed but it is not unlikely that the only reliable one will involve the routine use of the compression chamber.

All acute inflammatory conditions of the nose and throat are of the greatest importance, as they can interfere with the mechanism for adjusting pressure in the ear and sinuses. For this reason flying with a sore throat or cold in the head is forbidden. Chronic infections are for the same reasons regarded seriously. In this instance, however, the effect is less easy to forecast. We have seen some cases of longstanding sinus suppuration fly and not complain of discomfort. Probably a chronic post-nasal catarrh, associated with a mild and recurrent aero-otitis is as difficult to manage and to legislate for as any case one is likely to meet. Polypi sometimes occur, and in one or two cases of experienced personnel a radical external ethmoidectomy has been followed by absence of symptoms on resumption of flying. The septum is not operated upon unless very definite obstruction and discomfort is complained of. It is not certain that high deviations of the septum have much effect upon the incidence of sinus pain, but it cannot be denied that a submucous resection operation appears to have resulted in the relief of this in a number of cases. Epistaxis, an unpleasant occurrence when the airman is wearing an oxygen mask, is not often met with, but when present the usual leash of vessels in the "bleeding area" is always seen.

My general impression is that considering the number of men flying and the strain imposed upon them, it is remarkable that we do not meet with more aural and nasal disturbances, but this may be due to the careful selection of flying personnel.

AVIATION NOISE DEAFNESS AND ITS PREVENTION

Group Captain E. D. D. Dickson, Consultant in Otorhinolaryngology, Royal Air Force: Noise is one of the features of uncontrolled development in a mechanical age—an undesired by-product of the machines which are employed for industrial and war operations. Noise as it affects Air Force personnel arises from the engine and propellers used in modern aircraft. The ill-effects of it have become apparent since the introduction of enclosed cockpits in multi-engined aircraft and the discarding whenever possible by pilots and crews of the flying helmet. In some instances resort has been made to various forms of ear plugs, but these either on account of construction or unsuitability cause discomfort and/or fail to afford the required protection.

Since the introduction of audiometry, studies made by different observers have helped to crystallize the clinical entity of acoustic trauma. Many types of sound stimuli are

in his helmet. It seems unlikely that such a technique will in the end prove practicable for use at all Service medical boards and an alternative one is being developed in which both the background noise and the speech signals are delivered into the candidate's telephones. This is most easily effected by making gramophone records in which the test words or sentences are spoken against a noise background. When this is done it is possible to vary the relative levels of speech and noise, i.e. to change the signal to noise ratio, and to work rather on the principle of the gramophone audiometer. One advantage of using gramophone records and of testing against a noise background is that the acoustic conditions for testing can be reasonably standardized, and a further advantage of this technique is that a number of candidates can be examined at the same time.

The above is a brief indication of the nature of the work which is being done. A great many questions concerning the functional aspect of hearing remain to be answered, and in conclusion I should like to mention one or two of those which are directly connected with the problem of signals.

(1) The importance of training in the reception of transmitted speech. How far can training compensate for defects of hearing (as shown by pure-tone audiometry)?

(2) How much more difficult is it to train a person with defective hearing than one with normal hearing?

(3) What is the effect of monaural deafness on the ability to receive signals? How far is it justifiable to employ in duties involving the use of auditory signals a person who has a gross defect in one ear?

(4) What is the effect of frequency and intensity limitations in the transmission system upon the reception of signals by a person with defective hearing?

These are some of the questions to which the answer is urgently needed.

AVIATION PRESSURE DEAFNESS

Squadron Leader J. E. G. McGibbon: In his survey of the aural lesions associated with flying, Wing Commander Simpson mentioned a group of symptoms which sometimes occurs owing to closure of the Eustachian tube during descent in aircraft—a syndrome known in America as "aero-otitis", and which I have called "aviation pressure deafness".

As I am attached to a Royal Air Force General Hospital which serves many Operational Stations I have had the opportunity to examine a relatively large series of patients suffering from this disability.

26.0% of the total number of aircrew personnel referred to the ear, nose and throat department of this hospital have complained of aural symptoms which have arisen during loss of height in aircraft; and for the purposes of the following notes I have reviewed 100 cases.

I propose to deal shortly with:

(1) *The main features of obstruction of the Eustachian tube, and*

(2) *The nature of the obstruction.*

(1) PROOF OF EUSTACHIAN BLOCKAGE has been based on:

A. *Presumptive evidence.* B. *Positive evidence.* (i) *Type of deafness.* (ii) *Otoscopic appearances.* (iii) *Impermeability of the tube to air.* (iv) *Impermeability of the tube to the passage of tympanic catheter or sound.* (v) *Impermeability of the tube to radio-opaque fluids.*

A. Presumptive Evidence

(i) *Deafness alone or in association with other symptoms was the most common complaint of these patients, and this is shown in Table I, which is an analysis of their predominant symptoms:*

TABLE I.—PREDOMINANT SYMPTOMS OF 100 PATIENTS

| | | | | | | | |
|-----------------------|-----|-----|-----|-----|-----|-----|----|
| Deafness alone | ... | ... | ... | ... | ... | ... | 31 |
| Deafness and pain | ... | ... | ... | ... | ... | ... | 55 |
| Deafness and tinnitus | ... | ... | ... | ... | ... | ... | 5 |
| Deafness and vertigo | ... | ... | ... | ... | ... | ... | 3 |
| Pain alone | ... | ... | ... | ... | ... | ... | 6 |

As to the methods of protection so far as flying personnel is concerned in the Royal Air Force, the wearing of a flying helmet with telephones attached affords the fullest protection. Tests have shown that the characteristic dip at 4,000 cycles per second after exposure to noise does not occur. The other appliances such as ear plugs, plasticine, &c., afford a certain amount of protection, but it must be realized that if full protection against the effects of noise is provided by such appliances, intelligibility of speech becomes greatly reduced. They are, therefore, impracticable from the flying point of view where the use of telecommunication becomes necessary. It is comparatively easy by such means to reduce or damp down the high-pitched tones, and if as recent experiments have demonstrated, high notes of high intensity cause more damage than low ones of the same intensity this may explain the success obtained in protecting ears against the injurious effects of noise by the wearing of a helmet with telephones attached.

A SUGGESTION FOR A NEW METHOD OF TESTING HEARING IN AVIATION CANDIDATES

Squadron Leader D. B. Fry: It is generally agreed that the hearing test at present in use for the selection of flying personnel is not a very satisfactory one, since it fails in some cases to achieve its object. This object is twofold, first, to select for flying duties candidates who will be worth training, for the Service naturally does not wish to train people who will prove eventually unfitted for their duties, and secondly, to ensure that no candidate is rejected who would in fact be capable of carrying out flying duties. Thus the test required to replace the present one is a functional test which will indicate the suitability of a candidate for the duties which he is to undertake.

In the Service hearing is most important from the point of view of signals—a man must be able to hear, recognize and interpret auditory signals, whether speech or morse. The problem of testing such ability is rather complex. Hearing acuity is important and where there are gross hearing losses for pure tones one can predict a certain degree of difficulty in understanding speech, but even here reservations have to be made. If the gross hearing loss is in one ear only, it is quite possible for the subject to be as good at recognizing signals as a normal person with no hearing loss. On the other hand, hearing acuity alone is not enough. Ability to interpret signals depends on both hearing and intelligence, i.e. the ability to guess the right answer. Here we have to notice too the great importance of training, for the ability to guess the right answer is very largely the outcome of training. The question is further complicated by the conditions in which the candidate will have to work if accepted. The chief factor here is a noise background for which a level of 120 phons is not uncommon. Hence we have to ensure that a man can receive signals satisfactorily in these conditions.

From the foregoing it is clear that any kind of threshold testing with pure tones, apart from the difficulties attendant upon such testing, will not tell the whole story. We have therefore had recourse to various kinds of articulation testing, i.e. testing the candidate's ability to receive correctly speech sounds, syllables and sentences. We have said that such tests have to be carried out in conditions approximating to those in which the candidate will have to work. One factor in these conditions, the noise background, has been mentioned already. Another very important one is the aid to understanding which will always be provided by the context; in other words, we are really interested in the candidate's ability to understand sentences, not isolated sounds or syllables. Unfortunately, testing by means of sentences is a long business, because one cannot obtain reliable results without using a large number of sentences. It is evident that if one can establish a significant correlation between ability to receive sentences and ability to receive sounds or syllables correctly, one would be justified in adopting the much quicker and more practicable sound or syllable test. Previous work on articulation testing, chiefly by the Bell Telephone Laboratories, has demonstrated significant correlations between sound, syllable and sentence articulation, but since all such work was done in quiet conditions, it has been necessary to discover first whether such correlations are to be expected when working in a noise field. This work is not yet completed, but we can say that there are indications that significant correlations will appear and it seems probable that the form of test which will be eventually decided upon will be a sound articulation test.

The technique of testing which has been used in the experimental stages is briefly as follows: The subject is placed in a high-level noise field from which his ears are protected by the standard Service flying helmet. The articulation tests, i.e. lists of words and sentences, are then conveyed to him through a pair of telephone receivers carried

Increased patency of the tube brought about by swallowing and

A further, though slight, increase of nasopharyngeal pressure caused by the air-content of the pharynx being forced into the nasopharyngeal and nasal cavities during the "closed phase of the pharynx" (Barclay, 1930) whilst the anterior nares were occluded.

(c) *With Politzer's bag the average maximum nasopharyngeal pressure obtained was 210 mm.Hg.*

(d) *With the Eustachian catheter in the tubal orifice estimation of the maximum pressure at its tip has been made and found to be 320 mm.Hg. In only one case was inflation by Eustachian catheter successful at the first visit.*

(e) *Inflation through a tympanic catheter was successful also in one patient, but the pressure created in the Eustachian tube was not measured.*

(iv) *Impermeability of the tube to a tympanic sound and/or catheter.*—An attempt to pass a tympanic sound or catheter was made in 57 patients and in all the passage of the instrument was arrested by a firm obstruction at a distance varying from 0.5 cm. to 2 cm. from the tip of the catheter.

(v) *Radiological evidence of obstruction after the injection of radio-opaque fluid into the Eustachian tube* has been demonstrated in 36 patients of this series by Squadron Leader Rees-Jones and myself. The technique and results in 34 of these have been published (1941), but I would mention that in two patients the obstruction was at the isthmus, in one at 0.5 cm. from its pharyngeal orifice, and in the remaining 33 at intermediate situations.

(2) THE NATURE OF THE TUBAL OBSTRUCTION

Commonly the lumen of the posterolateral half of the cartilaginous tube is closed by contact of its mucosal walls and possibly by a film of mucus; but the extent and degree of closure vary individually, and in a small number of people the tube is constantly open throughout its entire length.

The question of obstruction of a *normally closed* tube naturally leads to a consideration of the means whereby its *active opening* and *normal ventilation* of the middle ear are brought about.

The cartilaginous tube is cradled by muscles.

From its hamular process and its anterolateral aspect the greater part of the tensor palati muscle takes origin (fig. 1).

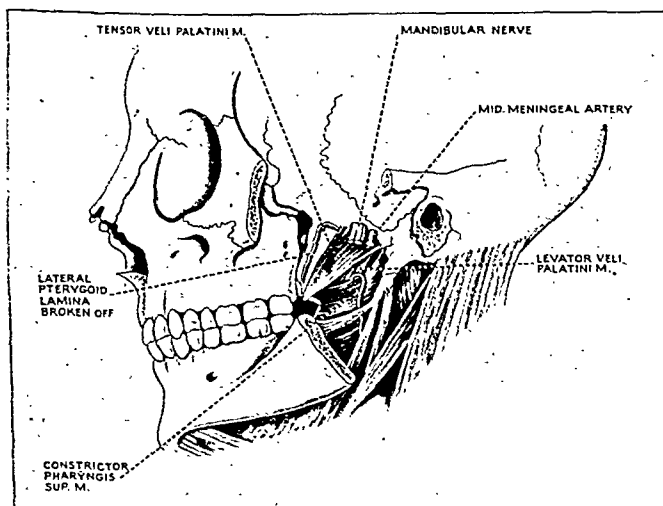


FIG. 1.

It will be seen that 94 patients complained of deafness, and probably had the last six cases been examined sooner after landing a deficiency of hearing would have been found.

I am convinced that some degree of deafness is a constant symptom of the lesion.

In 10 cases the hearing was normal at the time of first examination. In the remainder deafness was present, and this was of the "conductive" type with loss of acuity for low tones; and although some patients could hear a whispered voice at 20 ft. they mostly suffered from some loss of hearing for the forks C and C1, and from 10 to 30 decibels by the audiometer. In three patients only was there any loss for high tones, but not one of the series manifested the severe and permanent "inner-ear" type of deafness mentioned by Wing Commander Simpson.

It is noteworthy also that deafness was usually the last symptom to disappear.

(ii) *Otoscopic appearances* (six hours to seven days after onset).

(a) *Normal drumheads* were seen in 26 patients.

(b) *Invagination of the tympanic membrane* of a varying degree was the most common finding and this was noted in 47 patients. The appearances of this condition are familiar, but I would call attention to the word "*invagination*" which was used by Mr. Sydney Scott (1919) in his report of similar cases observed during the last war. It is more truly descriptive than "*retraction*", as the drumhead is *pushed in* by atmospheric pressure around the middle-ear contents. Invagination of the drumhead was always accompanied by some congestion of the region of the handle of the malleus and of the sheaf of vessels which sweep backwards to the posterior malleolar fold. This vascularity varied in intensity and shaded off into the two following changes:

(c) *Attic congestion* which was present in six cases, and

(d) *Congestion of the entire drumhead*, which was noted in eight patients. These two conditions simulate, and are often mistaken for, acute bacterial otitis media.

(e) *Effusion into the tympanum* was seen in six patients at the time of first examination. Two cases were as usually described, in three patients the effusion was confined to the posterior compartment of the middle-ear, and in one there was a localized effusion into Prussak's space. Two other patients developed tympanic effusion after admission to hospital.

(f) *Rupture of the drumhead* was present in seven patients. Six of the perforations were secondarily infected and moist when first seen, the remaining one was dry. In three patients the lesion was in the posterior segment and in four it was anterior to the handle of the malleus.

(g) *Evidence of old healed middle-ear lesions*—such as atrophic scars, calcareous patches and local or general increased opacity of the tympanic membrane—was present in several patients in addition to the appearances already described, and four patients had submitted to simple mastoidectomy in childhood.

B. Positive Evidence

(iii) *Impermeability of the tube to the passage of air. Inflation or "forced ventilation" of the middle ear*, i.e. passive opening of the Eustachian tube, leads to a consideration of the pressures that can be created in the nasopharyngeal and nasal cavities by different procedures.

(a) *Valsalva's method of inflation* is the generally accepted proof of tubal patency, and its successful performance is insisted on by the Royal Air Force before acceptance for aircrew duties. 21 patients could inflate their middle ears by this method when first examined. The average pressure found to be necessary to cause air-entry through a normal tube was found to be 30 mm.Hg. The average maximum pressure that could be produced by 100 unselected patients was 114 mm.Hg. and the maximum achieved by any one patient was 220 mm.Hg.

(b) *Valsalva's method combined with swallowing movements* was successful in one case in which Valsalva's manœuvre alone had failed. It has been difficult to measure the pressure created by this combined procedure but estimations have been carried out in five patients with a perforated drumhead and patent Eustachian tube. The manometer was connected by an accurately fitting nozzle into the external auditory meatus and, whilst the subject maintained a constant pressure by auto-inflation, when he swallowed the mercury rose on an average 20 mm., and then suddenly fell. I think the success of this method was due to two factors:

is easy and automatic. Most aviators are aware of periodic and involuntary escape of air from their Eustachian tubes. This was first described in 1879 (Hartmann) and more recently by Armstrong and Heim (1937). As a result of experiments they state that at 500 ft. (15 mm.Hg) there is a "click" and air escapes from the Eustachian tube leaving a residual positive intratympanic pressure of 3-6 mm.Hg. and that this escape recurs once at an average ascent of 425 ft.

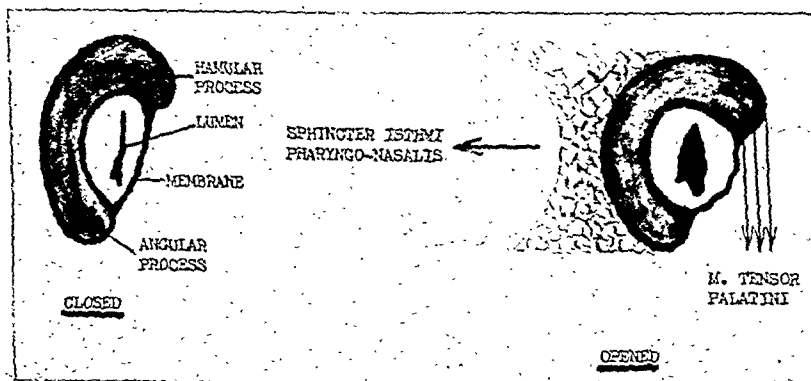


FIG. 3.—Mechanism of tubal opening.

During loss of height in aircraft or increase of pressure in a chamber, the Eustachian tube behaves in an entirely different manner; for, even with a normal tube, air does not enter the middle ear automatically. In the majority of aviators, however, the tube is opened and ventilation of the middle ear brought about by involuntary swallowing movements, which occur at the rate of once every sixty to seventy-five seconds in normal individuals; and in addition all aircrew personnel are taught to practise auto-inflation at every 1,000 ft. loss of height.

Non-ventilation of the middle ear during descent results from either:

- (1) Failure to open the tube, or (2) Inability to open the tube.
- (1) Failure to open the tube may be due to:
 - (a) Ignorance as to the correct means to achieve patency.
 - (b) Lack of opportunity to open the tube, and this has been responsible when energetic evasive action to escape searchlights or anti-aircraft shells has been necessary. *Rapidity of descent per se is not a cause of non-ventilation and is of importance only in this respect.* In a large number of patients of this series symptoms became manifest during a gradual loss of height from 14,000 ft. or 12,000 ft. when returning from an operational flight.
- (2) Inability to open the tube owing to the presence of some existing tubal abnormality may have occurred, but in two patients only was oedema of the tubal ostium observed and in one, an old injury of the torus.

Table II is an analysis of the upper respiratory abnormalities noted in the present series.

TABLE II.

| EXISTING LESIONS OF UPPER RESPIRATORY TRACT | | | | |
|---------------------------------------------|-----|-----|-----|----|
| "Cold" | ... | ... | ... | 6 |
| Deviated nasal septum 18: | ... | ... | ... | |
| Operation performed | ... | ... | ... | 8 |
| Operation not advised | ... | ... | ... | 10 |
| Chronic infection of adenoid | ... | ... | ... | 2 |
| Chronic tonsillitis | ... | ... | ... | 7 |
| Antral infection | ... | ... | ... | 10 |
| Dental lesion | ... | ... | ... | 1 |
| REMOTE LESIONS OF UPPER RESPIRATORY TRACT | | | | |
| History of earache and/or otorrhoea | ... | ... | ... | 10 |
| History of myringotomy | ... | ... | ... | 1 |
| Previous cortical mastoidectomy | ... | ... | ... | 4 |
| Previous injury of Eustachian orifice | ... | ... | ... | 1 |

Its postero-medial wall is in contact with the levator palati muscle and some of the fibres may arise from this wall. This muscle gradually comes to lie on the inferior aspect of the tube to form the floor of its pharyngeal opening (fig. 2).

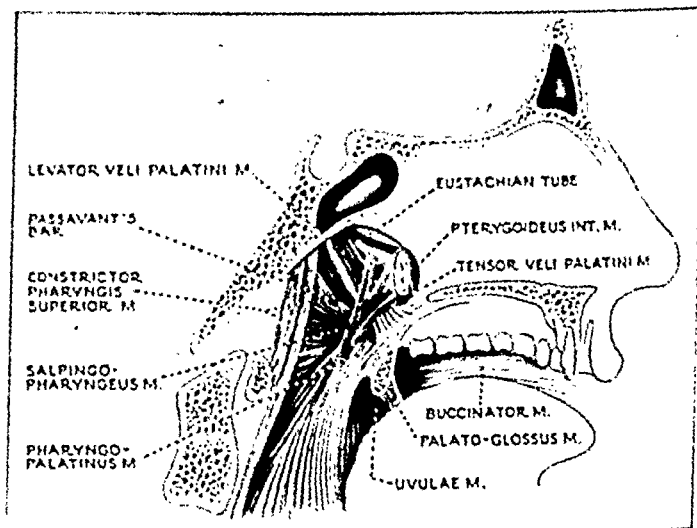


FIG. 2.

The slender salpingo-pharyngeus muscle takes origin from the cartilage near its free extremity, whilst below it is the upper margin of the superior constrictor muscle and that portion of the palato-pharyngeus muscle which forms Passavant's bar (fig. 2).

Functionally the muscles of importance are:

- (1) *The tensor palati muscle;*
- (2) *The superior constrictor muscle in its upper part; and*
- (3) *The sphincter of the pharyngo-nasal isthmus, which is formed by:*
 - (a) *The soft palate anteriorly;*
 - (b) *The palato-thyroidens (posterior pillar) laterally; and*
 - (c) *Passavant's bar posteriorly.*

The mechanism of active opening of the tube has long been controversial, and McMyn (1940) has recently and fully summarized the existing theories. He concluded that the tube is opened by the tensor palati muscle which causes relaxation of its membranous wall, and that other muscles (levator palati, salpingo-pharyngeus, superior constrictor) may act synergically.

My own conception is that the tube is opened in a sinuous manner by the combined action of the sphincter of the pharyngo-nasal isthmus and of the upper part of the superior constrictor muscle which drag the postero-medial cartilaginous wall inward; whilst simultaneously the tensor palati muscle braces the antero-lateral wall against the medial pterygoid lamina and draws down the hamular process of the cartilage to relax the membranous wall (fig. 3).

It is known that the tube is opened by swallowing, yawning and gaping movements; that opening may occur during phonation depending on the amount of contraction of the pharyngo-nasal sphincter; and that certain physiological factors such as posture and exercise influence its patency.

Closure of the tube in the majority of individuals is passive and due to the elasticity of the cartilage.

VENTILATION OF THE MIDDLE EAR DURING FLIGHT

During ascent in aircraft or decrease of pressure in a chamber, as the extratympanic pressure becomes less, egress of air from the middle ear along a normal Eustachian tube

of the recognized forms of inflation, and in many patients such relief has been achieved when the treatment has been applied within a *short time* of the onset of symptoms.

If, however, "forced ventilation" of the middle ear is not available or is neglected for one to two hours, the tube becomes impermeable to air even at high pressures, fluids and sounds as mentioned earlier.

It is reasonable to assume therefore that some biological change has taken place as a result of the primary (pressure) occlusion, and this I have called *Secondary (Vital) Occlusion*.

The nature of these secondary changes is not known but it can be surmised when one views the thickness of the submucosa and the large numbers of mucous glands and goblet cells which are present in the epithelial lining of the cartilaginous tube.

Secondary occlusion is a *very real* entity and it may take as long as fourteen weeks to resolve, as happened in one patient of this series.

The aetiology of this type of aural defect appears to be:

| | | |
|---------------------------------------------|----------------------------------|----------------------------------|
| Failure or inability to open the tube | } → Primary Pressure Occlusion → | (Secondary vital occlusion |
| | | |

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OBSERVATIONS ON AIR-SICKNESS

Squadron Leader R. H. Winfield: The causation of air-sickness may be considered from three points of view—physiological, pathological and psychological; although the condition may be due to any combination of these three factors.

(1) Physiological

When one flies for the first time, whether as pilot, observer or passenger, the labyrinth is subjected to a series of new stimuli caused by the unaccustomed motion of the aircraft; in a proportion of subjects the effect of these stimuli is to cause sweating, pallor, nausea and malaise, which may be followed by vomiting. 5 to 10% of those who fly for the first time vomit more than once during their first twelve hours in the air. In the vast majority of cases, however, a physiological adaptation occurs and air-sickness will then only recur if this adaptation is broken; this may be caused by such factors as a spell of prolonged severe aerobatics, a change in the type of aircraft flown, or by an absence from flying for about six months; again just as sea-sickness occurs in very rough weather amongst experienced sailors, air-sickness may occur in experienced personnel when prolonged flights are made in exceptionally bumpy conditions.

The incidence of air-sickness is influenced by what may be called the accessory factors, and the more important of these are:

- (1) *The weather*.—Air-sickness is obviously more common in bumpy weather, and in peace-time the pilots of civil aircraft are instructed to fly in strata of calm air in order to prevent air-sickness.
- (2) *Type of aircraft*.—Some types of aircraft have the reputation of causing air-sickness more readily than others. Variation of the wing loading is responsible for different types of movement and it is possible that the quick vertical movement associated with low wing loading is a potent factor.
- (3) *Noise and vibration*.—The presence of noise and vibration is an important factor, possibly because it predisposes to fatigue.
- (4) *Ventilation*.—Aircraft which are not properly ventilated tend to be either draughty or stuffy, while the presence of an oily smell from the engines is conducive to air-sickness.
- (5) *Comfort*.—Those travelling by air should be comfortable and not cramped. Room to move about the aircraft and clothing which is warm without constricting are essentials.
- (6) *Diet*.—The quantity and quality of food taken before flying and while in the air

It will be seen that of these 16 patients were suffering from coryza; 8 showed gross septal deformities; 19 had infected tonsils, adenoid or antra; and 1 an old injury—i.e. one-third only of 100 patients were suffering from lesions which *might* have affected the lining of the tube.

If ventilation of the middle ear does not take place from any cause during loss of height a positive pressure, depending on the amount of loss and the altitude at which it occurs, develops outside the middle-ear cleft. Armstrong and Heim (1937) have shown that an extratympanic positive pressure of 90 mm.Hg or more will cause the tube to "lock", as the muscles concerned are unable to overcome this force (fig. 4).

When one considers the cartilaginous portion of a Eustachian tube it is realized how easily its cartilaginous and soft membranous walls can be pressed together, completing the impermeability of what is already only a potential lumen.

This occlusion due to pressure I have regarded as *Primary (Pressure) Occlusion*; and this partially explains the obstruction. If this were all, however, it could be relieved by one

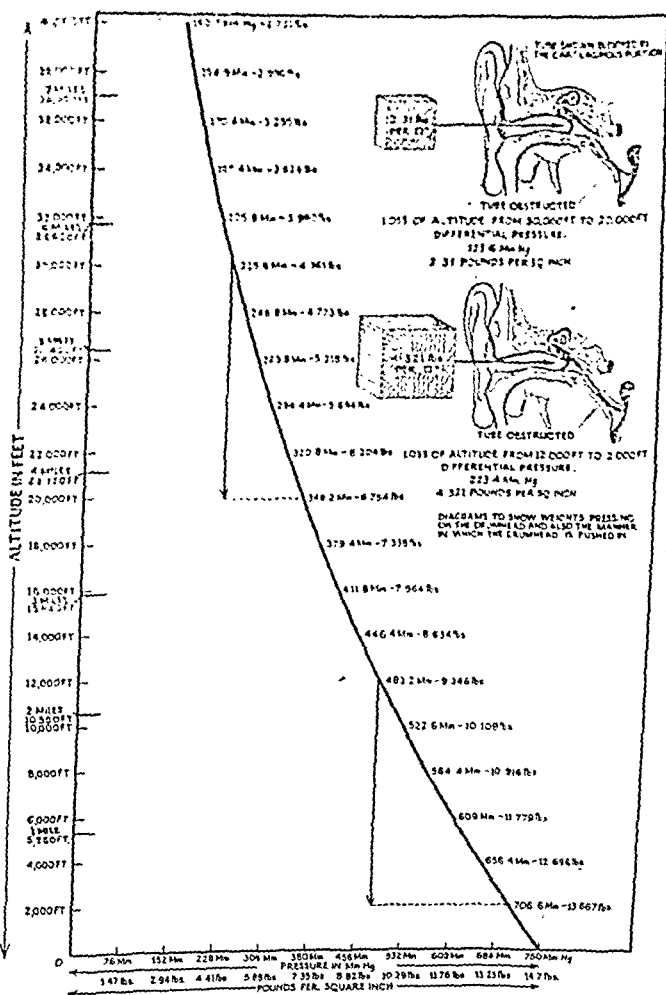


FIG. 4.—To demonstrate the amount of positive extratympanic pressure developed by equal loss of height at different altitudes.

Section of Dermatology

President—H. C. SEMON, M.D.

[November 20, 1941]

Case for Diagnosis. ? Seborrhoeid.—A. C. ROXBURGH, M.D.

Mrs. H., aged 51.

History.—Complains that for three weeks she has had an irregular, blotchy, brown staining of the skin of the face, chiefly of the cheeks, which are slightly scaly, and puffiness of the eyelids. Feels tired but otherwise well. She has a history of pyelitis twenty years ago and later subcapsular abscesses of both kidneys and later still a long period of ill-health with streptococcal infections and high temperatures. She has had two miscarriages. She lived in Egypt for two years during the last war but has not lived in the tropics. Her health of recent years has been reasonably good.

Examination.—The brown colour is seen to be due to the presence in the skin of very minute brown semi-translucent nodules most or all of which appear to be follicular. These nodules are seen best on the upper lip where the intervening skin is abnormally white and opaque. On the cheeks the nodules are confluent and the skin is slightly scaly. There is some redness over the ramus of the mandible down on to the neck on each side but the nodules are not easily seen here. The eyelids are only very slightly puffy. The patient has no evidence of rosacea and her head is not scurfy.

I have seen somewhat similar nodules in the past in cases of rosacea and also in one or two young women who had no rosacea. The latter cases I regarded as a form of seborrhoea. These cases cleared up with a few $\frac{1}{4}$ pastille doses of X-rays and the use of a sulphur and resorcin paste, but I have never been able to ascertain what this condition really is and whether it has been described before. It does not appear to be the "rare seborrhoeid of the face" described by Pringle (*Brit. J. Derm.*, 1903, 15, 41) which was a much more papular condition.

Dr. ELIZABETH HUNT: I would suggest the possibility of infection by *Microsporon furfur*, although I have never known it affect the face. The brown pigmentation and the scaling are suggestive. Cases of *Microsporon furfur* infection are not often seen nowadays but during the last month I have seen two women with it, both cleanly in their habits. Both had pigmentation of the trunk; one had been referred as a secondary syphilide, the other as an unusual case of Addison's anaemia.

POSTSCRIPT.—10.2.42: This patient was given four $\frac{1}{4}$ pastille doses of X-rays and a resorcin and sulphur paste to the face which caused great improvement. Then Dr. Geoffrey Evans prescribed vitamin B₁ and riboflavin. She had two more sixth of a pastille doses of X-rays and then a two weeks' holiday. At the end of this the face was nearly well and the patient's general health very much improved.—A. C. R.

Darier's Centrifugal Migratory Erythema.—A. C. ROXBURGH, M.D.

Mrs. B., aged 40. First seen October 1, 1941, when she had a large red area covering the back of the neck with a festooned lower border raised nearly $\frac{1}{4}$ in. above the surrounding skin and about $\frac{1}{2}$ in. wide. There were smaller similar lesions on the right temple and central forehead. Weekly injections of 10 c.c. of her own blood intramuscularly were advised and of these she had six; by November 12 the lesions had to a large extent involuted but portions of the raised border were still present. On November 12, X-rays ($\frac{1}{2}$ pastille) were given to the raised border and the lesions have now entirely flattened out and only the red staining remains. The patient has a seven years' history of attacks of fever of unascertained cause and has been an in-patient for three months, six months and six weeks respectively in three different London hospitals without the cause having been determined. Sulphapyridine controlled the fever but had no effect, she states,

must be reasonable; small appetizing meals taken frequently are the most satisfactory.

(7) *Ocular stimuli.*—The unaccustomed disagreement between ocular sensations and those from the labyrinth associated with aircraft movement will cause air-sickness in some subjects. This is well seen in those type of aircraft where the navigator sits at a table and is constantly shifting his gaze from the horizon to his logbook. In bumpy weather he may be subjected to a wave of nausea as he bends his head forward over the table.

Other factors such as cold, anoxia and boredom predispose towards air-sickness.

(2) *Pathological*

The presence of any form of organic disease will increase liability to air-sickness. The more common conditions associated with it are sinusitis and otitis and when these have been cured air-sickness ceases.

(3) *Psychological*

If any of the 5 to 10% of people who have been air-sick during the first twelve hours of flying are subsequently placed in an environment to which they cannot adapt themselves air-sickness may recur as a psychological escape mechanism.

It is interesting to note that when a pilot is flying the aircraft himself he is very rarely sick. This is possibly due to two causes: first, his attention is taken up below the levels of consciousness, and secondly, he is able instantly to counteract the movements of the aircraft by anticipation. That this anticipation of movement is an important factor is well shown by the fact that some pilots will become air-sick when flying with the automatic pilot, which anticipates the aircraft movement for them.

TREATMENT OF AIR-SICKNESS

Drug therapy is limited by the fact that whatever drugs are used cortical function must not be depressed. Treatment, therefore, is directed mainly towards taking care of the accessory factors, which have already been discussed.

The subject must be reasonably fit, adequately fed and suitably clad. He requires protection from noise, and oxygen at oxygen heights. The aircraft must be properly heated, satisfactorily ventilated and made as comfortable as possible.

He was taken into hospital; there was no fever, and now the infiltrations of the face and the nodules on the limbs and trunk have largely subsided, leaving pigmented areas. Since his discharge from hospital he has been giving himself injections of alepol and chaulmoogra oil.

Discussion.—Dr. H. C. STANNUS: The patient was brought to me some time ago to confirm the diagnosis. He is now much better than when I first saw him.

The PRESIDENT: Members should bear in mind that the diagnosis of leprosy must be confirmed by demonstrating the bacillus in the skin. It is not sufficient to take a nasal swab and say the case cannot be one of leprosy because there are no bacilli in the nasal mucosa. In only about one-third of the cases are bacilli recovered from this focus.

Dr. STANNUS: The question of infectivity in leprosy has never been satisfactorily answered. Of course there is danger, but usually the association has to be a fairly close one. In some of the old German leper colonies in Africa to which the leper was allowed to bring his wife and children, the whole family developed leprosy in time. Many of the children did not show signs of leprosy for three or four years, but one can say that eventually they all contracted the disease. Native attendants in the colony gave the treatment. Some of them had been there for many years without developing leprosy. There was a fairly close association but not sufficiently close for them to develop the disease. It was one of the most remarkable sights in these colonies to see a woman with loss of hands and feet due to leprosy nursing a beautiful, fat, perfectly normal newborn baby, and saddening to know that such babies if left in the colony would eventually develop leprosy. Since Great Britain took over the German colonies, families are no longer allowed to go into the leper colony with the affected individual.

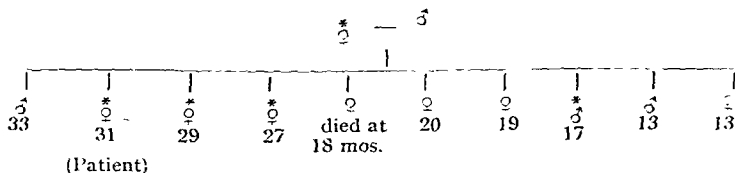
Familial and Congenital Basal-celled Epithelioma in the Distribution of Epithelioma Adenoides Cysticum, and Showing Some of the Histological Changes of the Latter.—LOUIS FORMAN, M.D.

E. S., female, aged 31.

At the age of 15, numerous small nodules appeared around the chin, in the nasolabial folds and in front of the ears, where they are aggregated into small plaques. The nodules are flat or hemispherical and skin-coloured.

Three other siblings and the patient's mother show the same lesions, which appeared at puberty. The two youngest members of the family may develop the disease when they reach puberty.

Section from one of the nodules in front of the ear showed a basal-celled tumour, with cells arranged in columns. One small cyst containing horny material was seen.



Affected members of the family are marked *.

Dermatitis Herpetiformis, with Unusual Limitation of the Disease.—LOUIS FORMAN, M.D.

A. T., female, aged 40.

Thirteen years' history of blisters on the feet and thighs. The blisters ruptured in two days, forming superficial ulcers, and these were diagnosed for a time as artefacts. Three years ago she developed grouped, small, thick-walled blisters on a pigmented patch on the right lower abdomen. There were no eosinophils in the blister fluid and no blood eosinophilia. Potassium iodide gr. v t.d.s. given for a week, provoked erythematous discs on the ankles and knees with numerous thick-walled blisters. 30% iodide ointment applied for five days to the pigmented area on the abdominal wall provoked a fresh crop of blisters.

Scleroderma with Long Antecedent Pigmentation.—GODFREY BAMBER, M.D.

Miss D. B., aged 20. About thirteen years ago began to get patches of pigmentation which have gradually increased in size and number, and three years ago another kind of lesion began to appear in a pigmented part of the left thigh.

on the erythema. In the past she has had ulcers of the tongue, middle-ear discharge, iritis, glaucoma and cataract of the right eye. Her health is now fairly good apart from the erythema.

THE PRESIDENT: In Daper's own description, the lesions have been on the abdomen and flanks in most of the cases. The cause of these peculiar urticarial lesions remains undiscovered.

Severe Chilblains on Right Leg Weakened by Previous Poliomyelitis.—A. C. ROXBURGH, M.D.

Mrs. G., aged 27.

The leg, though weak, is useful and the patient is able to get about easily and play tennis. For the last two to three weeks, since the recent cold spell, she has had a number of cold purple nodules and plaques about 1 inch in diameter on the back and on the lower third of the front of the leg. She states that she has had "ordinary chilblains" before on this leg. She has no chilblains elsewhere. There is no family or personal history of tuberculosis and the lesions seem to be too superficial to be those of Bazin's disease. To-day the lesions have very much improved owing to the warmer weather and are only brownish red in colour. I should be glad of suggestions for treatment, particularly whether any member has experience of bee-venom ointment which was advocated for ordinary chilblains by Watson (*Lancet*, 1941 (i), 301).

Discussion.—**DR. JOHN FRANKLIN:** I should be careful about rubbing in bee-venom ointment. I have seen several cases of severe local dermatitis in patients using it for rheumatism. I should be inclined to try galvanic baths, which often help chilblains and should be particularly useful in this type of case.

DR. F. PARKES WEBER: I suggest that the case is one of the chilblainy type of "erythrocyanosis crurum feminarum frigida", which is always bilateral excepting when one leg is a *situs minoris resistentiae*. The main treatment in such cases should be preventive by protecting the limb from cold.

DR. A. C. ROXBURGH: I thank Dr. Franklin for the warning about bee-venom ointment. As to Dr. Parkes Weber's remarks, I should have thought that chilblains of one sort or another on an old "polio" leg were quite common. The patient said they were previously "ordinary chilblains"; that is not my expression.

Acute Lichen Planus.—A. C. ROXBURGH, M.D.

C. L., male, aged 19, sheet metal worker. Acute lichen planus of two weeks' duration, very widespread, confluent over large areas of the trunk and affecting both the penis and the mucous membranes of the cheeks. A big, strong, apparently perfectly healthy youth with no worries. For the last two to three months he has been working twelve hours a day for five days a week and eight hours a day on Saturdays but does not complain of being tired. Can any member report good results from large doses of vitamin B₁ which have been recommended in acute lichen planus?

POSTSCRIPT.—10.2.42: This patient continued at work and was given twice weekly an intramuscular injection of Vitamin B₁ (Benerva 1 c.c.) and took tablets of Benerva 1 mg. thrice daily by mouth. In two weeks the rash was flatter and becoming pigmented, and it has continued to do so till last seen at the end of January. Three or four other patients with fairly acute lichen planus have improved similarly on vitamin B₁.—A. C. R.

Leprosy.—LOUIS FORMAN, M.D.

H. S., male, aged 69.

Lived on the Malabar coast for thirty years. Married, wife and four adult children all well.

Ten years ago he noticed an area of anaesthesia on the right leg, and received treatment for leprosy. Eighteen months ago he developed a general eruption on the face and trunk with fever. When seen a year ago, there was diffuse infiltration of the skin of the face with definite nodules on the forehead and infiltrated plaques and papules on the trunk and limbs. The nasal mucosa was thickened and bacilli were found in the discharge. Save for an absent left plantar reflex and blunting of sensation in the lower leg, there was no abnormality of the nervous system. The nodules on the face were not anæsthetic.

Section from a nodule on the back showed an infiltrate of endothelial cells in the cutis, a sarcoid reaction; leprosy bacilli were scanty.

complained of. The face was described as being "tight". (2) Shrinkage had occurred gradually; the clawlike tips of the hands had taken four years to develop; shrinkage of the face had taken place two years ago and of the body one year ago. (3) The hands were now hard to the touch, but there was no true process of sclerodermia present. (4) The appearance of the finger-tips and X-ray changes were as described by Sellei. (5) Telangiectasia and atrophic appearances of the skin were present elsewhere on the body.

Discussion.—Dr. PARKES WEBER: The case is a typical example of what was formerly termed "symmetrical atrophic sclerodermia with sclerodactylia". The muscular atrophy in the hands and the bony absorption in the terminal phalanges has been commonly supposed to be secondary to disuse and pressure from the hide-bound condition of the integument, but there may be other causative factors.

Dr. R. KLABER: I was impressed by the degree of wasting, which seems to involve not only the subcutaneous but also the muscular tissues. Is that degree of wasting a feature of either acrosclerosis as described by Sellei, or of generalized sclerodermia?

Dr. W. FREUDENTHAL: One thinks that sclerodermia might affect the muscle secondarily just by pressure of the skin getting tighter, but it seems also possible that the muscle is primarily involved. The aspect of the question has been widened since dermatomyositis has been included in the sclerodermia group. See papers read by T. Lewis, G. B. Dowling, W. Freudenthal and W. J. Griffiths, *Brit. J. Dermat.*, 1940, 52, 233, 242, 289 and 296.

The PRESIDENT: I remember reading of a post-mortem on one of the cases in which it was pointed out that not only were the muscles of the skin involved in the sclerodermatous process but also the endocrine glands, particularly the adrenal cortex, so that a general process must be hypothesized.

Dr. FREUDENTHAL: There are not very many cases of generalized sclerodermia on record in which a microscopic post-mortem examination of the endocrine glands has been done. Amongst these, as Dowling has pointed out, the thyroid gland was found to be nearly always involved.

Dr. GORDON: I cannot agree that this patient can be considered as a case of generalized sclerodermia. Her skin is unthickened all over the body. Though the hands are clawlike, this is due to a general process of shrinkage.

The PRESIDENT: I agree. The extremities are picked out in this case.

Ehlers-Danlos Syndrome.—HUGH GORDON, F.R.C.P.

Patient aged 46, single, housemaid.

She attended hospital two months ago with a history of falling in the street and splitting the skin of her right calf. This was said to have bled profusely, though bleeding was not apparent on examination.

The skin tear measured 2 in. and it was noticed that the deep fascia had also split about $\frac{1}{2}$ in. This wound healed by granulation within a month.

She reported that this skin splitting on trivial trauma had been common in the past. Bleeding was often profuse and had on occasions to be controlled by suture. She stated that she had bled profusely from a tonsillectomy and teeth extractions and, in addition, various members of her family had bled to death after fairly trivial trauma. Numerous operations had been performed on the feet as an infant.

On examination.—There are numerous linear scars on the subcutaneous surfaces of both ulnas, extending from the wrists to the elbows. On the back of the right wrist is an incompletely healed wound with a lump the size of a plum underneath it. This is stated to have been a spontaneous split like the others, which has not healed. There are similar scars on the legs on the extensor surfaces and numerous operation scars on the feet. There is marked hyperextensibility of the thumbs of both hands and to a slight degree also of the fingers. It is impossible to say what the state of the feet would normally be owing to the surgical operations which have been performed, nor was it possible to find out for what they were performed. The skin can be said to be hyperelastic over the elbows. No other abnormalities.

Bleeding time 2 minutes. Clotting time 90 seconds. Platelets normal.

Comment.—I was first shown this case by the surgeon under whose care she was admitted, as a possible artefact, which seemed a probable diagnosis, especially in view of the fact that the majority of the scars were on her right side and she was left handed, although the scars were not the typical broad papyraceous type over the knees and elbows, but were fairly linear.

Inspection showed deep pigmentation covering large areas on the lower half of the trunk and smaller patches with indefinite outlines on the upper half and on the proximal parts of the limbs. On the right side of the forehead was another pigmented patch, and examination of the face by Wood's light revealed fairly sharply defined areas of pigment on the other side of the forehead and on the cheeks, a distribution suggesting chloasma.

On the inner side of the left thigh were two nummular patches of scleroderma, and a fresh area was developing in pigmented skin above the left anterior axillary fold.

Over the upper part of the chest were dilated veins which had been present for years.

General health good. Catamenia regular. B.P. 120/70. No pigmentation of mucous membranes. R.B.C., no abnormality. W.B.C. 7,550, polymorphonuclears 58%, lymphocytes 30%, large monos. 7%, eosinophils 3%, basophils 2%.

Biopsy.—Dr. Freudenthal reported: (a) A pigmented area: Except for greatly increased melanin pigment the skin was normal. (b) The edge of a scleroderma lesion: One end of the section showed well-developed scleroderma with condensed collagen bundles, elastic fibres diminished and partly destroyed, rete pegs flattened. Amount of pigment normal or diminished. The other end showed a great increase in pigment but no obvious signs of scleroderma.

Discussion.—The PRESIDENT: I have under my care two elderly women with circumscribed scleroderma, whose lesions are situated in the perineum and, curiously enough, there are two strange atrophic lesions under the clavicle. One of these cases has gone on to almost complete atresia of the vagina.

My intention is to try to relieve the pruritus with thorium X ointment; it is extremely severe and keeps the patient awake.

The atresia above mentioned had been treated by a gynaecologist who did not realize he was dealing with scleroderma. The stitch wounds took months to heal and the patient was not relieved by the operation.

Acrosclerosis Scllei.—HUGH GORDON, F.R.C.P.

Patient, aged 49, unmarried, telephonist.

History.—Six years ago she complained of pains in the arms, which shot down to the fingers; these became red and inflamed, and small ulcers appeared on the finger-tips, which healed in a few days. The condition was regarded as Raynaud's disease, though no phase of pallor was recorded.

Physiotherapy was carried out for two years with very slight benefit. During the last year her general condition has deteriorated, she has lost weight and has pains in the chest and shortness of breath. The condition of the hands remains unchanged except that they are getting stiffer.

On examination.—She is a wasted woman, with marked wrinkling around the mouth and slightly mask-like facies.

Wasting of the body and the limbs appears to be due to loss of subcutaneous tissue and, possibly, muscle. Except on the hands, there is no suggestion of induration of the skin, which is freely movable and normal to the touch. Pigmentation and telangiectasia were present over the chest and back. The hands were slightly clawlike and hardened. This process was most marked on the terminal phalanges and spread upwards to above the wrist. The hands are bluish in colour and on the tips of all the fingers are stellate scars; the nails are atrophied. X-ray of the hands shows absorption of the terminal phalanges. The mucous membrane of the mouth appears slightly atrophic. General examination is essentially negative. Electrocardiogram normal. X-rays of the chest and heart are within normal limits. W.R. negative. Blood calcium is within normal limits.

The President showed a case in 1936 (*Brit. J. Dermat.*, 48, 653) as "Acrosclerosis (Scllei) connected with Raynaud's phenomenon". In this case, however, there was no sclerodermic process. I showed a case in 1937 (*Proc. Roy. Soc. Med.*, 31, 262 (Sect. Derm., 22)) with well-marked sclerodactylia and mask-like appearance with shrinking of the skin around the mouth. I pointed out at that time that the sclerodermic process which was present, appeared to be secondary to a true scleroderma which had started on the forearms, and had spread down to the hands. In the discussion the general opinion was that Scllei's subdivision could not be substantiated and that cases of scleroderma were common in which sclerodactylia and shrinkage of the face were well-marked features.

My reasons for putting forward the suggestion that this present case is a true acrosclerosis Scllei (so-called) are: (1) The process started apparently six years ago on both hands and face; in the case of the hands, vasomotor changes were present and pain was

complained of. The face was described as being "tight". (2) Shrinkage had occurred gradually; the clawlike tips of the hands had taken four years to develop; shrinkage of the face had taken place two years ago and of the body one year ago. (3) The hands were now hard to the touch, but there was no true process of sclerodermia present. (4) The appearance of the finger-tips and X-ray changes were as described by Sellei. (5) Telangiectasia and atrophic appearances of the skin were present elsewhere on the body.

Discussion.—Dr. PARKES WEBER: The case is a typical example of what was formerly termed "symmetrical atrophic sclerodermia with sclerodactylia". The muscular atrophy in the hands and the bony absorption in the terminal phalanges has been commonly supposed to be secondary to disuse and pressure from the hide-bound condition of the integument, but there may be other causative factors.

Dr. R. KLABER: I was impressed by the degree of wasting, which seems to involve not only the subcutaneous but also the muscular tissues. Is that degree of wasting a feature of either acrosclerosis as described by Sellei, or of generalized sclerodermia?

Dr. W. FREUDENTHAL: One thinks that sclerodermia might affect the muscle secondarily just by pressure of the skin getting tighter, but it seems also possible that the muscle is primarily involved. The aspect of the question has been widened since dermatomyositis has been included in the sclerodermia group. See papers read by T. Lewis, G. B. Dowling, W. Freudenthal and W. J. Griffiths, *Brit. J. Dermat.*, 1940, 52, 233, 242, 289 and 296.

The PRESIDENT: I remember reading of a post-mortem on one of the cases in which it was pointed out that not only were the muscles of the skin involved in the sclerodermatous process but also the endocrine glands, particularly the adrenal cortex, so that a general process must be hypothesized.

Dr. FREUDENTHAL: There are not very many cases of generalized sclerodermia on record in which a microscopic post-mortem examination of the endocrine glands has been done. Amongst these, as Dowling has pointed out, the thyroid gland was found to be nearly always involved.

Dr. GORDON: I cannot agree that this patient can be considered as a case of generalized sclerodermia. Her skin is unthickened all over the body. Though the hands are clawlike, this is due to a general process of shrinkage.

The PRESIDENT: I agree. The extremities are picked out in this case.

Ehlers-Danlos Syndrome.—HUGH GORDON, F.R.C.P.

Patient aged 46, single, housemaid.

She attended hospital two months ago with a history of falling in the street and splitting the skin of her right calf. This was said to have bled profusely, though bleeding was not apparent on examination.

The skin tear measured 2 in. and it was noticed that the deep fascia had also split about $\frac{1}{2}$ in. This wound healed by granulation within a month.

She reported that this skin splitting on trivial trauma had been common in the past. Bleeding was often profuse and had on occasions to be controlled by suture. She stated that she had bled profusely from a tonsillectomy and teeth extractions and, in addition, various members of her family had bled to death after fairly trivial trauma. Numerous operations had been performed on the feet as an infant.

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DERMATOLOGY



"And there the snake throws her enamel'd skin."
(SHAKESPEARE)

To the lower order Nature provides the power periodically to cast the skin. To the human species she does not offer such a simple process. It is the function of Science to deal with Nature as she is. The natural tendency to skin infections is aggravated by present-day conditions of public crowding in shelters, camps, factories, transport and the like. As a result of the consequent demand for rapid and effective treatment, EVANS DERMATOLOGICAL PRODUCTS are being prescribed on an increasing scale by general Practitioners, in Hospitals and by Public Health Authorities.

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| Blepharitis | Pediculosis | Collozin |
| Boils | Prickly Heat | Evans Dermal Powder |
| Burns | Pruritus | Kalsolac |
| Carbuncles | Scabies | Manganese Butyrate |
| Cellulitis | Seborrhoea | Medisoaps |
| Chilblains | Sunburn | Sarevan |
| Comedones | Sycosis | Streptocide Cream |
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Since Dr. Parkes Weber's paper in 1936 (*Brit. J. Dermat.*, 48, 609) quite a number of cases have been shown or reported in this country. I have no doubt that this is a case of Ehlers-Danlos syndrome.

Of the triad of symptoms, splitting is the only one at all well marked in this case, though the others are present. Excessive bleeding from the mucous membranes such as she describes is not usually a feature of this disease; there is no evidence of any abnormality in her bleeding or clotting time although the wounds as seen had certainly oozed rather more than usual.

Discussion.—Dr. W. FREUDENTHAL: Histologically the nodule situated in the subcutis and extending into the cross-striated muscle below consists of loose, irregularly arranged bundles of connective tissue which has in many places a greatly increased number of cells. The tissue contains a large number of capillary, small and medium-sized blood-vessels and large spaces mostly filled with blood with a scanty endothelial lining and strands of hyaline masses in the neighbourhood. Elastic fibres are almost entirely absent.

Dr. PARKES WEBER: The case is a genuine example of the Ehlers-Danlos syndrome, as it shows the three main features, though to a very minor degree. The splitting of the skin and broad atrophic scarring after traumata are only moderately marked. The "cutis laxa" is present only over the elbows, where it may be seen almost to the same extent in otherwise normal individuals. Over-extension of joints can be obtained only in the thumbs, where it is common (so-called "double-jointed thumbs"). The case is specially interesting, showing, as it does, how lesser forms of this developmental dysplasia are sometimes present in individuals who can be regarded as almost normal.

Chronic Recurrent Herpetiform Stomatitis.—GEOFFREY DUCKWORTH, M.R.C.P.

L. J., a draughtsman, aged 27, has complained of slightly painful ulcers in the mouth for the past six years. He has seldom been free from one or more during this time. The lesions develop as small vesicles, the roofs of which separate leaving superficial ulcers, sharply margined with a bright red halo. There is no induration, and healing usually occurs in two or three weeks, though occasionally one will persist longer. Scarring does not occur. The tongue, the inner surfaces of the lips, the buccal mucosa, and the palate have all been affected. The floor of the mouth is a favourite site. Fever is unusual. Sometimes the glands, submental and submaxillary, are a little enlarged and tender.

His health otherwise is good, and he has not had any serious illnesses. Six months after the present trouble began he had his teeth seen to and a few metal fillings were inserted. At present his dentist thinks the teeth need no attention.

Scrapings from ulcers showed short-chained streptococci (non-haemolytic) and a few *M. catarrhalis*, at one time, and Gram-positive lanceolate diplococci (? pneumococci), with a few short chains of streptococci, at another time. No Vincent's organisms have been identified.

The blood picture when last examined was: R.B.C. 4,620,000 per c.mm., Hb. 86%, C.I. 0.93, W.B.C. 7,400 per c.mm., polymorphonuclears 59%, lymphocytes 33%, mononuclears 7%, eosinophils 1%. W.R. negative.

A fractional test meal gave normal curves. An X-ray of the chest showed no evidence of pathological changes in the lungs. The heart's shadow was a little increased on the left side.

Alkaline powders, sedative and tonic mixtures, sulphanilamide, sulphapyridine, nicotinic and ascorbic acids, injections of aneurin, and bismuth, have been given at various times, but no material changes have followed. Locally, sedative applications have been the rule, with occasional applications of phenol. Inoculation of fluid from an early vesicle has not been tried yet.

Discussion.—Dr. L. FORMAN: Sutton, commenting on the results of treatment in two cases of recurrent ulcers of the mouth, stated that the ulcers cleared up more quickly with sulphathiazole than with any other treatment. He thought it the best method he had used for controlling these painful ulcers. ("Recurrent, Scarring, Painful Aphthae: Amelioration with Sulfathiazole in 2 Cases", Sutton, R. L., 1941, *J. A. M. A.*, 117, 175.)

Dr. STANNUS: The case might be one of recurrent herpes. Has any effort been made to identify the virus of herpes? This should not be very difficult. Has Dr. Duckworth tested the gastric acidity and are there any blood changes? A condition closely resembling that seen in this case occurs in sprue and some nutritional diseases occasionally seen in this country. Some will clear up with nicotinic acid. The only suggestion I would make is that you combine riboflavin with nicotinic acid.

Dr. DUCKWORTH: The patient has had a fairly long course of M & B 693, and nicotinic acid.

Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D., M.R.C.P., D.P.H.

[October 24, 1941]

The Trend of Fever Hospital Practice

PRESIDENT'S ADDRESS

By E. H. R. HARRIES, M.D., M.R.C.P., D.P.H.

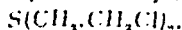
"MIDDLEMARCH," one of the later novels of George Eliot, first published serially in 1871-72, has a certain medical interest because of the allusions to Edward Jenner, Louis, Bichat, Wakley, and others; and to infectious diseases, including the first cholera epidemic of 1831. Middlemarch is a provincial town, and a secondary plot turns upon a new hospital to be provided in addition to the Old Infirmary, and upon a medical man Lydgate. In 1829 Lydgate had at the age of 27 settled in the town fresh from his studies in Edinburgh and Paris, and fired by the work of Louis, whose book on typhoid fever had, of course, appeared in 1829. Opposition to the new hospital was incurred and funds were refused on the score that people preferred to give to the Old Infirmary and thus its construction, mooted in the first "had lingered" into the second volume of the novel. Meanwhile the would-be patron Bulstrode "framed his plan of management". It reads as follows:

"The Hospital was to be reserved for fever in all its forms; Lydgate was to be chief medical superintendent, that he might have free authority to pursue all comparative investigations which his studies, particularly in Paris, had shown him the importance of, the other medical visitors having a consultative influence, but no power to contravene Lydgate's ultimate decisions; and the general management was to be lodged exclusively in the hands of five directors . . . who were to have votes in the ratio of their contributions . . . no mob of small contributors being admitted to a share of government. There was an immediate refusal of every medical man in the town to become a visitor at the Fever Hospital. 'Very well', said Lydgate to Bulstrode, 'we have a capital house-surgeon and dispenser, a clear-headed, neat-handed fellow; we'll get Webbe . . . as good a country practitioner as any of them, to come over twice a week and in case of any exceptional operation, Protheroe will come from Brassing. . . . The plan will flourish in spite of them and then they'll be glad to come in. Things can't last as they are; there must be all sorts of reform soon and then young fellows may be glad to come and study here.'" Lydgate, it is added, was in high spirits which, it must be observed, was unusual for him. In the end, more than 300 pages later, Lydgate leaves the town under an undeserved cloud. He specializes in gout and becomes wealthy, but dies at the age of fifty, surprisingly, but perhaps appropriately enough, of diphtheria.

Although the action of the book passes in the early eighteen-thirties it seems to me not improbable that George Eliot in introducing the theme of a new fever hospital had in mind the hospitalization of the infectious sick, which culminated in the passing of the Sanitary Act of 1866 empowering local authorities to erect and maintain infectious

THE CHEMICAL ACTION OF 'MILTON' ON MUSTARD GAS

Pure "Mustard Gas" is an organic chemical compound known as 2·2·1-Dichlorodiethyl Sulphide, and its constitution is represented by the formula,



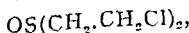
It is an almost colourless oil with a faint odour reminiscent of that of garlic or mustard. It has a boiling point of 217° C. at normal atmospheric pressure and, on cooling, the liquid freezes at 14·4° C. and crystallises in long colourless prisms. Mustard Gas is ordinarily a very stable compound, and may be described as the most aggressive organic chemical known towards human tissues.

The public have received detailed instructions from the Ministry of Home Security through the official handbooks and from the Local Authorities regarding the steps they should immediately take if, unfortunately, they become contaminated by mustard gas through enemy action. The importance of members of the public familiarising themselves with, and carrying out the procedure recommended officially for avoiding the effects of, mustard gas contamination cannot be too strongly emphasized.

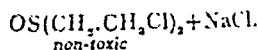
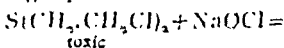
See Air-Raid Precautions Handbook No. 1 (2nd Ed. 1941). Personal Protection against Gas. Page 61—under the heading of Anti-Gas Treatment of Persons.

Any additional and convenient method which can be recommended with confidence for avoiding or minimising the effects of mustard gas contamination is highly important. "Milton" is a readily available material, the value of which for effectively cleansing the skin which has become contaminated with Mustard Gas has been conclusively demonstrated.

"Milton" is a solution of electrolytically-prepared sodium hypochlorite (NaOCl) which reacts rapidly with either pure or crude Mustard Gas at the ordinary temperature, converting it quantitatively into the non-toxic colourless crystalline 2·2·1-Dichlorodiethyl Sulphoxide,



m.p. 116° C., according to the following equation:



Applied rapidly and in proper fashion, the effectiveness of "Milton" against skin contamination can be guaranteed. Garments which are contaminated with Mustard Gas should be discarded at once and treated not with "Milton" but by the methods and at places which the Authorities have provided.

Any treatment applied to the skin which has been contaminated must be *immediate* as well as thorough. If "Milton" is available, it can be used with confidence. After removal of clothing (if necessary) the part affected should be wiped without spreading the area of contamination, using cotton wool or other suitable material which can then be discarded (burnt, or left for some time in a solution of "Milton") so as to get rid of as much of the Mustard Gas as possible. Without delay the affected part is swabbed gently but copiously with full-strength "Milton," the swabbing being continued for about 15 minutes, and then with "Milton" diluted with about four times its volume of ordinary tap water. Continue the gentle swabbing with this dilution at half-hour intervals three or four times. It has been found suitable to treat the affected part finally with 'calamine' lotion and bandage lightly.

If, unfortunately, it has not been found possible to apply the treatment within five minutes, a light mustard-gas burn may ensue after some four to six hours. If this should have happened, the treatment may nevertheless safely be continued at intervals with diluted "Milton" (1 in 5) until the healing process is established. The use of "Milton" as suggested, will diminish the inflammatory or destructive effect and accelerate healing.

The above may be described as home or emergency treatment for the skin and can be recommended in the great majority of cases.

Poor Law patients were admitted: in that year the civil disability was removed by the Local Government Board. Subsequently other infectious diseases were made admissible piecemeal by orders of the L.G.B.

Between 1870 and 1871 three fever hospitals were opened in London just in the nick of time, for the pandemic of smallpox which raged from 1870 to 1874 was, J. D. Rolleston (1937) says in his FitzPatrick Lectures, "the most malignant and extensive outbreak since the introduction of vaccination" (i.e. in 1796). During 1870-74 the new hospitals of the M.A.B. admitted 16,366 cases of smallpox: of these 3,064 were fatal, a case fatality rate of 19%. Although typhus and relapsing fever (one year only) were admissible to the Board's hospitals, the annual numbers did not approach some of Murchison's figures. The highest total was reached in 1874. Thereafter the numbers declined rapidly and the last admissions of typhus (3 cases) were recorded in 1910. *Absit omen!* Goodall (1934) in his "Short History of Epidemic Infectious Diseases" says that in England and Wales enteric fever reached its peak mortality in 1875. In that year 8,913 deaths from enteric fever (besides 1,599 from "continued fever") occurred in England and Wales. Basing his conclusion on a fatality rate of 16%, Goodall assumed that some 56,000 cases of enteric fever must have occurred in the country in that year. Enteric fever declined much more slowly than typhus, and bad years occurred in 1895 and again in 1905, when admissions to the M.A.B. hospitals reached the peak figures of 661 and 586 respectively. It is to be borne in mind that from 1881 until at any rate 1902 it was the practice of the M.A.B. ambulance service to remove, when desired, enteric fever patients from their homes to general hospitals and thus the M.A.B. hospital records only represent a proportion of these patients hospitalized. As late as 1910 the M.A.B. admitted only 51% of the cases of enteric fever notified in London.

In 1875 the great Public Health Act was passed and a commencement was made in the abatement of grosser nuisances, and this was the cause of the decline in prevalence of the continued fevers, louse-borne or water-borne, as they are now known to be.

THE CLOSE OF THE FIRST PHASE

The first phase of fever hospital practice which began with the century was drawing to a close. It was the pre-bacteriological era and medical and administrative practice was simple; separate wards, if accommodation permitted, for infectious diseases other than enteric fever, which was nursed with non-infectious diseases of mistaken diagnosis. The ventilation, "which is universally admitted to be necessary for preventing typhus spreading in a general ward was", said Murchison, "injurious to patients suffering from many diseases, such as nephritis, acute rheumatism and bronchitis", and therefore presumably would be discouraged in other than typhus wards. As to the danger to the nurses, Murchison relied upon a supply "protected by a previous attack of typhus or of an age at which it is not very likely to be fatal". Presumably those engaged in nursing smallpox were vaccinated, and although Murchison distrusted scarlet fever (diphtheria is not mentioned) he appeared to be unduly optimistic about enteric fever. Nowadays, of course, it is the usual practice to protect the nursing staff not only against smallpox, but to immunize them against diphtheria (Harries, 1930) and scarlet fever (Harries, 1935) and the enteric group of fevers. Recently, too, selected members of the staff of my own hospital have been inoculated against epidemic typhus.

THE OPENING OF THE SECOND PHASE

The second phase of fever hospital practice opens in the 'eighties, which also saw the commencement of the bacteriological era (Eberth, typhoid bacillus, 1880; Klebs, diphtheria bacillus, 1882; and Klein, streptococcus, 1887). For the next thirty or forty years fever hospital practice was dominated by scarlet fever; for smallpox, of course, then as now, separate provision was made. In 1882 Thorne observed that "scarlet fever had rarely been the immediate cause of hospital provision and this notwithstanding the fact that the mortality it occasions has been so greatly in excess of that resulting from smallpox". From 1866 until 1880 only some thirty local authority hospitals had been provided but Franklin Parsons (1914) remarks that during the course of the next two decades scarlet fever had come to be looked upon as the disease *par excellence* requiring hospital isolation. Some hospitals were reserved exclusively for it, more urgent cases of diphtheria and enteric fever not being admitted, and "in most isolation hospitals cases of scarlet fever formed the great majority of those treated". From 1881 to 1890 101 isolation

diseases hospitals, and the Metropolitan Poor Law Act of 1867 which resulted in the constitution of the Metropolitan Asylums Board

However this may be, Murchison (1862) devotes the last chapter of his classic on the "Continued Fevers of Great Britain" based upon his experience as physician to the London Fever Hospital to a discussion on "the relative merits of isolating fever patients and of distributing them in the wards of general hospitals". Some held that "it would be better to have no hospitals at all than to mix cases of typhus, smallpox and scarlet fever with patients suffering from other diseases", while others roundly declared that "fever hospitals and fever wards are at all times a crime against humanity and a disgrace to the age in which we live". A demand was made for the closing of the London Fever Hospital which had been opened with other "houses of recovery" in the provinces and in Ireland at the beginning of the century primarily to cope with epidemics of typhus. Among the conclusions reached by Murchison are that "fever hospitals are absolutely necessary in all large towns liable to epidemics of typhus, and that they ought to be provided with means of rapid extension; that in all general hospitals there should be, where possible, arrangements for the treatment of patients suffering from contagious fevers . . . but these patients ought not to be interspersed through the general wards but isolated in separate wards or better in a detached building". (John Haygarth of Chester had expressed much the same views in 1778.) Cases of enteric fever, however, Murchison thought might be distributed in the wards of a general hospital with impunity. Nor did he see any objection to the many cases of acute non-contagious diseases, constantly being sent in by mistake to fever hospitals, being treated in the same wards with enteric fever. Murchison's arguments for the retention of fever hospitals were received coldly by Simon, who accepted the Report of Bristowe and Holmes (1863). They were of opinion that "scattering the cases is the safest course if the hospital be spacious and the applications not too numerous" (surely a counsel of perfection in the conditions of the times), and emphasized that for safety large well-ventilated wards and beds well removed from each other are essential. Murchison (1864) argued that "whatever plan be adopted, that is [*sic*] mixing or fever wards, the attendants upon patients suffering from smallpox, scarlet fever and typhus must run a certain amount of risk". He pointed out that the objections urged against fever wards were that the concentration of the poison increases among the patients themselves and through the same cause the attendants are endangered. He advocated two thousand cubic feet of space per patient, a standard then obtaining at the London Fever Hospital, but concluded that "however perfect the ventilation of a hospital, smallpox and scarlet fever will spread among persons not protected". Murchison advised separate wards for smallpox, scarlet fever and typhus, because patients convalescent from these diseases are liable to contract the others. Separate wards for these diseases were, however, only introduced at the London Fever Hospital at the beginning of 1862, patients suffering from enteric fever being nursed in the same wards with non-infectious cases; the same "night-chairs" were shared and disinfectants were rarely used. A total of 3,555 enteric fever patients were nursed with 5,144 cases of non-infectious sick at the London Fever Hospital and not one of the latter contracted enteric fever. It may be added that the attendants were not so fortunate.

Murchison (1873) says that since 1862 London had been visited by great epidemics of typhus and relapsing fever, and that to meet the demands for beds the accommodation of the London Fever Hospital—then as now a voluntary hospital—was more than doubled: from 180 beds in 1864 it increased to 364 beds in 1869. During 1864, 2,500 cases of typhus were admitted and this appears to have been the peak year for admissions of this disease. In 1870 the Hospital admitted 631 cases of typhus, 903 of relapsing fever (which had reappeared in 1868, attained its peak in 1869 and finally disappeared in 1872), and 595 of enteric fever. In 1871 all pauper patients were transferred to the "New Fever Asylums", the first of which, the North-Western Hospital, had been opened in 1870 and during that year admitted 218 cases of relapsing fever. The origin of the "New Fever Asylums" is to be found in the great epidemics just mentioned, and the consequent appalling conditions under which the infectious sick were nursed in the London work-house infirmaries. These conditions, as already mentioned, resulted in the passing of the Poor Law Act of 1867 and the formation of the Metropolitan Asylums Board, merged sixty years later into the London County Council. The M.A.B. was empowered to erect hospitals "for the reception and relief of poor persons who may be infected with or suffering from fever or the disease of smallpox or who may be insane". Until 1883 only

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these patients were transferred to convalescent hospitals for the completion of desquamation, thus making room for more patients in the acute hospitals but tending to prolong the average length of stay.

During the last twenty years or so the duration of stay of patients in general, and of scarlet fever patients in particular, in many fever hospitals has been reduced very considerably. The traditional period of detention of six weeks is now four weeks or less for the uncomplicated case of scarlet fever, with, of course, a reduction in the average length of stay. Return case-rates have certainly not increased as a result of the shorter stay; in fact, return cases appear now to be few and far between, judging from the experience in my own hospital. The factors which have brought about this shortened period of stay with consequent release of beds for other diseases may be enumerated as follows:

- (1) The mildness of the disease with few cases of septic type.
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- (3) Maintenance of proper spacing (12 ft. of wall space) and avoidance of encroachment upon this spacing even in times of prevalence, shown to be equally important by J. A. Glover (1932, 1934) in other environments.
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investigation on diphtheria carriers, and as a result a carrier clinic has been maintained at the North-Eastern Hospital ever since. Especially in the case of release cultures, the use of selective tellurite media, the relentless search for the ultimate diphtheria bacillus, cause increased difficulties for the fever hospital clinician. Time marches on, and the child who enters a hospital harbouring a few diphtheria bacilli, to the toxin of which he can frequently be shown immune by the Schick test may remain many weeks before he attains bacteriological righteousness as determined by modern media. In my own hospital we have arrived at a compromise: tellurite plates for confirmatory diagnosis and Löffler medium for release cultures. I am sure that this procedure does not result in an undue acceleration of the first of the "velocities of infection" postulated by Sheldon Dudley (1926).

THE THIRD PHASE

The third phase of fever hospital practice may be said to commence during the first decade of the present century. From 1888 scarlet fever, diphtheria and enteric fever were the three notifiable diseases, other than smallpox, admitted to the M.A.B. hospitals. But Cuff (1910) reported upon the desirability of using some of the spare accommodation in the fever hospitals for the treatment of measles, whooping-cough and puerperal fever. As a result the M.A.B. fever hospitals were gradually brought into line with those in such cities as Glasgow, Edinburgh, and Liverpool. Glasgow had admitted measles since 1891, Edinburgh for some years, and Liverpool measles and whooping-cough from 1908. In Edinburgh puerperal fever was treated in the same ward as enteric fever. It was actually the admission of puerperal fever, erysipelas and infectious enteritis, among other additional infectious diseases, that led to the institution of bed isolation in Liverpool. Fazakerley Hospital at that time was new and possessed isolation accommodation sufficient for its ordinary practice, but the widened clinical field necessitated an increase, and bed isolation met the need without structural alterations or additions. Cuff recognized that the admission of measles and whooping-cough would be for curative rather than preventive reasons and, surprisingly, had to allay fears that these diseases might spread from ward to ward on the analogy no doubt of the alleged aerial convection of smallpox over considerable distances. Measles and whooping-cough were admitted to the M.A.B. hospitals at first to such accommodation as could be spared after the prior claims of diphtheria and scarlet fever had been satisfied. Only in 1926 were these diseases admitted on equal terms with scarlet fever, and in 1930 the L.C.C. provided for cases of measles in fever hospitals which hitherto would have gone to Poor Law Infirmeries. The successive epidemics of measles in London during recent years and the steps taken by the L.C.C. to meet the demands for hospitalization have been described in the well-known series of reports by the Council's Medical Officer, and Brincker (1932, 1936) has published several papers upon the control of measles in London. In 1936 the following table (quoted from the Annual Report of the Medical Officer, London County Council (1936), vol. IV, Part I) gives an indication of the practice of the L.C.C. fever hospitals in a pre-war year:

| Disease | Admissions | Case mortality percentage* |
|-----------------------------|------------|----------------------------|
| Diphtheria | 5,154 | 4.06 |
| Diphtheria carriers | 404 | 0.24 |
| Scarlet fever | 9,394 | 0.36 |
| Enteric fever | 117 | 9.61 |
| Puerperal fever and pyrexia | 181 | 8.17 |
| Measles | 12,788 | 3.98 |
| Whooping-cough | 2,774 | 8.41 |
| Other diseases | 7,881 | 3.44 |
| Total | 38,693 | Total 3.31 |

* According to principal disease diagnosed on admission (not necessarily cause of death).

In 1937 the nomenclature of the acute specific infectious diseases admissible to the L.C.C. fever hospitals was revised and 28 different diseases were enumerated. With the exception of exotics such as plague and cholera, few of the diseases named in this list are unrepresented among the "other diseases" accepted for admission during a year. Earlier in this Address I remarked upon the vicious circle which had been established in the case of scarlet fever as a result of long stay and overcrowding tending to a high complication-rate and cross-infection; as late as 1910 Cuff noted that the average length of stay of scarlet fever patients in the hospitals of the M.A.B. was nine weeks. Many of

these patients were transferred to convalescent hospitals for the completion of desquamation, thus making room for more patients in the acute hospitals but tending to prolong the average length of stay.

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Section for the Study of Disease in Children

President—A. G. MAITLAND-JONES, O.B.E., M.D.

[November 28, 1941]

DISCUSSION ON THE EFFECTS OF WAR-TIME RATIONING ON CHILD HEALTH

Dr. E. M. Widdowson: Unpublished data which were obtained in a dietary survey, carried out during the years 1935-1939, on 1,000 individual middle-class children have been used to determine the pre-war intakes of foods that are now rationed. A comparison of these intakes with the actual rations to-day gives a fair idea of how the present system of rationing has or has not cut across middle-class children's dietary habits. It was found that the rations provide children up to 8 years with as much meat, bacon, sugar and jam as they were having before the war. Children over 8 years were formerly eating more of all these foods, and adolescent boys were eating two to three times as much as their present rations provide. The average pre-war intake of butter was more at all ages than a ration of 2 oz. per week, while the mean consumption of cheese was always very much less than the present weekly allowance of 3 oz.

It is suggested that, in spite of the dietary restrictions to which older children have had to submit, appetite and instinct will have led them to maintain their calories by an increased consumption of bread, potatoes and other plentiful foods. The adolescents' loss of protein, iron and vitamin B₁, brought about by the rationing of meat and bacon, can readily be made good by an increased consumption of National wheatmeal or whole-meal bread. So long as they receive their full domestic milk allowance, together with $\frac{1}{2}$ pint of school milk a day, and if they eat their full ration of cheese, there is no reason why school children's calcium intakes should fall below the corresponding pre-war figures. The calcium available for boys and girls who leave school at 14 or 15 years may, however, be inadequate. If the margarine is vitaminized so that it is equal to summer butter, children up to the age of 12 years can obtain more of the fat-soluble vitamins from their present rations than they had from the same foods before the war. If the margarine is not vitaminized they will get less of these vitamins at all ages above 1 year. Vitamin C intakes have been severely curtailed as a result of the shortage of fruit. It is not known whether this is a serious matter or not.

Dr. Dagmar Curjel Wilson: It is important to appreciate the difference between examination for signs of malnutrition carried out during routine school inspection, and the use of growing children as indicators for the detection of signs of dietary deficiency within a community. Until recently in this country the clinical signs of deficient intake, apart from the malabsorption due to organic defect, have received but little attention. Yet, as Lord Horder says, dietary deficiency may leave scars on our people even before its presence is recognized.

During school medical inspection the state of nutrition is assessed according to the standards laid down by the Board of Education, in order primarily to benefit the individual child. Such examination is largely subjective, and the results of different observers are not comparable. It is, however, useful in drawing attention to the many different

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their laboratory findings, suggested that a daily dose of about 15 mg. of ascorbic acid should be sufficient to meet the theoretical requirements of man.

Rickets.—The slighter and earlier signs of vitamin D deficiency may be recognized in the epiphyses at wrist and ankle, and are worthy of record. Very often further inquiry in home or school will show another member of the same family with more definite rachitic deformity.

Dr. Helen M. M. Mackay: In any attempt to arrive at general conclusions as to war-time feeding we must bear in mind that food conditions have differed widely at different periods as well as in different areas, and for different groups of children since the beginning of the war; and what is true of food supplies at one time and place may not be true in the next district or even in the next school. Dr. Widdowson has shown in her graphs quantities of rationed food which, on paper, are available to-day for children. I have none of the exact figures that she is able to show for pre-war children, but I am daily questioning mothers as to the feeding of their children, and this experience gives me a much less reassuring picture than that which has just been drawn. Dr. Widdowson has scarcely mentioned eggs, fish, rabbit, chicken and other forms of unrationed meat, all of which figured largely in children's pre-war diets, and are now in short supply. Some wives of Service men cannot afford to buy their share of available foodstuffs. Distribution has often been very uneven. Liquid milk has provided, I think, to the present date an outstanding example of unsatisfactory distribution in children's homes, and milk is, of course, a food of primary importance. Not only that, but some schools provide no milk, and children may spend their halfpennies on sweets and so get no school milk even when it is available. Certainly very few of the school children whom I see are getting 7 pints of milk weekly, though one hopes that the new priority arrangements may effect a considerable improvement soon. Unrationed carbohydrates, particularly white bread, must be consumed in much larger quantities by children to-day than pre-war. Perhaps too, when considering the availability of foods, one might mention breast-milk. Is there a general reduction in the number of mothers able to provide their babies with breast-milk? In the maternity hospital with which I am connected there was a marked falling off in lactation this year.

Suppose an American observer, over here to report on the effects of war-time rationing on child health, asked for information from which to compile his report what could we tell him? That we are thankful that we have had no devastating epidemics so far, though what with war-time feeding and shelter life and disorganization from bombs, we had expected them; that many people have the impression that evacuees in country districts are improving in looks, and that there are no reports published in the journals of large increases in deficiency diseases. But if he said he wanted more precise facts I wonder how many we could give him. Although individuals have made valuable contributions to our information, as witness the two speakers whom we have just heard, yet it is, I think, impossible at present to get any broad view of the effects of war-time diets on children on account of the paucity of available facts. Is, for example, the impression that evacuated children improve in health in part accounted for by the return to the cities of the sickly children? In London hospitals we see a lot of poor specimens back from the country, which makes one wonder. Can anyone here help us with facts on this point?

To guide policy and safeguard the well-being of children more official information is required. If lowered resistance to infection results from most dietetic deficiencies, then publication of the death-rates of children at different ages is badly needed. The wide use of chemotherapy should have brought about a material fall in mortality, particularly from respiratory disease. If this has not occurred, what are the counter-balancing causes? The last analysed figures from the office of the Registrar-General are, I think, for 1937, and we are left guessing as to the trends of mortality figures during the two and a quarter years of war. In the last war there was a large increase of tuberculosis in Germany, acknowledged, I think, to be mainly due to inadequate food. At the Queen's Hospital for Children the proportion of tuberculous children we see nowadays is much greater than in peace-time. From what we hear from other pediatricians and from doctors in the Public Health Service in different parts of the country, there is a wide impression that tuberculosis in children is increasing. One may try to guess how much of this is due respectively to the wholesale discharge from sanatoria of cases of open tuberculosis at the beginning of the war, or to the consumption of unboiled tubercle-infected milk by city

causes in home and school life which may adversely affect the child's well-being and lead to increased liability to infection; and thus permits of application of appropriate remedy. Accurate height and weight records, taken of individual children at regular intervals, may supply additional evidence of how far from optimum is the nutritional state of the so-called "normal" child.

For the early detection of specific dietary deficiency within any communal group, special tests are necessary. It is a matter of opinion how such examination is best carried out, whether on limited clinical material in a highly equipped laboratory, or by the application of selected tests on large numbers in the field.

For four years previous to the outbreak of war, I held under the Royal Society, an appointment for nutritional research in India. During this time I worked in close co-operation with Dr. W. R. Aykroyd and the staff of the Nutritional Research Laboratory at Coonoor. India offers particularly favourable conditions for dietary investigation, since it is possible to study populations maintaining their customary diets at many different nutritional levels, and thus to correlate deficiency disease with dietary intake. Indian experience shows that after careful laboratory investigation of clinical cases showing signs of dietary deficiency, the trained worker gains sufficient knowledge to go out to distant areas and apply selected, standardized clinical tests in the field.

That some conditions easily recognized by such tests are closely related to deficient intake of essential food factors was shown by dietary surveys worked up later with Dr. Widdowson in the Department of Medicine at Cambridge. During the past two years I have looked for the same conditions amongst groups of rural English school children. The existence amongst them of a slight degree of dietary deficiency was indicated by the occurrence of cheilosis and of low-grade rickets. It seems probable that more signs of deficiency would have been found but for the widespread provision of milk in schools. The results of this inquiry have been summarized recently in the *Lancet*, 1941 (ii), 405.

The signs of dietary deficiency applicable under war-time conditions in English schools are:

Follicular hyperkeratosis.—Keratinization in and around the pilosebaceous follicles of the skin is readily noted. In India the same standard of skin roughness to the examining finger-tip has been maintained in all dietary surveys, the degree of roughness recorded is similar to that described by Nicholls in Ceylon as "phrynoderma" or toadskin. This is probably not a simple vitamin A deficiency. Dr. Widdowson and I found on analysis of my Indian records that in addition to specific dietary deficiency, other unfavourable dietetic conditions or unhealthy surroundings are necessary before the disease becomes manifest.

Angular stomatitis.—Various observers have described ulceration of the mouth associated with deficient diets. Sebrell and Butler considered cheilosis as evidence of riboflavin deficiency. Varying results, however, have been obtained on treatment in different parts of the world, which Stannus has suggested may be explained by the absence of different essential factors in the enzyme system with which the vitamin B₂ complex is concerned.

Angular stomatitis is convenient for record, though a similar appearance may be seen at other sites where skin and mucous membrane meet. The extent of the white patches of epithelium at the corners of the mouth may vary considerably, and closer inspection often reveals an ulcerated surface in addition.

Xerosis and keratomalacia.—There is loss of lustre and wrinkling of the bulbar conjunctiva, and accumulations of epithelium, white or foam-like in appearance, which usually appear first to the outer side of the cornea, and form Bitot's spots, an easily recognized sign of vitamin A deficiency. We do not always recall that Bitot also noted that many of the children who showed these ocular conditions were living largely on carbohydrate and had a plump appearance.

Bleeding gums.—While all agree as to the desirability of an optimum intake of vitamin C, it is well in these times of dietary restriction to be able to recognize minimum requirements. I had an opportunity of examining 500 children aged from 5 to 15 years, chosen at random from large numbers in a famine relief camp in North India. Though purple discoloration was noted, no bleeding from the gums was detected. For the past three months the diet had contained no oil or fat but had consisted of whole wheat coarsely ground and about half an ounce of fresh green chillies daily, which would provide about 15 mg. of ascorbic acid. It is interesting to note that Silva and Kellie as the result of

mining how the food available will be distributed among members of the family. Often young children get all the—very few—eggs available, and much of the butter; but in many families they tend to be deprived of their cheese and their bacon and perhaps of most of their meat in favour of their elders, particularly the father. The other day I was remonstrating with a mother for giving most of her rationed food to her husband and children only to be met with the reply: "Well it's only human nature." The campaign in favour of well-balanced school meals for children is deserving of strong support, but cannot something be done on similar lines and at very cheap rates for mothers and young children? I suggest, too, that it behoves us to keep constant watch on the feeding provided in hospitals and residential institutions for children, for I suspect that the working-class mother, with all her handicaps and her lack of scientific knowledge, often feeds her children better than does the institution. All honour to her achievement.

Dr. W. W. Payne described an investigation of children to determine the minimal dose of vitamin C in the form of black-currant purée required to supplement adequately the routine diet in an E.M.S. hospital. The children, who were all convalescent cases, were saturated with ascorbic acid either synthetic (50 mg.) or as black-currant purée (1½ oz., equivalent to 40 mg.). Ascorbic acid in the form of black-currant purée was then given as a dietary supplement and the level at which saturation could be maintained was determined by the urinary response to a test dose of ascorbic acid. This level was found to vary from 15 to 6 mg. per stone according to age, corresponding to average daily doses of 1½ to 2 oz. purée per child. For full details of the investigation see Payne, W. W., and Topley, E. (1941), *Lancet* (ii), 596.

Dr. J. A. Glover said that his personal impression was that, so far and speaking generally, neither the health nor the nutrition nor indeed the spirits of the elementary school child had deteriorated. There were, perhaps, special areas where an exception might be made. The school child started this war a bigger and better nurtured child than his predecessor of 1914 who had improved on the whole during the last war. It was impossible to disentangle the effects of rationing from those of the many other abnormal factors which had influenced his nutrition in war-time.

Evacuation, often more than once, closure of schools, shorter school hours, smaller classes, shelter-life—these factors with city children, and with all children, such factors as higher wages in the home and the black-out, had for better or worse, affected the nutrition of the school child as well as rationing, though school closure was practically over before rationing began.

The majority opinion of school doctors and nurses, and of teachers was; he thought, against any deterioration in nutrition having so far occurred.

Few knew better than he did the pitfalls of statistics of the clinical assessment of nutrition, but for what they were worth, the aggregate returns of the six years' assessment of nutrition of the children seen each year at routine medical inspections (a yearly average of about one and a half million children) showed no deterioration.¹

Owing to war-time shortage of staff, figures for average measurements were hard to get for 1940.

He would have expected some decrease in average weights with the rationing of fats, jam and sugar but the evidence of the few areas for which 1940 averages were obtainable was evenly divided. The decreased incidence of infectious diseases in 1940 was indirect evidence against deterioration.

Rationing had greatly accentuated the inadequacy of the "brought lunch", the dinner which the "long distance" child in rural districts brought with him to eat in school.

The Board of Education and the Ministry of Food were doing their utmost by liberal grants in aid, special concessions to school canteens, and persuasion to induce local education authorities to provide dinners and milk in school for every school child.

It is to be hoped that one good result of the war will be the inclusion of the school dinner as an essential part of the school curriculum and the provision of school milk for every child.

¹ A table was shown at the meeting which included some official figures not yet published.

evacuees accustomed to pasteurized milk, or to lowered resistance from dietetic defects, but at present it is only guesswork, for we do not even know whether the increase is general, or, if so, its magnitude. The subjective classification of nutrition as fair, good, &c., by a variety of observers is of no use for comparison of groups. The school medical officer is required to place the children of the batch he is inspecting into certain groups, and having put a few into the best and worst classes, the rest go into the intermediate groups. But average rate of growth is one of the important indices of the adequacy of the diet, though it may of course be influenced by many other factors. A comparison of average weights of children pre-war and now would not be a simple procedure, since it would involve a comparison of both country and city groups, with due regard, for example, to possible falsification of figures from the return of children in poor health to their own homes from billets. Nevertheless, with the co-operation of statisticians, clinicians and public health officials, useful figures should be obtainable. The survey should, if possible, include adolescents, for these groups in particular, as Dr. Widdowson has demonstrated, are having their usual foodstuffs drastically curtailed, and I fear they may not get enough animal protein to allow of normal growth.

One deficiency disease, nutritional anaemia, is certainly extremely common among infants and young children. I believe there has been a large increase in this anaemia in London in the last two years. Say twelve years ago, severe anaemia was common at this age; but in the East End of London in the immediate pre-war years, though there were plenty of children with their haemoglobin in the seventies, it had become difficult to find cases of severe anaemia, as I well know, for I hunted for cases for certain therapeutic trials. Now these cases are again common. Dr. Wills and Lady Bingham from the Royal Free Hospital, and Dr. Dobbs and myself from the Queen's Hospital for Children, are trying to get objective evidence of the incidence of anaemia in various groups of supposedly healthy women and children: so far we have not had the opportunity of examining school children. Of the children between 6 months and 4 years old already examined, only about one in six reaches the level of 85% on the Haldane scale, and one in nineteen is severely anaemic, i.e. has a haemoglobin in the fifties or lower. Babies whose haemoglobin level averages about 72% have a greatly increased susceptibility to infection. Is it not a reflection on our common sense that we let this anaemia exist? Is it wise to impose a purchase tax on the substance needed for the control of a deficiency disease affecting the majority of our young children? The average haemoglobin level of these children could be raised to over 80% and the incidence of infection among them drastically lowered, by the simple means of regular iron administration. Dr. Sheldon commented the other day on the very different prognosis for an anaemic baby and for a baby of normal haemoglobin level if both get bronchitis. We can be sure that respiratory infections are going to be a serious problem among the small children congregated together in day and residential nurseries. Young mothers won't stay in the factories if their babies are ill, therefore on the score of wasted woman-power alone we would do well to look to the health of these infants, and raise their resistance by giving them iron.

Turning to other deficiency diseases, I speak from impression alone. I think there was some increase in the incidence of rickets in London last winter. I have seen no cases of scurvy during the past year, and ascorbic acid seems to be widely used in London to replace orange juice for babies. Vitamin C deficiency is probably commoner in children past infancy than in babies in London. I do not think there is any increase in cases of angular stomatitis or gingivitis in my out-patient clinics. Not long ago, however, I was asked to see a group of children suffering from gingivitis. All were refugees housed in a large building with communal feeding. The children affected were almost all of school age, though no school had been open to them for most of their time in the hostel—and no school meant no school milk. Children under 5 who got more milk escaped: hence the condition was primarily dietetic in origin. The affected children all had gingivitis and conjunctivitis, some had Vincent's angina, some had angular stomatitis. Most had scars of healed sores on their bodies and looked in poor health. The diet probably had multiple deficiencies, but riboflavin deficiency seemed, I think, the most likely to account for the symptoms observed.

The opener of the discussion has shown in a previous paper how war-time rationing has cut right across the feeding habits of men. When there are food shortages, preconceived ideas and the wife's care for the health of the wage-earner play a large part in deter-

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Prof. A. St. G. Huggett: The treatment and care of the infant really begin before birth. The development *in utero* is ultimately dependent upon the food supply of the mother. The effect of restriction is shown by animal experiments and human observations. In proportion to the severity of the deficiency, there is seen: (i) Depletion of maternal food reserves; (ii) loss of maternal weight; (iii) depletion of the foetal food reserves, and (iv) loss of foetal weight (low birth-weight). This last is only seen with severe shortage. A limited comparison (of no statistical value) of the birth-weights of the first six months of 1941 and 1938, suggests that there is no such severe shortage, even with twin pregnancies, where the greater competition might show itself in individual loss of birth-weight. Up till this week (beginning Monday, November 24, 1941) there has been a reasonable supply of milk, so that the mother has had at least 2 pints of milk available. This week there has been a cut in the ration so that, allowing for the shortage of milk and difficulties of distribution, the minimal ration for a pregnant woman of 1 pint per day will be the maximum for many pregnant women throughout the country. Allowing that in addition to the rationed foodstuffs she has weekly 3 lb. of cabbage, 7 lb. of brown bread, 2 lb. of carrots and 4 lb. of potatoes, there is evidence the protein intake and the vitamins A and B, are borderline. There is reason to suppose the daily ration of calories will not exceed 2,400, of calcium 1.3 mg., of iron 14 mg. per day, of vitamin D 70 I.U. and of ascorbic acid 90 mg. All these last five are in varying degree below the generally accepted minimal requirements. The calcium deficiency can be compensated in some degree after birth as also can the ascorbic acid; but this does not apply to iron. It is probable, therefore, that there may occur neonatal signs of malnutrition, notably hypochromic anaemia, infections and chest troubles, and possibly poor musculature.

Section of Anæsthetics

President—A. D. MARSTON, M.R.C.S., L.R.C.P., D.A.

[November 7, 1941]

The Centenary of the First Anæsthetic Use of Ether

PRESIDENT'S ADDRESS

By A. D. MARSTON, M.R.C.S., L.R.C.P., D.A.

As the centenary of the first anæsthetic use of ether occurs during the present session, I have taken this event as the subject of my Presidential Address, and will begin by a brief survey of the happenings of the Autumn of 1841 and the Spring of 1842.

Ether had been known for many years, and, according to Dudley Buxton [1], was discovered in 1540 by Valerius Cordus, who described it as "*oleum vitrioli dulce*". During the last quarter of the eighteenth century ether was employed medicinally for a number of diseases, amongst others, curiously enough, asthma and pulmonary tubercle, while at the time our story opens it had been increasingly used as an alternative to nitrous oxide gas for the production of the "*frolics*". These "*frolics*" were the strange parties in vogue at that time. They seem to have occurred only in Great Britain and America, and it is interesting to note that a French writer, M. Filvée, in his "*Lettres sur l'Angleterre*", published in 1802, described them as a vice peculiar to these countries, and an example of the increasing decadence of the Anglo-Saxon race. From these "*frolics*" the participants secured a thrill and much amusement, and in the Autumn of 1841 Crawford Williamson Long, a young physician practising in the obscure little town of Jefferson in the State of Georgia, U.S.A., gave a series of such parties at his house. At first he used nitrous oxide, but supplies of this were difficult to get, and so he used ether which he obtained from a local chemist named Goodman.

A studious brain and an observant eye were the fortunate possessions of Crawford Long, and he noticed that although bruises and minor injuries resulted from the tumbles and the reelings of the participants of the "*frolics*", neither they nor he himself suffered any immediate pain. Such a deduction led to the epic discovery of ether as an anæsthetic, and on March 30, 1842, Long administered ether to a young man named James M. Venable for the operation of removing a cyst from the occipito-cervical region. The administration of ether, which was performed before witnesses, was the simple inhalation of ether from a towel, and it was very successful. The patient was delighted at the complete absence of pain, and Crawford Long realized that he had made a great discovery. It needed courage on Long's part—more courage than is generally appreciated—to perform this operation on Venable without parental consent and against the advice of his friends. Here is an account from the writing of Mr. Jim Vandiver:

"The day that James Venable had a tumor cut out, I happened to be in Jefferson. I well remember it; can never forget that memorable day—March 30th, 1842. A group of excited men were gathered on the public square of the village not far from the little office of Dr. Long. I enquired the cause of this gathering and was told that Venable, in opposition to the wishes of his family, was letting Dr. Long put him to sleep to cut out a tumor. They vowed that should the boy fail to arouse from the effects of the ether, they would lynch Long. I did not see the operation, but I did station myself at the steps of the office in which the operation was being done, to beg the crowd to stand back and quietly await the result.

"Dr. Long was my friend and I believed in him. Not long to wait; the door opened, and we were told all was over. James Venable was safe and all right, the tumor out and no pain felt by the patient, the tumor in the possession of the miracle man". [2]

OPUS ARTIFICEM PROBAT

VALERIUS CORDUS (1515-1544) was the first to write an authoritative work of the pharmacopœia type : the complete title of this was "Pharmacorum Conficiendorum Ratio, Vulgo Vocant Dispensatorium." Cordus was also the first to prepare ether, but this remained a chemical mystery for more than a century and was known by various names, such as "Oleum dulce Paracelsis," "Oleum vitrioli dulci," "Aqua Temperata," and "Aqua Lulliana." Not until 1842 did Crawford Long first use ether as a general anæsthetic.

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Read by Mrs. Eugenia Long Harper of Atlanta, Georgia before "The Circle"—Hon. Students' Society of the Marquette University School of Medicine—on April 30, 1934.)

Crawford Long operated on Venable for a second tumour on June 6 of the same year, and this undertaking proved almost as successful. On this occasion, however, Long only induced anæsthesia with ether, and then proceeded with the operation. This resulted in the anæsthesia becoming exceedingly light towards the end of the operation, which was probably concluded in a state of analgesia, because Venable noted that something was being done to him although it was not painful.

Long deduced from this that continuous inhalation of ether was probably necessary. This technique he observed in his next operation performed on July 3, 1842, for the amputation of a toe.

Subsequent operations were undertaken by Long with the help of ether on September 9, 1843, for the removal of a tumour from the head of Mary Vincent, of Jackson, and on January 8, 1845, for the amputation of a finger of a negro boy, the property of Ralph Bailey, Esqr., of Jackson, Georgia.

In 1844, nearly three years after Long's first anæsthetic, Horace Wells, a dentist of Hartford, Connecticut, started his pioneer work with nitrous oxide. Wells experimented with ether, but observing the choking sensation resulting from ether inhalation, decided to continue his anæsthetic work with nitrous oxide alone. As is well known, Horace Wells became insane, and according to Dudley Buxton "died in prison, by his own hand, having inhaled ether to secure euthanasia" [3]. Although he made no claim himself for the discovery of ether, his relatives and friends did so after his death.

To, William T. G. Morton, a dentist of Boston, a pupil and sometime partner of Wells, must be given the distinction of conducting the first successful public demonstration of ether anæsthesia, which took place at the Massachusetts General Hospital on October 16, 1846. Although responsible for the practical application of ether as an anæsthetic, it seems that the agent was suggested to him by one of his teachers, Dr. Charles T. Jackson, of Boston.

Morton deserved great praise for his painstaking experiments with ether. He used to anesthetize his pet dog, and he also placed himself under the influence of ether. In September 1846 he succeeded in putting himself to sleep for eight minutes whilst sitting in his dental chair. On recovering consciousness, he was much elated, and decided to extract a tooth under ether as soon as possible from a human patient. This he was quickly enabled to do, for a few minutes later his surgery bell rang, and Mr. Eben Frost was ushered in suffering from toothache and a swollen face. Mr. Frost requested Morton to mesmerize him for the extraction, but Morton said "No! I have something much better", and proceeded to give ether. It is recorded that "to the joy of the operator and the astonishment of the patient, the attempt was perfectly successful" [4].

After the Massachusetts demonstration, the use of ether quickly spread over the United States of America, but was hindered by the misguided attempt of Morton to patent ether as a compound known as "letheon". Morton also requested in 1849 that the Senate of the United States should give him a gratuity for his claim to be the discoverer of anæsthesia. This claim was vigorously contested by Jackson and the relatives of the late Horace Wells. This application, which was never granted, had at least one good result in bringing to light the undoubted claim of Crawford Long, who had independently discovered ether anæsthesia and, desiring neither notoriety nor reward, has received them from a grateful posterity.

About nine weeks after the Massachusetts demonstration, the elder Bigelow communicated with his old friend, Dr. Boott, in London, giving him details of his son's paper on the first use of ether. Thereupon, on Saturday, December 19, 1846, Mr. Robinson, a dental surgeon, administered ether and extracted teeth from a patient at the house of Dr. Boott in Gower Street, close to University College Hospital, in which institution on the following Monday, December 21, Mr. Liston amputated a leg, the ether being administered by Mr. Squire. An illustration of Squire's original ether inhaler may be seen in *Proc. Roy. Soc. Med.* (1930), 23, 1130 (Sect. Anæsth., 48).

At this time, Dr. John Snow lived and practised medicine at 54, Frith Street, Soho, and was destined to become the first and possibly the most famous of British anæsthetists. It is just one hundred years since John Snow read his first scientific paper entitled "Asphyxia and on the Resuscitation of New-Born Children" before the Westminster Medical Society on October 16, 1841. Reading in the *Lancet* Bigelow's account

of the Massachusetts demonstration, Snow at once interested himself in the matter, and during the next few years accomplished two great feats of pioneer work. First, he noted the imperfect nature of the ether administration, and devised an inhaler (fig. 1). Secondly, he carried out a series of experiments which placed the pharmacology and physiological effects of ether on a scientific basis. These experiments were carried out on mice, birds, guinea-pigs and frogs, and were remarkable not only for their exactness but for the degree of clinical information they yielded.

As soon as Snow had perfected his ether inhaler, he obtained permission to use it in the dental out-patient department of St. George's Hospital, and this proved so successful that he was soon asked to attend on the in-patient operating days. A little later he was appointed to University College Hospital where he gave ether for Mr. Liston, one of the busiest and most brilliant surgeons of his day. John Snow soon built up a considerable private practice, and in the last ten years of his life administered about 450 anæsthetics per annum. He had the privilege of anæsthetizing Queen Victoria in the last two of her several confinements, and Her Majesty was graciously pleased to express

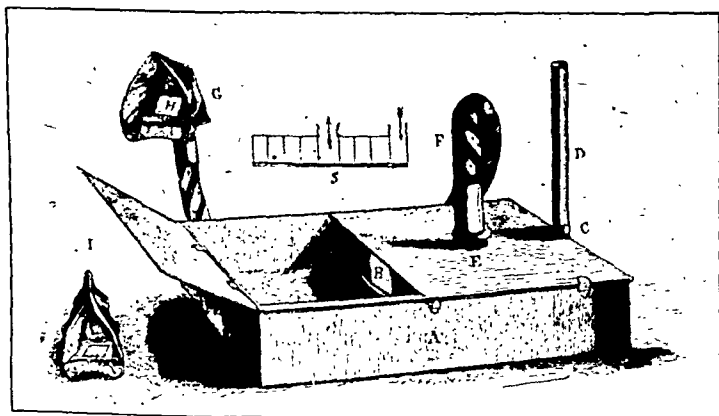


FIG. 1.—Snow's Ether Inhaler, 1847.

A. Box of japanned tin "the size and form of a thick octavo volume". It served as a water bath. B. Ether chamber. C. Opening for filling and emptying. D. Tube for the ingress of air. The weight of air in the tube was intended to prevent evaporation into the room. E. To the opening E was screwed. F. A flexible tube about 3 ft. long. G. Facepiece. H. Inspiratory valve. I. Facepiece compressed to fit a smaller face. S. Section of ether chamber. [From the Nuffield Department of Anæsthetics, Radcliffe Infirmary, Oxford.]

her approval not only of the chloroform but also of the services of Dr. John Snow.

Most of Snow's work was with chloroform, but he always evinced great interest in ether. In his book he remarks:

"I prefer the flavour of ether vapour to that of chloroform, and the sensations I experience from the inhalation of ether are more pleasurable than those from chloroform" [5].

In Richardson's "Life of John Snow" we read:

"Once, he observed that in his opinion sulphuric ether was a safer narcotic than chloroform. Why, then, said a listener, do you not use ether? I use chloroform, he resumed, for the same reason that you use phosphorus matches instead of the tinderbox. An occasional risk never stands in the way of ready applicability" [6].

John Snow was a Yorkshire man, the eldest son of a farmer, and was born at York on June 15, 1813. He died on June 16, 1858, in his 45th year. He was attacked by his

fatal illness as he was writing the concluding sentence of his classic work on *anæsthesia*, which to this day remains famous and widely read (fig. 2).

Snow was aged 33 at the time he became an *anæsthetic* specialist, and he practised for only twelve years. Few men have accomplished so much original work in so short a time, and this is even more remarkable when one remembers that he was handicapped by poor health, and also that a good deal of his time was spent in research in subjects other than *anæsthesia*, including *cholera* and public sanitation.

Among those present at the first administration of ether at University College Hospital was a senior student named Joseph Clover (fig. 3), who was much interested in the proceedings. From that day until his death in 1882 he took an active part in the advance of *anæsthesia*. Joseph Clover was a Norfolk man and was born at Aylsham in 1825. His character has been described as gentle and amiable, and it has been written of him that "he was beloved by his patients and much respected by his medical colleagues". Clover was Resident Medical Officer of University College Hospital for five years, and during that period administered many *anæsthetics*. At this time Dr. Snow was probably the only *anæsthetic* specialist, and Clover was the first to follow his example. This choice of career was imposed upon Clover by his delicate constitution. In 1853 he became a Fellow of the Royal College of Surgeons, and showed great promise; but the hard life of a practising surgeon was too much for him, and so he decided to become an *anæsthetist*.

At this time the supremacy of chloroform, which, when introduced in 1847, became so popular as to supersede almost entirely the use of ether, began to wane on account of its dangers, and *anæsthetists* experimented with new methods to utilize the safer properties of ether.

The chief objections to ether were its slow and turbulent induction, and the difficulty of securing any steady level of maintenance *anæsthesia*. Apparatus was then primitive, and consisted in the use of a folded towel, a Rendle's cone, or a closed apparatus such as that of Squire, which entailed in the main re-breathing into a flask of two compartments, containing pieces of sponge and ether. All were unsatisfactory, and Clover's ether inhaler, which was produced after many experiments and much hard work, speedily displaced them. Various modifications were made from time to time, and this inhaler has been used by some *anæsthetists* right up to the present.

In 1864 the Royal Medical and Chirurgical Society of London, now the Royal Society of Medicine, appointed a committee to report upon the safety and efficiency of chloroform and ether. The committee, of which Clover was an advisory member, reported that ether was safer than chloroform, but that it was not an ideal method of *anæsthesia* because of its lengthy induction and the variable and uncertain character of its maintenance.

Clover described his completed apparatus in 1877. With it patients were induced comfortably with nitrous oxide gas, this being followed by ether and finally by ether vapour and air. By means of this apparatus, induction took from one and a half to two minutes, and afterwards it was possible to maintain a smooth *anæsthesia* for as long as might be required.

At about the same time as the introduction of Clover's inhaler, Dr. Ormsby, a Dublin physician, described his ether apparatus. Ormsby's inhaler was widely used and was preferred by some *anæsthetists* to that of Clover. Discussing the respective merits of the two inhalers, Sir Frederic Hewitt writes:

"For inducing *anæsthesia* by means of ether there is no apparatus which can be compared to that invented by Clover, for the *anæsthetic* vapour may be admitted so gradually that the initial discomforts are reduced to a minimum. But for maintaining ether *anæsthesia* Ormsby's inhaler is equal, and in many cases superior to Clover's. I have on several occasions changed from a Clover's to an Ormsby's inhaler with marked improvement in the symptoms of the patient. For example, I have often known cyanosis to quickly vanish and the breathing to become less hampered by effecting this change of inhalers during deep ether *anæsthesia*" [7].

This question of cyanosis with Clover's inhaler has always been its chief defect. Clover himself recognized this and invariably desired ether to be given with enough air to eliminate asphyxial symptoms. In later times this difficulty was finally solved by Lieutenant-Colonel Ashley Daly, who, as Nosworthy says: "employed the simple expedient of admitting sufficient oxygen through a tap in the angle-piece" [8].

I think it is correct to state that Clover's apparatus has been employed for ether



John Snow

FIG. 2.—Autotype from a presentation portrait, 1856, and autograph facsimile. [From the Nuffield Department of Anaesthetics, Radcliffe Infirmary, Oxford.]



FIG. 3.—Joseph Thomas Clover (1825-1882). [From a photographic portrait in the Nuffield Department of Anaesthetics, Radcliffe Infirmary, Oxford.]

anæsthesia from its introduction in 1876 right up to the present time. But most anæsthetists abandoned its use in the last war when open ether or semi-open inhalation became popular. According to Rawdon-Smith: "The 'open' method was invented by L. H. Prince, of Berlin, U.S.A., and independently by Ferguson, of Boston. It was introduced into England in 1906 after the Toronto Meeting of the B.M.A." [9].

Gwathmey, in America, Shipway, Pembrey and Geoffrey Marshall, in England, did valuable work in directing attention to the advantages accruing from the suitable heating of ether vapour. Shipway's portable ether and chloroform apparatus had a considerable vogue and it was a useful and successful apparatus. "Mennell's bottles" were also largely used at this time, and were both simple and efficient.

About 1921, Pinson introduced his ether bomb, which was indeed an effective means of heating ether vapour, and it was the use of this apparatus which first led observers to query the advantage of heating ether vapour past a certain limit. Under the full anæsthesia obtained from Pinson's bomb, patients were sometimes observed to become flushed and actually to sweat. When much sweating occurred it was thought that more heat was perhaps thus lost than in the open administration of cold ether. On the Continent of Europe elaborate apparatus has been constructed such as the Tiegel-Henle-Sauerbruch and the Tiegel-Lauer, and these seem to have no advantage, for if ether is overheated it may well condense in the respiratory passages, with the production of harmful results.

As I have said, the epoch of ether warming had a definite vogue from 1915 until 1923, after which period most anæsthetists thought the matter had been overstressed and that the delivery of ether vapour at body or room temperature was the best.

In 1924, Langton Hewer wrote a paper entitled: "The Endo-tracheal Administration of Nitrous Oxide-Ethanesal as the Routine Anæsthetic of Choice for Major Surgery." In it he discussed the pros and cons of ether warming, and concluded by remarking: "As the result of these investigations the writer has now abandoned all extra heating." [10]. This paper was a valuable one because it also directed attention to the undoubted value of endotracheal administration of ether not only for the surgery of the face, lips, jaws and thorax, but also for major abdominal operations.

The subsequent development of ether administration by inhalation has been chiefly furthered by the work of Gwathmey, Guedel, Wesley Bourne, and Ralph Waters in the United States of America and in Canada, and by Boyle, Shipway, Rowbotham and Magill in this country.

Boyle's apparatus enabled ether to be used in a minimal capacity, that is, as an adjuvant to gas and oxygen. Boyle first introduced his apparatus in the Great War, and not excepting Clover's apparatus, it has probably had the greatest popularity of any anæsthetic apparatus. Boyle has been honoured for his work in anæsthetics both in this country and in America. He was an Honorary Member of our Section, and I am sure we are all very proud of the way in which he handed on the torch of progress in our special branch of medicine during the last generation.

In 1925, Webber introduced his modification of Boyle's bottles, which places the bottles in parallel instead of in series. In practice this arrangement works well and prevents any contamination of ether by previous transit of gas through the chloroform bottle.

RECTAL ETHER

Rectal ether (at first pure) was used by Pirogoff and Roux in 1847, but although this was found to be efficient as a mode of inducing anæsthesia, it was abandoned on account of the discomfort experienced by the patient and the damage caused to the local tissues.

Pirogoff later perfected an apparatus which warmed the ether and allowed only ether vapour to enter the rectum. This was more successful but was sometimes followed by unfortunate after-results such as meteorism, diarrhoea and melæna. No eminently satisfactory apparatus was produced in England until that of Dudley Buxton in about 1885. It was made for him by Messrs. Mayer and Meltzer, and in a personal communication Mr. Ernest Mayer says:

"The writer assisted Dr. Buxton at various operations at which this apparatus was used; once at King's College Hospital, Sir Joseph Lister being the operator, and on a brain case, Sir Victor Horsley being the surgeon."

This apparatus was a success, but Buxton in his description of "after-effects" quite honestly says:

"Colicky pains in the intestines, urgent tenesmus, diarrhœa, sometimes dysenteric in character, painful distention of the intestinal tract with more or less severe collapse are the complications which have been recorded."

and he concludes this rather dismal summary with these words: "Deaths have occurred" [11].

Dudley Buxton, M.D., was born in 1855 and died in 1931. He did an enormous amount of work in investigating the use and actions of ether, and encouraged its routine use instead of chloroform. In this he encountered opposition. He disagreed with the findings of the first Hyderabad Commission in 1889, and also of the second Commission held a little later on. A further service to ether administration was his rooted objection to any cyanosis. In this crusade also he met with opposition.

Buxton was a founder of the Society of Anæsthetists (in 1893) which later became the Anæsthetic Section of the Royal Society of Medicine. He was first appointed anæsthetist to Soho Square Hospital for Women, and afterwards to University College Hospital where he instituted systematic lectures on anæsthesia and introduced the custom of allowing students to administer anæsthetics under the supervision of a teacher.

The first Hyderabad Commission Report was reviewed by Dr. Buxton in the *Lancet*, and he pointed out that it was entirely inadequate and that the experiments were too few, were crudely conceived and were unscientifically carried out.

Conditions in the specialty of anæsthesia were indeed chaotic at the commencement of Buxton's career. We read that the status of the anæsthetist was almost negligible. For instance, if a surgeon needed a short anæsthetic to be given when examining a patient in his consulting room, the butler or footman would be called in to administer some nitrous oxide or a whiff of chloroform. In some hospitals the porters gave chloroform under the direction of the surgeon. Buxton is recorded as describing the occasion on which he was invited by a distinguished surgeon to meet the then President of the College of Physicians at a private house for the purpose of giving an anæsthetic for an examination. The surgeon conducted him upstairs, and, on opening the bedroom door, Buxton was amazed to find the physician and the patient rolling about the floor, locked in one another's arms. Not expecting an anæsthetist, the physician had attempted to start the anæsthetic, with—as Buxton observed—but indifferent success. Surely, argued Buxton, if so untoward an occurrence could happen under the auspices of such an august administrator, there was every reason for the existence of specialists in anæsthesia.

In 1913, Gwathmey introduced his ether-in-olive-oil technique, which was satisfactory and had a considerable popularity, specially in the Great War.

The chief use of rectal ether was to establish a method whereby operations could be successfully performed on the face, mouth, head, larynx and thorax. We seldom, if ever, hear of rectal ether to-day, partly because other agents such as avertin and paraldehyde given by the technique of Rowbotham have proved superior, but more particularly because it has been superseded by the development of the endotracheal method.

ENDOTRACHEAL ETHER INSUFFLATION

Kuhn (1900) and Elsberg of New York (1909) were the pioneers of this new method which was further developed by the work of Meltzer and Auer, and was first used in this country by Kelly of Liverpool in 1912. His apparatus is described in Minnitt's revised edition of Rose and Fairlie's "Handbook of Anæsthesia". It was modified and improved by Shipway in 1916.

Two advantages have been claimed for ether insufflation under positive pressure:

(1) The return flow of air issuing between the rigid gum-elastic catheter and the trachea was said to prevent the descent of any blood or oral secretion into the pulmonary alveoli, and experiments, which Kelly and other workers have performed on animals, have supported this theory. Clinical experience has, however, cast doubt upon this assertion, and Langton Hewer (*Brit. M. J.*, 1940 (i), 318) reported cases of tonsillectomy in which blood had actually been seen to gravitate along the catheter, and at the end of the operation was observed by bronchoscopy to have entered the lower respiratory system.

(2) The positive pressure of insufflation could be used to produce apnœa, so that the immobility of the chest might facilitate operative procedures in the upper abdomen

and thorax. Clinically, this practice was sometimes found to result in hypostatic congestion of the lungs.

I mention these claims to special advantage accruing from insufflation because protagonists of this technique have recently advocated it in preference to the type of endotracheal inhalation now in general use.

ENDOTRACHEAL INHALATION

This is low-pressured inhalation through a wide-bore soft rubber tube which fills (or nearly so) the trachea. This method was introduced by Rowbotham and Magill as the result of much experimental work performed in the plastic clinic at Sidcup during the Great War (1914-18).

In the opinion of most anaesthetists this method is more physiological than positive insufflation and produces fewer undesirable after-results. If used with ether, it enables the drug to be given in minimal quantities mixed with nitrous oxide and oxygen.

I must not conclude without a mention of Sir Frederic Hewitt and the great work he did with ether.

Sir Frederic Hewitt was born in London in 1857 and died in 1916 aged 59. He was educated at the Merchant Taylors School and Christ's College, Cambridge. He had a distinguished academic career at St. George's Hospital, winning several prizes, and graduated M.D. at Cambridge in 1884. He was made an M.V.O. in 1902 for his service as anaesthetist to King Edward VII, and was knighted in 1911. He did much to develop the use of ether, and his researches advanced our knowledge of its pharmacology.

Hewitt was originally a physician, and but for ill-health (he suffered from an incurable defect in his vision) would probably have practised as such. He did a great service to our specialty in elevating its status. This he accomplished by successfully persuading the examining bodies to include the subject of anaesthesia in the medical curriculum. He held strongly to the opinion that only qualified medical men should administer anaesthetics, and influenced the Home Office authorities to such a degree that a Bill for an Act of Parliament to this effect was actually introduced in the House of Lords in 1912. The congestion of legislation before the Great War, and the delay caused by a certain amount of opposition, prevented the matter from proceeding any further, however.

His textbook on anaesthesia, published in 1897, after ten years of careful and patient preparation, had reached its fifth edition at the time of his death in 1916. It has been described by Blomfield as "probably the best systematic exposition of the theory and practice of anaesthesia in any language" (12).

Hewitt was a man of great determination and courage. It is recorded that on one occasion, when confronted by a complete spasm of the glottis in the dental chair, he produced his penknife and coolly performed the operation of tracheotomy. This relieved the symptoms and the patient's life was saved.

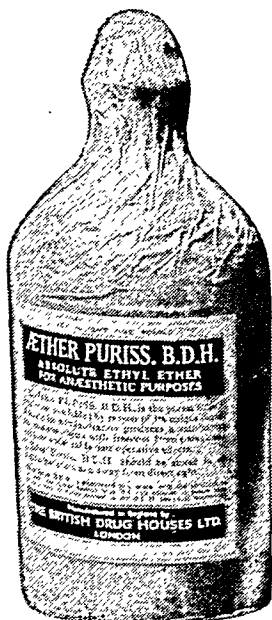
It is interesting to note that as the centenary of ether approaches, new and valuable work has been carried out by Professor Macintosh and his co-workers in the Nuffield Research School at Oxford.

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(1) *paper read before the Urological Section of the A.M.A., June 6th, 1941.*

(2) *Lancet, No. 5, Vol. 1, 1941, p. 144.*

(3) *B.M.J., 2 : 8 : 1940.*

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Section of Urology

President—G. E. NELIGAN, M.C., F.R.C.S.

[November 27, 1941]

DISCUSSION ON RUPTURE OF THE URETHRA AND ITS TREATMENT

Mr. Clifford Morson: Rupture of the urethra is the most serious of all injuries to the genito-urinary tract, not on account of the immediate mortality rate, which, *per se*, is negligible, but because of subsequent ill-health. I know of no case where the urethra has been completely torn across, in which the patient has been restored to perfect health. There may be a long interval between the date of injury and the onset of some lesion in the urinary tract directly attributable to the damage done to the urethra, but come it will. However, there are many ways of minimizing these dangers.

The causes of ruptured urethra are well known, but in view of the times we live in, one type of laceration of the urethral mucous membrane is of particular importance. Where there has been a comminuted fracture of the arch of the pubis due to a war wound, there is considerable danger that, subsequently, a spicule may be detached and penetrate the urethra. This complication may occur some years after the fracture has united. Thus in a case admitted to St. Peter's Hospital under my care, micturition had been normal ever since the pelvis had been fractured by a piece of shell during the 1914-18 war. Suddenly in 1925 this soldier was seized with pain in the perineum and retention of urine. The passage of a catheter was blocked by what was thought to be a stone, but X-rays demonstrated the presence of a small spicule of bone lying across and within the lumen of the membranous urethra. At operation it was found that this piece of bone had worked its way from the soft tissues adjacent to the arch of the pubis. Such a complication can obviously be prevented, when treating a gunshot wound of the pelvis, by removing all isolated fragments of bone. The site of the rupture can usually be determined by the nature of the injury. Blows of all kinds upon the perineum such as falls and kicks damage the posterior part of the bulbous urethra. This part is also involved when the penis itself is violently pulled on.

Crush injuries, causing fracture of the pelvis, affect the posterior urethra between the internal meatus and the triangular ligament. I have never met with a case of rupture of the urethra in the region of the compressor muscle.

The diagnosis of a rupture due to an accident or a gunshot wound is rarely difficult. If the urethra is completely torn across there will be retention of urine and escape of blood from the external meatus. Immediately following the injury there is an urge to micturate. An inability to carry out this act is a sure sign that the rupture of the urethra is complete. Many of the older textbooks teach that the patient must be told to hold his water. There is no need for this instruction. Micturition if at all possible will have taken place before the arrival of the doctor. If the rupture is incomplete a few drops of blood may appear at the external meatus followed by micturition in which the urine is blood-stained.

I am in entire agreement with the late Frank Kidd that on no account should a catheter be passed for diagnostic purposes. Damage to the mucous membrane is

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I am in entire agreement with the late Frank Kidd that on no account should a catheter be passed for diagnostic purposes. Damage to the mucous membrane is

thereby increased, the very thing which must be avoided. The more the tissues are torn the greater will be the formation of scar tissue. Bruising of the perineum indicates that there has been some degree of injury to the urethra, but the deciding factor in determining the extent of the injury is the ability of the patient to micturate. Frank Kidd stressed the value of the urethroscope. If there is any doubt at all as to whether the urethra is completely ruptured this is the only instrument which should be used. In every case of suspected injury to the urethra, the pelvis must be X-rayed, to exclude fracture. It is fortunate that this serious injury is unusual. Frank Kidd stated that out of a total of 14,000 in-patients admitted annually to the wards of the London Hospital there were only 4 cases of ruptured urethra. In ten years there were 41 (1909-1919). At the Whipps Cross Hospital from 1933-1938 there were 8 cases admitted out of an annual average admission of 17,000 patients. Obviously, in London at any rate, this accident is a rare one.

Treatment varies according to whether the rupture is complete or incomplete. If the latter it is only necessary to confine the patient to bed for a few days provided the pelvis is not fractured. Hæmaturia never persists for longer than a day or so. Instrumentation is entirely contra-indicated. Should a large hæmatoma form in the perineum it may be necessary to make a small incision over the swelling and remove the blood-clot. Most patients can be discharged within a week of the accident. Subsequently, urethroscopy is needed every six months to study the formation of scar tissue. At the end of a year it will be possible to determine by this method of investigation the degree of stricture formation, and how often dilatation will be required.

A study of the literature dealing with complete laceration of the urethra reveals many different opinions as to what should be done to the urethra itself, but every experienced urologist is agreed that as soon after the accident as possible the urine must be diverted. Delay is dangerous. A self-retaining angular tube of any of the well-known patterns must be inserted suprapubically into the bladder by the trocar and cannula technique.

The patient is severely shocked and, therefore, the tube should be inserted with a minimum of dissection. Painful sensations in the region of the bladder are never entirely abolished by anaesthesia infiltration; the anaesthesia of choice is either a spinal anaesthetic, or gas and oxygen, or pentothal.

No plastic operation should be performed on the urethra until primary and secondary shock have passed off, which may take from twenty-four to forty-eight hours. Therefore, about the third day, the surgeon can proceed, by whatever technique he favours, to join together the torn ends of the urethra. To delay longer will not only add to the difficulties of the operation but more scar tissue will have had time to form.

If the rupture is in the bulbous portion, the union of the torn ends is not particularly difficult.

A perineal dissection with the patient in the lithotomy position gives an excellent exposure. Union is accomplished with the finest plain catgut. The chromicized variety should not be used on account of its slow absorption. On no account should a catheter be tied in. Its presence causes irritation of the bruised mucous membrane, with a consequent increase in scar tissue at the site of the rupture. Suture material which is slow in absorption also produces the same result. It is my practice to leave a rubber wick as a drain, for forty-eight hours, in the peri-urethral tissues.

The suprapubic tube should be removed at the end of about a fortnight, or when the perineal wound has healed; and at the same time under gas-oxygen anaesthesia, a metal bougie, 10/12 Charrière size, is passed through the urethra into the bladder. This instrument must be used with the greatest gentleness.

The closure of the suprapubic fistula is always slow and does not occur until healing within the urethra is firmly established—a period of one or two months. During this time metal bougies should be passed once weekly. Unfortunately treatment does not end with the closure of the suprapubic fistula. The patient must be told that he will have a stricture for the rest of his life, and intermittent dilatation is necessary if serious complications are to be avoided at a later date. For the first year after this injury the dilatation has to be performed every six weeks, afterwards the intervals may be three to six months. Woe betide the patient if he ignores this advice. The gradual narrowing of the lumen of the urethra will produce all those changes in the bladder and upper urinary tract due to urethral obstruction which are so well known to all experienced urologists.

Laceration of the urethra at the junction of the prostatic and membranous portions

is an extremely serious injury, and attended by sequelæ which lead to permanent disabilities of the most distressing kind. Often the bladder is completely dislocated, so that there is a space between the internal meatus and the triangular ligament, the intervening portion of urethra having been crushed. In such cases the pelvis is always badly fractured. The initial treatment is the same as that of rupture of any other part of the urethra, namely immediate suprapubic cystotomy. Also, when the patient has recovered from the shock due to his injuries, an attempt must be made to repair the damage done to the urethra.

It must be admitted that ultimate results, no matter what technique is used, have been uniformly bad. In many cases the scarring is so extensive that micturition is impossible and the patient is condemned to a permanent cystotomy. In other cases the urine can be passed *per urethram* only if the stricture is dilated at frequent intervals, and this treatment supplemented by internal urethrotomy when the bougies fail to stretch the scar tissue. Often bladder sepsis is so severe that cystotomy becomes imperative. A recent experience with enucleation of an adenomatous prostate, so badly performed by one of my house surgeons that the bladder was torn completely away from its attachments to the pubis and its arches, suggests a technique which may produce better results than any of those already published. It is based on the modified Harris technique for prostatectomy and is briefly as follows:

With the patient in the Trendelenburg position the interior of the bladder is exposed in the usual way and flood-lit. The posterior lip of the torn internal meatus is picked up with a boomerang needle and stitched to the lacerated proximal end of the membranous urethra. The lateral margin of the mucous membrane of the internal meatus is then stitched to the superior surface of the triangular ligament on either side of the urethral opening. The insertion of a suprapubic angular tube completes the operation. With good illumination and skilful use of the boomerang needle this technique is not unduly difficult, but it presupposes the absence of the prostate. The whole of the prostatic urethra with the surrounding glandular tissue will have to be dissected away before it is possible to unite the internal meatus to the triangular ligament. The removal of the prostate may be criticized on the grounds that it will lead to sterility, but after the crushing of the urethra between the internal meatus and the triangular ligament, the patient is always sterile. Unfortunately the only case on which this technique was tried died from pneumonia a few weeks after the operation. The end-result, therefore, cannot be estimated.

The prognosis in rupture of the urethra is determined not so much by the character of the operation, as by the amount of scar tissue formed in and around the urethra.

Many of the factors controlling fibroblastic growth are well known, but others are surrounded by mystery.

In the case of the urethra, first, the position of the rupture plays an important part. Less scar tissue forms in the region of the anterior urethra than in the posterior position.

Second: the more extensive the laceration of mucous membrane, the worse is the stricture. At least we can avoid increasing the tear of this tissue by not jabbing at it with a catheter.

Third: the longer the delay in performing the plastic operation the more difficult it will be owing to development of granulation tissue. The latter consists chiefly of fibroblasts, the forerunners of scar tissue.

Fourth: the escape of urine into the tissues around the rupture will produce dense scarring. Here again we should be able to avoid this by performing cystotomy immediately after the accident. Thus it will be noted that though the surgeon cannot prevent complications at least it is within his power to control them.

Mr. Charles Wells: This subject is clearly defined by conventional classification into rupture of the external type in which the bulb is torn by direct violence, and the internal type in which the membrano-prostatic urethra is ruptured by indirect violence. To these may be added a small group of miscellaneous cases due to exceptional injuries.

In each group we have to consider problems of diagnosis, immediate treatment (both general and local), the prevention, occurrence and treatment of complications such as extravasation, the after-treatment and prognosis. We must then turn our attention to the late cases which are brought to our notice months or possibly years after the accident, having had either too much or too little, and almost certainly wrong, treatment in the

meantime. In this formidable programme, fortunately, the boundaries of the territory are clearly defined and the subdivisions sharply cut.

In external cases where the extent of the injury is in doubt, in which there is retention due to spasm and a little perineal bruising with some bleeding from the urethra, I attempt a rubber catheter and if it can be passed I leave it in position for a few days. Instrumentation should be carried out only with real aseptic precautions, preferably in the theatre where operation can be undertaken at once if necessary. How else is one to examine a doubtful case? The patient must not be allowed to micturate for fear of causing extravasation. If the passage of the catheter is at all difficult or impossible the bladder is opened with local anaesthesia. This is important, as under general anaesthesia the patient may void and extravasate. He is next anesthetized and placed in a high lithotomy position. Instruments are passed in both directions and the urethra dissected out and sutured as a ribbon. A small rubber catheter (No. 4) is then drawn through as a guide to further instrumentation. The bladder is kept empty by the suprapubic route with an Ainsworth Davies metal tube attached to a suction pump. This keeps it empty and prevents urine from trickling down around the catheter into the perineal wound where it is the probable source of such infection, necrosis and, ultimately, the stricture commonly attributed to the catheter itself (Silverstone).

Similar suprapubic drainage with suction is part of the treatment in practically every case whether early or late.

One recent case of external rupture was reported to me by phone from a distance. Unable to go at once I advised a suprapubic cystotomy only, under local analgesia, and for various reasons a week elapsed before the urethra was explored. I then found a very large haematoma with wide separation of the ruptured ends. It is of some interest to report that this man made as good a recovery as any other, with as satisfactory an end-result. The lesson of this is clear—namely that, in the absence of extravasation, drainage will help the patient over and that thereafter there is no objection to waiting a few days for expert help in the perineal part of the operation. Deliberate postponement of repair has been advocated as a routine by Lepoutre. The danger of this course lies in the possible presence of undetected extravasation.

To the contrary, the undoing of a clumsy repair may take months of hard work. A second case illustrates this point. "M"—a soldier—sustained an external rupture early in 1939. I saw him for the first time at the beginning of this year. He had had at least two deliberate operations. There were stones in his right kidney with infection; cystitis with back pressure; and half a dozen fistulae in his perineum, which were sodden and thickened. We drained him above the pubis and opened up all his fistulous tracks. After some weeks the stricture was excised and repaired and his urethra now functions well. In the meantime, however, stones had formed in his left kidney, and one became impacted in his ureter from which we removed it as an emergency. Weeks later he developed a peri-ureteric abscess which discharged spontaneously; and so on. The stones on the right side have still to be dealt with. All of this could have been avoided at the beginning.

Intrapelvic rupture through the membrano-prostatic zone may be missed and one should not be deceived by the finding of a little blood-stained urine upon catheterization. Once the diagnosis is made, operation with a view to reposition of the soft parts follows. By one means or another a catheter is threaded through from glans to bladder. My own practice is to stitch this firmly, with silkworm-gut, to a bladder tube. The prostate is then pushed down on to the triangular ligament and traction is made upon the catheter. It is undoubtedly desirable at this stage to reduce and fix the pelvic displacement which, having caused the rupture, is inevitably tending to hold the soft tissues apart. I believe that, as always, a well-planned and thoroughly completed operation is the surest road to a successful issue. In the case in point, every effort should be made to combine orthopaedic with urological repair from the beginning. This is my view and I shall endeavour to put it into operation in future, even to the limit of immediate fixation in plaster which hitherto I have not done.

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wore a urinal day and night. The first essential was suprapubic drainage with slow decompression of the dilated upper urinary apparatus. Next we had to determine the extent of the damage. The bony distortion was apparent radiologically but urethrograms were disappointing. With double instrumentation under anaesthesia combined with rectal examination it was apparent that the prostate had risen high in the pelvis and that it was resting upon the upper surface of a large block of scar tissue uniting the separated pelvic bones. On the lower side of this huge scar was the under-developed bulbous and penile urethra.

The operation suggested by Watson in which the urethra is split so as to afford greater length was considered, but it did not promise sufficient urethra to meet the case from the atrophied bulbous portion, nor was there much prospect of finding anything at the prostatic end to which to suture it.

The perineum was duly opened up and a channel cut through the block of scar tissue so as to allow the shortest course between the separated ends, which were identified by passing bougies. The bulbous urethra was then dissected up freely but proved to be at least an inch too short. It was therefore split in its length so as to afford a flap, hinged at the blind end, sufficient to reach the bladder with ease.

The next difficulty was to find anything at the bladder end to which this urethral flap could be sutured. We eventually decided simply to anchor it in position and passed a catgut stitch through its extremity and drew this up through the prostate into the bladder. Finally this stitch was attached to the anterior abdominal wall by an intermediate length of elastic, thus affording continuous gentle traction. A fine catheter was put in as an aid to later instrumentation and the bladder drained by suction.

The subsequent history of this most difficult case was interesting. He micturated normally and attended irregularly for the passage of a small soft instrument which presented no difficulty. He worked at his job for rather more than two years after the operation and then died from an intercurrent acute infection of his irretrievably damaged urinary tract.

"J. J.", aged 9, was crushed by a heavy stone falling upon him. An early suprapubic drainage had saved his upper tract but left him with a grossly deficient urethra and a high prostate lying very close to the rectum. An operation, identical with the one just described, successfully reconstituted his urethra but the first subsequent instrumentation gave him a urethro-rectal fistula. An attempt to close this locally failed and we had resort to mobilization of the rectum as admirably described by H. H. Young. The muco-cutaneous junction at the anus is incised and the anal canal mobilized from within the anal sphincter. The bowel is then drawn down until the damaged portion is all outside, when the external part is cut off and the remainder re-sutured to the skin. This operation was a complete success but we still had difficulty in instrumentation because of a curious flap formation (the nature of which remained obscure) in the prostatic urethra. This was destroyed from the bladder aspect and since then all has been well.

In "L. M. J.", another small boy run over by a bus, the unsplit urethra was fixed with a similar transvesical anchor, the prostatic end being quite unsuitable for suturing. This is a most valuable manoeuvre.

In all these cases, assiduous dilatation at regular intervals conduced to a happy result.

I do not wish to generalize from these particular cases except to emphasize (1) the advantages of suprapubic suction drainage and (2) the fact that with care a very little urethra can be made to go a very long way. In my opinion practically no case should ever be abandoned to permanent suprapubic drainage or converted to colonic implantation as has been suggested elsewhere.

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meantime. In this formidable programme, fortunately, the boundaries of the territory are clearly defined and the subdivisions sharply cut.

In external cases where the extent of the injury is in doubt, in which there is retention due to spasm and a little perineal bruising with some bleeding from the urethra, I attempt a rubber catheter and if it can be passed I leave it in position for a few days. Instrumentation should be carried out only with real aseptic precautions, preferably in the theatre where operation can be undertaken at once if necessary. How else is one to examine a doubtful case? The patient must not be allowed to micturate for fear of causing extravasation. If the passage of the catheter is at all difficult or impossible the bladder is opened with *local anæsthesia*. This is important, as under *general anæsthesia* the patient may void and extravasate. He is next *anæsthetized* and placed in a high lithotomy position. Instruments are passed in both directions and the urethra dissected out and sutured as a ribbon. A small rubber catheter (No. 4) is then drawn through as a guide to further instrumentation. The bladder is kept empty by the suprapubic route with an Ainsworth Davies metal tube attached to a suction pump. This keeps it empty and prevents urine from trickling down around the catheter into the perineal wound where it is the probable source of such infection, necrosis and, ultimately, the stricture commonly attributed to the catheter itself (Silverstone).

Similar suprapubic drainage with suction is part of the treatment in practically every case whether early or late.

One recent case of external rupture was reported to me by 'phone from a distance. Unable to go at once I advised a suprapubic cystotomy only, under local analgesia, and for various reasons a week elapsed before the urethra was explored. I then found a very large hæmatoma with wide separation of the ruptured ends. It is of some interest to report that this man made as good a recovery as any other, with as satisfactory an end-result. The lesson of this is clear—namely that, in the absence of extravasation, drainage will help the patient over and that thereafter there is no objection to waiting a few days for expert help in the perineal part of the operation. Deliberate postponement of repair has been advocated as a routine by Lepoutre. The danger of this course lies in the possible presence of undetected extravasation.

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The boy recovered surprisingly well and two days later his wound was reopened and a urethral catheter was tied into the bladder. It was removed a month later, but he never regained control of his sphincter, and in spite of several dilatations a stricture soon developed. This led to an ascending infection and it has been necessary to dilate the suprapubic opening again and defer further instrumentation. He still is of course a long way from a happy result. The bruising of the costal margin was misleading but the case illustrates how severe hæmorrhage may be in this type of ruptured urethra.

I would like to submit the following simple rules for the emergency treatment of ruptured urethra.

ANTERIOR OR EXTRAPELVIC RUPTURE

The diagnosis is made if there is bruising in the perineum and bleeding from the external meatus. No catheter should be passed.

If the patient passes a normal stream of urine no special treatment is necessary at this stage, but he should be kept under observation, and his urethra should be examined after three weeks.

If there is any difficulty an immediate suprapubic cystotomy is performed to short-circuit the urine.

When there is a large perineal hæmatoma present it is incised and the urethra exposed. If the lesion is a complete rupture the proximal end is identified by passing a retrograde catheter and the roof is united by a few interrupted stitches. The wound is left open. The perineal incision and suture of the urethra are not essential at this stage and may well be left for a week and to a more experienced surgeon. Suprapubic drainage must never be neglected.

POSTERIOR OR INTRAPELVIC RUPTURE

If necessary a catheter may be passed to clinch the diagnosis. Unless a normal quantity of clear urine is obtained the diagnosis of rupture of the urethra or bladder is made. Accessory investigations are unnecessary and may be misleading. If the patient's condition permits it, the pelvis is X-rayed.

Immediate suprapubic cystotomy is performed, preferably under local anæsthesia when the patient is shocked.

If the lesion is incomplete a self-retaining suprapubic catheter is left in and the pre-vesical space is drained.

If the lesion is complete (i.e. when the base of the bladder is dislocated), the anatomy of the soft parts should be restored at once. A urethral bougie is passed till it just emerges through the membranous sphincter. A finger is then placed in the internal meatus and it is pushed down till the bougie can be made to enter the bladder. A self-retaining urethral catheter is then withdrawn. If the bladder still tends to redisplace, a rubber cuff is fitted to the catheter and continuous traction maintained. This type of rupture is the only one where an indwelling urethral catheter is indicated as well as suprapubic drainage.

All cases should be examined, preferably by a genito-urinary surgeon, within three weeks and regular dilatation commenced.

Finally it may not be too irrelevant to refer to the practice of "subincision" among the Australian aborigines. It is their custom to incise the urethra from below, and in many of the elderly men the incision extends from the external meatus to the scrotum. While studying water metabolism with a Sydney University expedition in Central Australia I often had to collect twenty-four-hourly specimens of urine from these men. I cannot remember any strictures and one is tempted to conclude that this was because doctors were very scarce and catheters were unknown.

Mr. E. W. Riches thought that the opening speakers had done well to raise so many controversial points. On the question of suprapubic drainage there seemed to be a general agreement that it was desirable. There was some difference of opinion about a perineal incision in bulbar ruptures; in many of the cases mentioned in the discussion or quoted in the literature such an incision had become necessary later because of abscess formation or extravasation, and he considered that in most cases it should not be deferred. Immediate or delayed repair was another debatable question; it must be decided by the general condition of the patient and the amount of local trauma. Whilst immediate repair was the theoretical ideal, delay might be dictated by a condition of extreme shock, but such delay should be minimal, especially in intrapelvic ruptures.

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Mr. Hugh C. Barry: These cases belong to the realm of accident surgery and it is mainly the chronic cases and the failures that find their way to genito-urinary specialists. In no condition is it truer to say that prevention is more important than cure. Many of the tragedies are caused by mismanagement in the first few hours. Moreover it is important to realize that at this stage often other complications, e.g. hæmorrhage, shock and a fractured pelvis, may need treatment as well.

Because the condition is so rare no single method has had an extended trial and even now the casualty surgeon is faced with a number of conflicting views. Moreover because it is an inexperienced casualty surgeon and not a genito-urinary specialist who has to treat these cases first we must give preference to some simple and safe method of management, and if possible make it standardized. I would like to report three acute cases I have treated recently as they illustrate three distinct types, and, I regret to say, most of the complications that may arise.

CASE I.—K. T. S., a schoolboy aged 11, was climbing a fence when he slipped and fell astride. He had a painful swelling of his scrotum and was unable to pass water. There was no bleeding from the meatus when he was first examined. A soft rubber catheter was passed and withdrew clear urine. As there was now a little bleeding from the meatus it was tied in for twenty-four hours. Three days later he got acute retention and it was reinserted and left tied in. Nine days after the accident as his scrotum was still painful and urine was leaking around the catheter a suprapubic catheter was inserted and the urethral catheter removed. A week later I opened a periurethral abscess and found an inch of his urethra in a sloughing condition. Fortunately in spite of this large gap the perineal wound healed and after four months he was passing water normally. He will however require regular dilatation for the rest of his life.

This case illustrates some of the pitfalls of an indwelling catheter. Even the change from a perineal hamatoma to an actual peri-urethral abscess was not as apparent to an unskilled observer as one might imagine. There was probably at first only a minute linear tear and I have no doubt that an immediate suprapubic cystotomy would have prevented this distressing result.

CASE II.—G. A., a man aged 32, was riding a motor cycle which skidded and crashed. He was able to walk home, but about an hour later noticed blood dripping from his urethra, and found he was unable to pass water. There was no evidence of bruising in the perineum. His lower abdomen was tender and dull on percussion, especially on the left side. There was no rigidity and intestinal sounds were present. A soft rubber catheter was passed and withdrew urine and a little dark altered blood. A subsequent X-ray showed a fracture of the left pubic ramus with very slight separation. A lower mid-line incision revealed an extravasation of blood in the prevesical space and an intact bladder. Suprapubic cystotomy was performed and a de Pezzer catheter tied in. The urethral catheter was withdrawn, and the prevesical space was drained. Three weeks later the urethra was dilated without difficulty and the de Pezzer catheter was removed. The suprapubic wound healed rapidly and he has remained well since, but is attending as an out-patient for regular dilatation. He has a stricture of the membranous urethra which though easily dilated has still a strong tendency to recur.

CASE III.—R. J. W., a schoolboy aged 7, was admitted to hospital, having been knocked down by a lorry. He was extremely shocked on admission (blood-pressure 68/40), showed signs of increasing pallor, and was obviously bleeding internally. There was a large bruise over the left costal margin, no bruising in the perineum and no bleeding from the urethra. The abdomen was tender, especially suprapubically, and no intestinal sounds were audible. X-ray showed slight separation of the symphysis pubis. A diagnosis of ruptured spleen was made.

An upper paramedian incision revealed a large extravasation of blood in the extra-peritoneal tissues but no intraperitoneal lesion. On prolonging the incision downwards the bladder was found distended and dislocated backwards. A urethral catheter was passed and entered the prevesical space. Suprapubic cystotomy was performed and the hæmorrhage arising from the base of the bladder was controlled by packing. A blood transfusion commenced before the operation was continued after his return to the ward.

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The use of an indwelling catheter provided the greatest bone of contention; it appeared to him that if stricture was inevitable, as was suggested by most speakers, the objections to an indwelling catheter lost most of their validity. In a case of intrapelvic rupture with dislocation of the bladder such a means provided the simplest method of accurate repair, and whilst the operation suggested by Mr. Morson was ingenious he thought that reposition around a catheter would probably achieve the same result with less disturbance.

He agreed with Mr. Morson on the value of internal urethrotomy in some of the late cases of stricture. He quoted a case where a large renal calculus of the "recumbency" type had developed within a year of a urethral rupture for which the patient had been in bed for six months.

Mr. R. L. Benison: I am surprised by Mr. Morson's advocacy of urethroscopy for the diagnosis of acute urethral injuries, and think that the urethroscope is possessed of all the disadvantages of a catheter, with the additional possibility of air embolism. In dealing with injuries of the posterior urethra, there is a danger of waiting too long before undertaking operation, as the injury itself is aggravating shock all the time until it is properly dealt with. I think that eight hours is about the maximum time one ought to wait. I have found that the introduction of a small-sized self-retaining catheter, with a sound in the anterior urethra and a forefinger in the internal urinary meatus, is the easiest method of dealing with these injuries.

Mr. Morson in answer to Mr. Benison's remarks pointed out that the technique of urethroscopy was totally different from that of catheterization. Thousands of urethroscopies were performed every year in this country and it was rarely that a case of so-called air embolism was reported. When a death occurred during urethroscopy post-mortem always showed cardiac degeneration.

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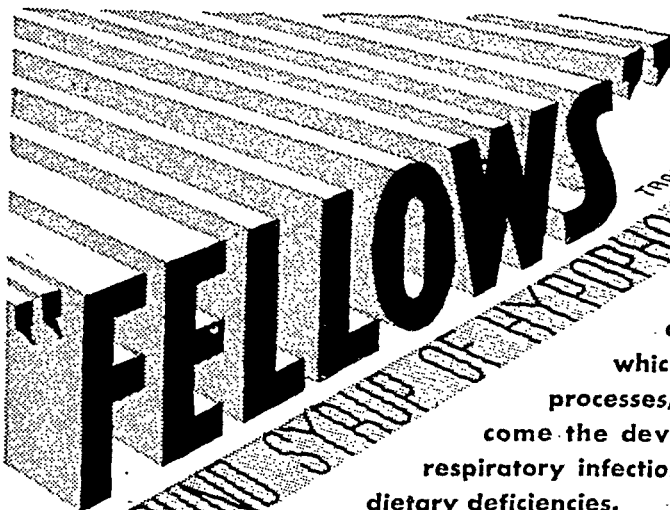
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Section of Neurology

President—GEORGE RIDDOCH, M.D.

[December 18, 1941]

DISCUSSION ON REHABILITATION AFTER INJURIES TO THE CENTRAL NERVOUS SYSTEM

Professor Geoffrey Jefferson (*in absentia*. Read by Colonel Riddoch): In peace-time most doctors were fully aware that a problem existed in the after-care of those who had suffered a head injury. It was a problem that interested not only the neurological fraternity who saw only a fraction of the cases that existed in the country as a whole, for it was not then, nor is it now, a matter in which any single type of specialist is concerned. The general practitioner is deeply interested, or at least heavily involved, and has a vital part to play. If we disagree sometimes with what he teaches his patients we have ourselves to blame for our teaching to him and, more generally, for our acquiescence with a state of affairs that has vaguely troubled our consciences without our becoming sufficiently aroused to excite concerted action. In civil hospital practice there have been great difficulties (and I fear that they will for some time continue) in pursuing a more rational policy, the chief being the lack of control over the conduct and welfare of his patient once he leaves hospital. One chief difficulty has been the deficiency in bed accommodation in first-class hospitals in which the patient might have been kept until his first stage of convalescence was completed. This has led to the patient rehabilitating himself as best he could under his doctor's supervision. And in an industrial population, which has formed the major part of my own material, this has proved to be a slow process, how slow the following figures testify. I looked up a consecutive series of 50 head injuries seen in private practice during the last two years to find out how soon these cases had been referred for specialist advice for the first time: The average time was six months (from three weeks to two years). Many were not seen until seven, eight or nine months or more after injury. By that time a neurosis had become firmly established in a number of the cases, and few were within measurable distance of returning to their work, although most were free from organic nervous defects. The worst offenders are the Insurance Companies, who allow the cases to drag on inconclusively for long periods, before asking advice from a neurological specialist. It is rarely that a case is settled in the High Court or Assize Courts in less than a year.

As to the validity of all headache after a head injury there are many honest doubters (and be it admitted, we have doubts ourselves). We are learning steadily about the mechanism of headache and are abandoning some time-honoured creeds, notably the relationships between pressure and headache as against local distortions of dural septa and traction on, or pulsation of, vessels. We do not yet know with certainty how to apply this knowledge to those with head injuries, except that we have comparative knowledge that pain that continues with unwavering intensity for months on end rarely has an organic cause. But we appear to be at length on the right lines. Attractive as the "unresolved contusion" of Trotter was, it seems unlikely to survive critical scrutiny either as an entity in itself or, it follows, as a cause of persistent pain. I must avoid speaking too much about the well-known post-contusional syndrome, and we must beware, also, of the danger of coming to regard all who have had a head injury as neurotics, though it would not be far wrong to regard all of them, as I believe that we do, as potential neurotics. Too rigid a belief in such a generalization would place the post-traumatic invalids under the shadow of suspicion and medico-legal abuse from which Trotter did so much to rescue them.

What plan can we evolve to rationalize the treatment of these cases, what can we do to improve the lot of those with head injuries? Anything that we, as a profession, do must have as its first aim the benefit of the patient. I do not think that we need have any qualms of conscience on that point. Rehabilitation serves the patient primarily; it protects the social fabric indirectly. I mean that we are not sacrificing him to the needs of the State, and my experience of our own people is that they are resisting sacrifices at any such altar. Gross abuses have occurred in the past, but they have been wholly in the patients' favour; they are likely to occur again if we find ourselves unable to agree upon a plan and to act accordingly. There is first, the treatment of the patient himself; second, the bringing in of the general practitioner and regimental or unit officers; thirdly, the re-education of the general public which makes up the patient's family, his friends, his employers, his lawyers, and his judges.

Rehabilitation of the patient: after head injury.—Rehabilitation may be defined as: "The planned attempt under skilled direction by the use of all available measures to restore or improve the health, usefulness and happiness of those who have suffered an injury, or are recovering from a disease. Its further object is to return them to the service of the community in the shortest time." Although it is a new word, rehabilitation is an old purpose. All medical treatment has basically no other aim. In the early stages of hospital treatment the ministrations and supervision of the physician and surgeon, with their staffs, is to the forefront. It was well recognized in peace-time practice that the discharge of the patient did not denote the end of treatment and by the use of convalescent homes, out-patient follow-ups, and especially by the co-operation of lady almoners, a basis for subsequent supervision was present. It was, however, most used for the purpose of scientific follow-up, much less with the object of placing the patient back in work, and so was imperfect. It would be untrue to say that rehabilitation in the sense in which the term is now used would start only at the point where ordinary in-patient treatment ends, for it should have begun before. But it begins especially at the convalescent stage if we speak of measures which our hospital system has, except in special instances, hitherto lacked. And, what is more, it is carried on until the patient returns to work. It is a continuum closely interwoven throughout with the general medical care of the patient. It is a work in which all can help, and does not fall within the ambit of any one man or of any one speciality. It is planned so as to give the patient a sense of his own importance as an integral part of the social structure, to give him not only physical but mental employment day by day, and throughout the day to ensure a sense of accomplishment, of achievement. It gives him the opportunity to discover that he is, however much he is damaged, an individual with powers that he can still usefully employ. It affords him the chance of discovering that his wage-earning capacity is not lost and it should allow him to turn to other trades or means of livelihood more fitted to his powers, if that be necessary. Rehabilitation has hitherto been applied only sporadically and there are great inequalities. Indeed, as a national and planned effort rehabilitation cannot as yet be said to exist and the present fluid state of the hospital system makes it a favourable time for an experimental beginning on a reasonably wide scale. These measures should be regarded as an essential part of treatment and should be commenced, in a graded way, *as soon as the patient's condition allows*. Thus, occupation in bed and in the ward is as important as occupation elsewhere for the ambulant patient. In this way the boredom, laziness and loss of adaptability, so often acquired during a prolonged stay in hospital, will be prevented. From the first the patient must be made to realize that he is an active collaborator in his own treatment and that he must accept some responsibility in his progress towards a successful result. At this point it would be as well to emphasize the need for discipline in the E.M.S. Hospitals which varies not only from one centre to another, but from ward to ward in the same place. Complaints about indiscipline reflect on the hospital as a whole.

We do not advocate the imposition of dictatorial rule, believing that co-operation of the patient can be achieved by explanation and reasoning and that habits of tidiness and punctuality can be inculcated without hardship and by example. This is an essential principle in treatment and applies to the relationship between doctor and patient in all hospitals, whether E.M.S. or not. The methods to be employed in such treatment should include:

(1) *Diversional and also constructive occupations* of sufficient variety and varying degrees of difficulty for those still confined to bed, as well as for those who are ambulant.

- (2) *Hospital maintenance work* in the ward and outside.
- (3) *Physiotherapy*, remedial exercises, massage, physical training.
- (4) *Intellectual and recreational pursuits*.

STAGES IN REHABILITATION

Rehabilitation may be divided up into stages corresponding with the progress of the patient from his original admission to hospital, through to his discharge and back to his home and employment.

Stage I

This covers the period during which the patient is in hospital—confined to bed or ambulant and is undergoing the early treatment appropriate to the local repair of his injury. Both Service and civilian casualties are included in this period under the agreement by which the E.M.S. cares for the injured, whatever their employment. In this stage the actual time at which the patient's interest is specifically aroused cannot be laid down definitely, because it will vary with the severity of the injury, the speed of recovery, the patient's age and intelligence, and the degree in which mental alertness and ability to co-operate have been depressed by the injury. But it should be laid down that rehabilitation in a diversional form should be instituted early whilst the patient is confined to bed. It will take the form of: Reading or being read to; jig-saw, cross-word, and other puzzles; drawing or colouring; needlework, string net and basket making; sewing, knitting, rug-making.

Speech therapy.—The retraining of speech by a Speech Therapist will be required in most cases of dysphasia. This treatment should be started when convalescence is well established, about the third or fourth week. For ambulant patients and those in wheel-chairs rehabilitation will be continued by book-binding; string and basket work; leather and metal work; simple carpentry, &c. Entertainments by concerts and films and, for the walking cases dancing is already available in some Brain Injury Centres. Patients after head injuries are often disturbed by noise and especially by general conversation. They may become distressed in a room where a gramophone is playing and other people are talking or playing games. A silence room of fair size and attractiveness should be set aside for them.

One of the most important services which rehabilitation will perform is that by instituting occupation to a time-table, the patient's day, or a considerable part of it, is dedicated to some pursuit. Clearly all such arrangements must be suited to the clinical state of the individual and the time-table will be most applicable to those advancing well in their convalescence. No doctrinaire rigidity is intended in any of the suggestions here advanced, which are to be applied with insight and judgment and modified from day to day, as needs be.

The scheme of rehabilitation above outlined refers to the patient with an uncomplicated head injury. If serious injuries exist elsewhere they might predominate and necessitate such modification of the plan as common sense dictates.

Physiotherapy will be required (it has of course already been provided in all the Brain Injury Centres). Physiotherapy should not dominate the general rehabilitation scheme which is a co-operative affair in which all branches play a helpful part. Undue emphasis has come to be laid on physiotherapy (massage, medical electricity in its various forms, and remedial gymnastics) because it has been the only kind of treatment commonly provided hitherto. Our hospitals, whilst well equipped for these treatments have lacked any organization for other convalescent pursuits, especially those that retrained the mind and took account of the personality and idiosyncrasies of the patients. It is our belief that of the two kinds of treatment, mental occupation and encouragement to do things, to make things, to use the whole body, is much the more important. Physiotherapy occupies a very small part of the patient's day and, except for remedial exercises, does not demand his co-operation. He is free for the rest of the day to follow the path of least resistance so that he tends to become the passive recipient of occasional treatment, dependent on others to get him well. However skilful physiotherapy may be, and it is commonly technically very skilful, many patients (not surprisingly) make slow progress.

The patient convalescent from head injuries is something very different. He should be urged to use his mind and to take an active part in his treatment, which will, without his interested co-operation, surely fail. With the other types of occupation included in rehabilitation there are things which excite interest over many hours of the day. In

several, e.g. where things are made with the hands, the important stimuli of competition and mimicry come into play, whilst in his constructive attempts he makes those very movements of his own with which physiotherapy teaches him for such short times and at intervals.

That the benefit of physiotherapy is often chiefly psychological cannot be denied, but this very fact calls for its intelligent use and the avoidance of misuse. During that stage of rehabilitation which coincides with the earliest stages of recovery, massage and encouragement to move paretic limbs will be the chief function of physiotherapy. Electrical methods are not advisable in the upper neuron lesions which form practically the whole of the material—the only exceptions will be the occasional facial nerve injuries and injuries to a limb or to peripheral nerves.

Stage I will last for about six weeks. This time might have to be extended in patients with chronic infections, cerebral fungus and the like, and those needing extended speech retraining. Modern surgical methods, and especially the new antiseptics give us grounds for the belief that long residence in hospital for purely surgical reasons connected with the wound will be uncommon.

The place of psychology in rehabilitation.—A bold statement on this question is necessary, and my belief is this that the proper use of the psychologist is that he should give reports on cases of head injury in much the same way that reports are given by other specialists, such as the radiologist and pathologist.

Whilst it is often instructive to know the intelligence quotient of a patient, it is more important for our purpose to assess his character, his emotional qualities, his recognition of values, his reactions to problems, his aims in life.

In the more serious injuries, where some degree of traumatic dementia has occurred, the psychiatrist's experience will fortify us in estimating the degree of mental damage.

Stage II

After some six weeks have elapsed since the injury we should be in a position to gauge the prospects of a case. Judged by experience from neuro-surgical operations, where variable degrees of considered and carefully inflicted damage is done to the brain, we know that in some six to eight weeks the effects of the operation have passed away. Reflections of this kind are important. Who can doubt that the two to three week period of invalidism that follows a section of the trigeminal root for neuralgia, or the slightly longer period after an operation for a pituitary adenoma, would be enormously extended in time were it the result of accident. Most scars are healed in two months, and after that period has elapsed the pain of a fractured skull (and I believe that most clinicians underate bone pain) should have faded away.

Once symptoms have abated, the second stage or period of hardening commences. It seems desirable that military cases should be taken over by the respective Services for this purpose, since this gives an opportunity for canalization towards the specific goal of their line of work, whichever it may be. I shall leave discussion of the management of Service cases to the proper authorities. I will, however, outline the types of activity which are useful once the patient is fully ambulant and relatively free from complaints.

It is at this point, when daily medical and surgical supervision is no longer needed, that we advise separation of the military or Service casualties from the civilian, and it is here that the chief reconstruction and replanning between the E.M.S. and the Services will be advisable. Service patients recoverable for Service purposes ought to be rehabilitated in Service Depots under discipline and in Service pursuits.

Invalided Service and civilian patients.—The methods of treatment to be employed will be, in the main, the same as in Stage I, but with additions such as: (1) Gymnastics and physical training. (2) Continuation of physiotherapy when necessary. (3) Basket making, leather and metal work, the patient being allowed to keep the finished article on paying the cost of materials. (4) Speech-training for those with dysphasias. (5) Work in the garden. (6) Work in the carpenter's and engineer's shops. (7) Organized games—cricket, football, netball, baseball, rounders, and open-air games. (8) Walks outside the hospital grounds. (9) Visits to the town, shops and cinema, but usually with the purpose of finding out how the patient reacts to the jostle of people and the noise of traffic, to navigating himself as an entity amongst his fellow men. The same of visits to the cinema—how well does he sustain visual and auditory attention over a period of two hours or so and is he upset by the incidental noise? No patient with a head injury should go on

a bus or train journey until he has proved his ability to look after himself in this way.

It is not intended that these visits should be purely educational or therapeutic, but it is important that the doctors, in whose charge the patients are, should realize the lessons to be learned from these extramural activities. The patients must therefore be questioned about their reactions to these excursions, and progress notes made.

Physiotherapy will be applied where it is needed. The recreational side, such as attendance at dances, will be more in evidence.

An essential of rehabilitation is that it should be planned to a time-table each day. Patients should continue with a pastime or constructive work only for definite periods; he should be stopped whilst he still wishes to go on. In that way interest is maintained. Hence application at any one thing should be relatively short, especially in the earlier days of convalescence. The best plan will be to arrange 6 sessions for each patient in the day, of from forty-five to sixty minutes each, say from 9 to 12 and from 1.30 to 4.30. Everyone should be told his duties for the week with, say, Wednesday and Saturday afternoons free.

After six weeks or so in hospital with plenty of interest during Stage I he might feel himself quite well enough to return to home and to work without further preparation. It may be objected that patients will be reluctant to continue with further treatment, preferring to go home. The confidence of the patient in his treatment and the enthusiasm with which his doctors are able to advocate a continuation will determine his willingness.

The most important stage of rehabilitation is the first. The patient should be frankly told what sort of an injury he has had and what we expect the course of it will be. A great deal of our difficulties in the past have arisen because we have not been able to retain our head injury cases for long enough. Much can be done by giving the patient's doctor not only the history of the case, but also warning him against the possibility of the patient becoming a passive resister to cure. The mental resources of many of the industrial population are so few that the wonder is how they fill in the weeks and months and sometimes years that they spend in recovering. We can do much by establishing contact with the general practitioner and his counterpart in the Army, the Regimental Medical Officer. Further, by speech with the wife or relatives of the patient we can make them aware of the most important facts about head injuries and their course and can divert them from a degree of over-solicitude.

Professor Hugh Cairns: In any discussion on rehabilitation of head injury patients it is important to consider the main factors which influence the patients' disability.

(1) *The time factor.*—The time factor varies greatly from case to case. After a head injury the patient goes through a cycle, beginning with unconsciousness, thence through a stage of confusion and a period in which he is liable to headaches, to a final stage in which he is restored to normal or near-normal. This is a spontaneous process. The time taken for its completion may be in one case only a few weeks, in another as long as eighteen months, depending to a large extent upon the severity of the initial injury.

This time factor must always be taken into account in planning the rehabilitation of head injury patients, the more so because we do not yet possess adequate tests by which to measure the recovery of the higher levels of intellectual function. A man may appear on testing to have recovered his faculties completely, and yet he will fail on the intellectual level when he returns to his work. In deciding when a patient should be fit to return to work it is therefore necessary to bear in mind the severity of his injury, and the best yardstick at present available is the duration of the post-traumatic amnesia (P.T.A.). As a working rule I would suggest that the shortest time in which ability to carry out full work may be expected to return is as follows:

| | | | | | |
|-------------------------|-----|-----|-----|-----|------------|
| P.T.A. 5 minutes—1 hour | ... | ... | ... | ... | 4—6 weeks |
| P.T.A. 1—24 hours | ... | ... | ... | ... | 6—8 weeks |
| P.T.A. 1—7 days | ... | ... | ... | ... | 2—4 months |
| P.T.A. over 7 days | ... | ... | ... | ... | 4—8 months |

I put this forward tentatively as a rough measure to be used in planning rehabilitation. It can be no more than a rough working guide because the duration of post-traumatic amnesia is influenced by other events besides the severity of the brain injury, e.g. the amount of bodily fatigue present at the time of injury, large doses of morphia or a general anæsthetic shortly after the injury, epileptic seizures in the first days after injury, and various psychoneurotic factors. Furthermore, we know that in certain cases where

there is a fracture of the base of the skull and the amnesia is shortlived, damage to the cranial nerves may interfere with return to work long after the higher cerebral functions have recovered. The type of work must also be taken into account; a simple task may be resumed earlier than one which involves the higher levels of intellectual function.

(2) *Disturbance of mental capacity.*—The central disturbance, the main organic cause of disability after head injury, is disturbance of mental capacity. It may occur at various levels of mental activity. At the lower levels there may be disturbance of speech, reading, calculating, or orientation. Initiative, memory and concentration may be affected. At higher levels there may be impairment of judgment, or of the capacity for abstract reasoning. The patient cannot be satisfactorily guided through the stages of rehabilitation without an attempt on the part of the doctor—however halting it may still be—to assess his mind in terms of these functions. After severe head injury the question will arise whether the patient is likely to be fit for his previous work, and this will often largely depend upon his retention of the capacity to learn. While the patient continues to show improvement an adverse decision should usually be withheld.

(3) *Emotional disturbance.*—Emotional factors are inextricably bound up with the process of recovery. The unstable type of man will have additional difficulties in adjusting himself to the task of returning to work, and it is therefore incumbent on those responsible for rehabilitation to assess the man as well as the severity of his injury. This is best done not only by observing the patient's behaviour while under observation, but also by a systematic inquiry into his family history, and into his earlier life and illnesses. The doctor may learn, especially in the cases of soldiers in war time, that before his accident the man was not employed, or felt that he was not employed, in the type of work for which he was best fitted.

Emotional disturbance may interfere with all aspects of intellectual activity and may be itself precipitated by these defects. The patient who becomes confused at a task tends to have a feeling of inadequacy which may lead not only to headache and outbursts of irritability but also to complete suppression for the time being of his powers of thinking: the phenomenon described by Goldstein as the catastrophic reaction.

(4) *Post-traumatic headaches.*—Anyone who is responsible for the rehabilitation of patients with head injury must formulate his ideas about the post-traumatic syndrome—the attacks of headache, dizziness, irritability and confusion which occur so commonly after the initial recovery from head injury. There are large gaps in our knowledge of this syndrome, and no little disagreement as to its nature. It is necessary, nevertheless, to have some working hypothesis, otherwise what shall one say to the man who complains of headaches during convalescence? I believe that almost every patient who makes a full recovery from concussion suffers at a certain stage of his recovery from headaches. In the mild head injuries the headaches may occur sporadically for some weeks and then disappear. In the severe cases the headaches may not come on until long after the patient has left hospital, and may not come on at all if there is any serious degree of residual organic defect. The patient must have recovered a certain amount of mental clarity before he appreciates headaches.

Now it is one thing to have a headache; it is another thing to complain about it. Although attacks of headache occur fairly constantly the reaction of the patient to them varies considerably, depending on a variety of factors, such as the emotional make-up of the patient, his anxieties and fears, his ability and desire to cope with his work once more. Emotionally stable, well-adjusted, and reliable witnesses—for example, most doctors—will describe their difficulties in concentrating when first they return to work; they will ascribe their headaches to attempts to concentrate, their headaches or dizziness to exertion or change of posture. Identical symptoms are complained of by other patients who are unstable and badly-adjusted, but nevertheless reliable witnesses. The difference is that the first type will manage to carry on with their rehabilitation with the minimum of interruption, while the second type are likely to break down on return to work unless they are given special treatment, treatment that inevitably comes within the purview of rehabilitation.

If this view as to the incidence of post-traumatic symptoms is correct then we must regard the syndrome as organic, just as syncope is; but, like syncope, it is influenced by emotional and psychoneurotic factors.

The early stages of recovery.—Rehabilitation begins when the patient begins to talk and respond properly. How does he discover that he has had a head injury? As his con-

fusion clears he slowly puzzles it out for himself, with the aid of explanations which come better from the doctor, with reassurance, than from the relations. The explanations and reassurance may need to be repeated more than once, for although the patient may be able to conduct a conversation in a manner that gives a superficial appearance of normality, yet he is at this stage confused, disorientated, and forgetful, for a period varying between minutes and weeks, according as the brain injury is mild or severe.

This is the stage at which assessment of the brain damage and of the type of the patient can first be undertaken. Without such investigations the rehabilitation cannot be adequately planned.

Getting up.—Notwithstanding the recommendations of Symonds these patients are often kept in bed too long, on the assumption that the liability to post-traumatic headaches is thus diminished. There is no evidence in favour of this view, and the practice becomes a bad one when, as so often happens, the next stage of rehabilitation is hurried.

There is rarely reason why the patient should not be allowed to get up a few days after he has recovered consciousness. Thus, a man whose total amnesia is six hours or less can be got up gradually towards the end of a week. If he gets a headache, he can be put to bed with some aspirin, and can get up again next day. Recourse to bed is, however, rarely necessary, for headache is not a conspicuous feature of this stage of recovery. The fact that the patient is got up early does not mean that he is to go back to work early. In cases of prolonged confusion and irritability, after unconsciousness of a week or more, the effect of sitting the patient out of bed and of giving him baths is often most soothing.

Graduated physical activities.—From the stage of getting up graduated physical (and mental) exercise may begin. It is necessary to grade the exercises, and the speed with which the patient passes from one stage to the next depends, not only on his progress with the exercises, but also on the estimated severity of his injury. For the mildly concussed the physical exercise can take first the form of walking and light physical training, thence by stages to more strenuous physical training, games, and heavy physical work. For the severely injured who, when they first get up, are still confused and even disorientated, light exercises to music, gentle indoor ball games, and deep breathing exercises are useful. These patients need much individual attention, and plenty of rest between exercises. Patience is necessary, also, for they often forget a game and need to have it explained to them all over again. No exercise should be pushed to the point of provoking a catastrophic reaction.

What is the value of physical exercise? It hardens the patient physically, thus preparing him to adjust himself the more easily to the physical demands of his normal life. It provides also a sense of achievement which is valuable in a man who is mildly confused and incapable of concentrated mental effort. It helps him to escape boredom. And it is perhaps also beneficial in modifying intracranial pressure.

At what stage should the patient be called upon to do violent physical training, Swedish exercises, and the like? Opinions differ on this question. There are some who would start it within a few weeks of quite severe injuries; others would introduce it more gradually and at a later stage. Before doing severe exercises the patient should, in my opinion, pass successfully through a stage of light competitive games.

Occupational treatment.—While physical re-education is going on handicraft can provide simple tasks within the powers of most patients, tasks which occupy their time at a stage when they are incapable of reading or talking consecutively for any length of time and which give them a sense of achievement: leather-work, basket-work, poker-work, weaving at a hand-loom, and book-binding. Heavier crafts such as carpentry, metalwork, weaving on large looms, and outdoor occupations, such as gardening and house-decorating, come later.

In craft work the teacher must constantly bear in mind that what really matters is not the initial standard of performance, but the degree of improvement shown from day to day. So often the star pupil of the class proves to be a psychoneurotic with headache after a trivial head injury.

Mental exercise.—The simplest mental exercises are those connected with movement, where the alternations of movement require concentration. For the confused patient simple games of a childish kind without competition are useful, for example, sorting cards according to colours, shapes, or what is written on them. It is an index of their degree of confusion that grown men will play these games with considerable pleasure.

In the later stages mental exercises should be directed towards the specific defect. Testing of the mental processes comes first and is in itself treatment. Special exercises can be introduced for lower level defects, such as those of reading and calculating. Sometimes it is possible to supplement the damaged functions, as, for example, by encouraging the patient with a defect of reading to trace or copy the words he is trying to read, or by teaching lip-reading to a patient whose difficulty is in understanding the spoken word. The prognosis depends on the residual learning capacity.

It is doubtful whether special, graded mental exercises can seriously compete with the patient's own attempts to re-educate himself, once he has, by testing, been shown the way; but the value of encouragement by means of continued supervision cannot be overestimated.

Visual treatment.—No patient can relearn intellectual tasks unless his sight is good, and as many of these patients have visual symptoms, diplopia, phorias, or errors of refraction, it is important that ophthalmological treatment should be given whenever necessary.

Psychological factors.—The importance of psychological factors has already been indicated, and no attempts at rehabilitation are likely to be successful unless the patient's anxieties and fear are assuaged and unless he is helped through the phases of depression and the other disturbances of feeling that so often beset him during recovery from head injury.

The final test.—It is very difficult in hospital or convalescent institution to provide a test of full recovery, and the final test is the patient's return to work. It is the duty of those in charge of rehabilitation to see that the man is adequately supervised during this period, and that any special points concerning graduation of his work in the initial stages should be made known to his family doctor, his works doctor, or his Unit medical officer.

Dr. W. Russell Brain: For a long time it has seemed to me that the high incidence of neurosis after injury, particularly after industrial injuries, and our continued neglect to deal with it as a substantial medical and social problem is a considerable reproach to us as doctors. The first step must necessarily be an attempt to ascertain why traumatic neurosis occurs. This question can be approached in two ways, either by a psychological investigation of patients suffering from neurosis after injury, or by a statistical inquiry into the characteristics shown by a group of such patients. The two methods are supplementary. Psychological investigation of injured individuals is difficult for many reasons. My method this afternoon is the second one and I shall concern myself only with such psychological facts as seem to emerge from a study of the group as a whole.

I have long had an impression that traumatic neurosis occurs much more frequently in patients suffering from the effects of industrial injuries than in those injured in road accidents, and this supposed difference suggested itself as a promising basis for an inquiry. I have therefore taken as my material 100 consecutive cases of male patients suffering from the effects of a head injury acquired in a road accident, and 100 consecutive cases of male patients suffering from the effects of an industrial head injury. All patients were seen some time after their injury, the average interval before they came under observation being ten months. I have divided the patients in each group into three sub-groups according to whether their head injuries were mild, moderate or severe. I have called mild those injuries in which either consciousness was not lost or the patient was merely dazed for a short time. I have called moderate those injuries in which consciousness was lost, but the patient had recovered normal mentality in twenty-four hours and I have classified as severe all cases in which loss of consciousness or mental confusion lasted for more than twenty-four hours. The state of consciousness, however, though important, is not the sole criterion of the severity of a head injury and I have taken into account severe focal cerebral contusion when this occurred with only brief loss of consciousness. I have also divided each group into three sub-groups according to whether I consider the patient's symptoms to be purely organic, purely neurotic or mixed. It is clear that this classification depends to a considerable extent upon personal judgment. There is no difficulty in deciding about most hysterical symptoms. In assessing the nature of other symptoms I have depended upon such criteria as the characteristics of the headache, especially its constancy or otherwise and its relationship to precipitating factors, the effect of the passage of time upon symptoms and the attitude of the patient to them. No doubt others would have classified individual cases differently, but perhaps this is not of great importance since the same methods have been applied to all the cases by the same observer.

INCIDENCE OF NEUROSIS AFTER ROAD ACCIDENTS AND INDUSTRIAL INJURIES

Table I shows the frequency with which neurosis follows head injuries (1) after road accidents and (2) after industrial accidents.

TABLE I.

| | Neurotic | Mixed | Organic |
|------------------------------------|----------|-------|---------|
| Road accidents—100 cases ... | 8 | 15 | |
| | 23 | | 77 |
| Industrial accidents—100 cases ... | 23 | 32 | |
| | 55 | | 45 |

The next step is to inquire whether there is any correlation between the incidence of neurosis and the severity of the injury. This is shown in Table II.

TABLE II.

| | Neurotic | Mixed | Organic | Total |
|---------------------------------|----------|-------|---------|-------|
| Road accidents—Severe ... | 1 | 11 | 48 | 60 |
| Moderate ... | 3 | 2 | 18 | 23 |
| Slight ... | 4 | 2 | 11 | 17 |
| Total | 8 | 15 | | |
| | 23 | | 77 | 100 |
| Industrial accidents—Severe ... | 4 | 8 | 22 | 34 |
| Moderate ... | 8 | 12 | 10 | 30 |
| Slight ... | 11 | 12 | 13 | 36 |
| Total | 23 | 32 | | |
| | 55 | | 45 | 100 |

Table II shows, first, that the injury is severe in a much higher proportion of patients who have had road accidents 60% compared with 34% of those who have had industrial accidents; and secondly that the industrial group shows a high incidence of neurosis associated with the less severe injuries, which is not present in the road accident group. This is better shown in Table III.

TABLE III.

| | Neurotic and Mixed | Total | Percentage |
|---------------------------------|--------------------|-------|------------|
| Road accidents—Severe ... | 12 | 60 | 20 |
| Moderate and slight ... | 11 | 40 | 27 |
| Industrial accidents—Severe ... | 12 | 34 | 35 |
| Moderate and slight ... | 43 | 66 | 65 |

It is quite clear that a severe head injury as such is not an important cause of traumatic neurosis, but we have still to explain the apparently paradoxical correlation between neurosis and the slighter injuries.

It must be remembered that we are here dealing not with totals of injured persons, but with persons selected from the total number injured because the severity of their symptoms (1) in the road accident group calls for compensation (2) in the industrial accident group incapacitates them for work. It will generally be agreed that the incidence of severe head injuries is much higher in those who have been involved in road accidents than in a group exposed to the varied hazards of a large number of industries. Allowing for the fact that in some industries the risk of head injuries is much greater than in others it is still true that a mixed group of industrial workers is exposed to somewhat rare severe injuries, and fairly frequent moderate or slight injuries. If now the incidence of neurosis is independent of the severity of the injury, if, in other words, there exists

in the exposed community a proportion of individuals who react to even a slight injury by developing a neurosis, the high proportion of less severe injuries will lead to a selection of these susceptible persons, since those with slight organic symptoms will soon return to work and therefore will never reach the neurologist. Hence there will be an apparently high incidence of neurosis complicating the slighter injuries, in the industrial group.

But in the industrial group the 35% incidence of neurosis complicating severe injuries is significantly higher than the 20% in the road accident group which may perhaps be regarded as a sample of the general population at these ages; and the generally raised liability to neurosis in the group calls for explanation.

AGE AND OCCUPATION

There is no significant difference in the average age of patients in the two groups, nor between the average ages of those with and those without neurosis in either group, so that there is no evidence that age plays any part in the aetiology of traumatic neurosis.

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| Industrial | Professional and clerical ... | 2 | — | — |
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This table shows first that in both groups the incidence of neurosis is highest in the unskilled labourers; in fact out of 23 patients in the industrial group who developed neurosis after slight injuries 17 were heavy unskilled labourers. Secondly, men of any occupation are substantially more likely to develop traumatic neurosis if they are injured in an industrial accident, even though their organic injury is likely to be less severe. I would briefly suggest two reasons why a slighter industrial injury is more likely to produce neurosis than a more severe road accident.

First, an industrial injury is an occupational injury, as a road injury usually is not. It therefore suggests to the workman that his occupation is a dangerous one, and it is likely to cause a sense both of physical danger and of economic insecurity. Secondly, the legal procedure is widely different in the two groups. In a road accident the insurance system is a guarantee of compensation in most cases: in the machinery of the Workmen's Compensation Acts it operates in the opposite sense, since the workman's right to compensation is repeatedly under review and frequently contested. Hence although the workman injured at work receives immediate compensation which his fellow knocked down by a car does not, his economic security is really far more seriously threatened. There are other defects, of a similar kind, in the Workmen's Compensation Acts which contribute to the development of neurosis but which I cannot now discuss.

SUGGESTED REMEDIES

(1) Rehabilitation in all cases of slight injury begins with the general practitioner or casualty officer who first sees the patient. These front-line workers need to learn the right psychological handling of the injured man; to avoid suggesting serious injury when none exists and equally "a narrow escape". They should learn also to avoid the hospitalization of really slight cases.

(2) All big centres need units of doctors trained to deal with head injuries, both slight and severe. In this way only is it possible to avoid the ill-effects of scattering these patients in general wards under the care of surgeons who have neither the knowledge nor the interest needful.

(3) Occupational therapy should merge into therapeutic occupation, i.e. organized light work, adjusted to each man's capacity.

(4) The social responsibility for the injured should be more widely recognized and the emphasis changed from compensation to rehabilitation. The need for compensation is to a large extent a measure of the failure of rehabilitation.

(5) The Workmen's Compensation Acts should be revised so that an industrial injury should not immediately involve a potential dispute about the ownership of a sum of money. Medical issues should be decided by medical boards closely linked with rehabilitation units; and an adequate maintenance allowance should be guaranteed to the injured man as long as the medical board regarded him as disabled and as long as he co-operated in his treatment and rehabilitation.

(6) At the same time it is important to avoid an exaggerated paternalism. Here we have to face an aspect of the largely unsolved problem of modern society, how to provide social services without robbing the individual citizen of his sense of responsibility for his own welfare. There would be no traumatic neurosis if all the injured were like an Irish hunting woman who consulted me because after the last of many falls she thought her double vision was worse than it used to be. Such toughness is doubtless partly innate, partly cultural. Society can do a good deal to foster it if it is conscious of the need. As doctors we have the double task of educating both individuals and society.

Dr. Ludwig Guttmann: Rehabilitation after peripheral nerve lesions means restoration of the working capacity of the injured person. It includes, apart from the actual surgical treatment, all methods which accelerate functional recovery:

(1) Therapeutic arrangements of a more passive or preventive nature, such as precautions for keeping the paralysed limb in the right position with and without splintage and physiotherapy.

(2) All arrangements which accelerate functional recovery with the patient's active co-operation until he is enabled to resume his former occupation, or failing that to do useful alternative work in the same industry. Arrangements of this group are (a) remedial exercise; (b) occupational therapy; (c) some physiotherapy; (d) reconditioning.

(3) Vocational training, i.e. training of those crippled by peripheral nerve injuries for special occupations adapted to their permanent disability.

As there are some transitions between the individual groups this distinction may not be considered as absolutely strict. According to this definition it is obvious that rehabilitation after peripheral nerve lesions has to start immediately following injury. Dr. E. A. Nicoll's opinion expressed on this subject in a recent paper on fractures "Rehabilitation starts on the first day of treatment" can be accepted in the full meaning of the words also for peripheral nerve lesions.

General organization.—The installation in this country of several centres for the treatment of peripheral nerve injuries is a great step forward. The congregation of cases in a single department under the same specialized staff, with continuous treatment under the same supervision, is certainly the best guarantee for a systematic study of the whole question, and for better results. The success of a centralized treatment and care of peripheral nerve injuries in other countries was shown by the "Peripheral Nerve Centres" in the U.S.A. during the last war and particularly by Foerster's work in Germany during and after the last war. His material included about 4,000 cases. Although he worked under conditions by no means ideal compared with those of a modern centre in this country, his results were remarkably good and better than those of many other authors of that time. Foerster has emphasized again and again the secret of his better results. It was only in some respects a specialized surgical technique; the main reason was a better and systematic after-treatment and after-care, in other words, a good understanding of rehabilitation.

The installation of centres for peripheral nerve injuries, however, does not cover the whole problem of organization in the rehabilitation work. In practice it is not possible to bring all cases into these centres, particularly in the early days after injury. Therefore precautions should be taken in all General and Military hospitals, particularly in *Military Base-Hospitals*, that the injured can be seen immediately by a Nerve Specialist versed in the after-treatment of peripheral nerve lesions. Neglect of this vital principle of rehabilitation in the first period, even in the first days after injury, accounts for much of the prolonged disability of the injured person, with all its economic consequences. The

in the exposed community a proportion of individuals who react to even a slight injury by developing a neurosis, the high proportion of less severe injuries will lead to a selection of these susceptible persons, since those with slight organic symptoms will soon return to work and therefore will never reach the neurologist. Hence there will be an apparently high incidence of neurosis complicating the slighter injuries, in the industrial group.

But in the industrial group the 35% incidence of neurosis complicating severe injuries is significantly higher than the 20% in the road accident group which may perhaps be regarded as a sample of the general population at these ages; and the generally raised liability to neurosis in the group calls for explanation.

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unsolved. Whatever splintage may be used it is of vital importance that any direct pressure of the splint on the paralysed muscle must be avoided, and "splint-free" intervals should be given.

STRENGTHENING OF SYNERGISTS

The taking over of the function of a paralysed muscle by one or several of its normal synergists can be of great practical value in rehabilitation. Besides the well-known compensatory function of the brachioradialis in a biceps palsy and other instances the following example illustrates this principle: In a case of complete paralysis of the axillary nerve abduction of the arm may be carried out astonishingly well by the supraspinatus muscle. It is possible in such cases to dispense with splints altogether and to restore function and working capacity by strengthening such synergistic muscles.

ELECTROTHERAPY

It is one hundred years since John Reid in Edinburgh (1841) first described the beneficial effect of galvanic current on denervated muscles. Since then opinions about the value of electrotherapy in the treatment of peripheral nerve lesions have oscillated a great deal. Experiments on the subject do not allow satisfactory conclusions, and even the great clinical experience of the last war has not settled this problem. Yet experts are agreed that electrotherapy is useful in accelerating rehabilitation after peripheral nerve lesions. My own experience has endorsed the value of electrotherapy carried out by galvanic exercise of the paralysed muscles. If carried out regularly and properly by a reliable person having the necessary knowledge of the anatomy and physiology of movement and under permanent medical supervision it is a valuable auxiliary method in the treatment of atrophy and in maintaining or improving the elasticity of paralysed muscles. This has been proved recently by an experimental study on galvanic treatment of denervated muscles in rabbits carried out in Dr. J. Z. Young's Department of Comparative Anatomy in the University of Oxford by Dr. Ernest Gutmann and myself (1942). Although in these experiments the daily galvanic exercise of the paralysed muscles did not entirely prevent the onset and progress of atrophy immediately after denervation, it had, however, a definite delaying and diminishing effect on the atrophy in later stages. Moreover it accelerated the recovery from atrophy, once the muscles had become reinnervated and recovery of function had commenced. The histological findings in our cases eliminate the suspicion that the greater volume of the treated muscles might be the effect of a hypertrophy of the connective tissue. Exactly the reverse was found, for the treated muscles showed far less fibrosis.

It may be noted that in cases of complete paralysis galvanotherapy is the only method (in contrast to massage) which enables the paralysed muscles to exercise their original function—or to put it in Foerster's own words: "Electrotherapy reminds the paralysed muscle of its normal task." Furthermore electrotherapy is most useful in augmenting active exercise in those cases in which regeneration is taking place but the patient co-operates poorly. On the other hand, it would be overrating the value of the method to imagine that atrophy of muscles can be prevented or considerably diminished in all cases of denervated muscles. In my own cases in which great vascular damage had occurred in addition to the nerve injury or in which the muscles themselves had been very considerably damaged the beneficial effect of galvanic treatment was not so convincing.

REMEDIAL EXERCISE

Active exercise is of *cardinal* importance in achieving the most complete and quickest rehabilitation. In recovering lesions three different stages require special consideration:

(1) As long as voluntary movements are still weak the exercises should only be directed towards overcoming the inertia of movement, and should not work against gravity. The simplest way to achieve this is by placing the limb in such a position that movements are carried out in the horizontal plane. Exercises in a bath are in some cases helpful.

(2) Having overcome the inertia of the limb without any other help, exercise should be attempted against gravity.

(3) If the muscle is able to move the limb against gravity, graduated weights are added against which the muscle has to work.

importance of this point can hardly be exaggerated. "An integral part of the organization of what might be called "Primary rehabilitation service" is a thorough record of all treatment given in the first period after nerve injury. Undoubtedly such a service would greatly facilitate the work of the centres for peripheral nerve injuries and would play a big part in improving the end-results.

Of the same importance as the primary supervision immediately after injury is the late supervision of these cases after their discharge from hospital, from the centres and from the Army. This late supervision also includes the post-war supervision of peripheral nerve injuries. Experiences in all countries after the last war have clearly shown that any successful late supervision of these cases can only be achieved by a loyal co-operation of the medical authorities with the public health services and—as Cairns and Young pointed out (1940)—with the Ministry of Pensions and, last but not least, with the employers. Such an organized co-operation of the various authorities concerned with the rehabilitation work is of particular importance in the reconditioning period of the injured. One of the main tasks of the "after-care service" is (1) to provide the injured man with light and graduated work in his former occupation until he is fit for heavy work; (2) to supervise this light and graduated work. In my own experience the best results in supervising the injured persons during the reconditioning period were obtained with the help of industrial medical officers and general practitioners. Experiences in all countries have shown that many patients, left alone in the reconditioning period, will never make sufficient effort to reach their full working capacity.

In discussing some methods of particular importance for a speedy and, if possible, complete rehabilitation only a few points can be considered. Cases with peripheral nerve lesions can be grouped into those in which restoration of nerve conduction is possible and those in which there is no chance of nerve regeneration. In regard to treatment, however, this distinction is not an absolutely strict one as similar principles have to be considered in both cases up to a certain point.

POSITION OF PARALYSED LIMBS

The position of paralysed limbs follows the principle of close approximation of the points of attachment of the paralysed muscles. The principle underlying treatment is to maintain and increase elasticity of the affected muscles. All experts on the subject agree that overstretching of a paralysed muscle even in the very first period after injury means a severe and often irreparable additional damage of the paralysed muscle. In 1916 Sir Robert Jones expressed it very clearly: "The most skilful operation performed on the most suitable case will prove a fiasco unless the affected muscles are continually kept relaxed until recovery takes place." Unfortunately, this well-known and most essential point is in practice not always duly considered and may even be completely neglected. It is for instance amazing how often this principle is neglected in cases of ulnar and median nerve palsy in spite of the fact that special splints have been described for the approximation of the intrinsic muscles of the hand.

In preventing the claw position of the fingers in cases of ulnar nerve palsy two types of splints are recommended. The one is designed to maintain the proximal phalanges in a position of medium flexion, at the same time preventing flexion of the middle and terminal phalanges. The other, recommended particularly for isolated paralysis of the interossei, maintains the greatest possible extension of the long flexors of the fingers by means of narrow elastic splints which are applied to the dorsal side of the fingers, thus extending all phalanges as far as possible. The advantage of these splints, according to Foerster, lies in the fact that the middle and terminal phalanges remain sufficiently stretched during all the finer activities of the fingers in which small objects are grasped and moved between the tips of thumb and index or middle finger.

In median nerve palsy, in which relaxation of the thenar muscles is so important, splints have also been designed to bring the thumb into a position of abduction and opposition while its terminal phalanx is extended. Among other authors Foerster (1929) has recommended such a splint, which, however, tends to be too inflexible. On the other hand splints, such as recommended by Sumner L. Koch and Michael L. Mason (1939), which are more flexible have the disadvantage that the thumb is held in adduction and in too strong flexion at its metacarpo-phalangeal joint, and they do not give the necessary abduction and opposition of the thumb. The problem of suitable splints for isolated median palsy and also for combined median and ulnar or radial palsies is therefore still

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The principle of the exercises is to effect quick movements over the widest possible range. The movements are followed by fairly long rests, after which they are again repeated. This kind of exercise has always to be supervised since the patient when left alone is usually inclined to exhaust himself in multiple partial performances as opposed to a few maximal responses which are of greater benefit. Since many patients never make progress without assistance a daily supervision of active exercise by the doctor has undoubtedly the best effect in obtaining the patient's energetic co-operation and unremitting effort which are so necessary for successful rehabilitation.

OCCUPATIONAL THERAPY

Occupational therapy at the centres for peripheral nerve injuries might be a valuable addition to active exercise treatment. In cases where restoration of nerve conduction is possible it will accelerate rehabilitation. In cases of permanent paralysis it will help to develop and to improve subsidiary and trick movements. It should, however, be emphasized that in order to build up occupational therapy as a really useful and reliable method for accelerating rehabilitation, a carefully elaborated organization and a plan of indication, time of onset and duration, have to be established. These, as far as I know, have not yet been developed in regard to peripheral nerve lesions. Although N. A. Haworth and E. M. Macdonald (1940) published a very suitable list of suggested occupations in peripheral nerve lesions and some authors let their patients work with suitably altered tools, a special technique of occupational training is not yet fully formulated, and the various crafts used in occupational training have not yet been analysed and suitably modified from the physiological and psychological point of view. In a median or ulnar nerve paralysis, for instance, all the arrangements for occupational training must be conditioned by the anæsthetic and atrophic state of the skin and subcutaneous tissues. Exercises which can macerate the skin such as with moist clay, must be avoided, also tools producing a permanent and too strong pressure. Therefore in every case clear direction should be given to the occupational therapist about the details of motor, sensory and trophic disturbances and supervision of this therapy by the doctor is of great importance. A close co-operation between doctor and occupational therapist is also of immense value for the mental state of the patients, particularly in overcoming the various mental disabilities due to injury and hospitalization. The latter particularly, should not be underestimated in war time. In every case the occupational therapist should record all his arrangements and observations, for these can be of great help to the doctor, giving information about the patient's behaviour with regard to the restoration of working capacity.

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JOINT DISCUSSION No. 2

Section of Therapeutics & Pharmacology
with Section of Medicine

Chairman—R. D. LAWRENCE, M.D., F.R.C.P.
(President of the Section of Therapeutics and Pharmacology)

[November 11, 1941]

DISCUSSION ON THE KIDNEY AND HYPERTENSION

Professor Arthur Ellis: The problem of hypertension and the kidney has interested clinicians since the time of Bright. When the occurrence of hypertension was first appreciated its dependence on disease of the kidney was generally assumed. Doubt as to the invariability of this association was first aroused by the remarkable observations of Mahomed (1874) of Guy's Hospital, who described patients with hypertension without albumin in the urine. There followed the clinical observations of Allbutt (1895) and of Huchard (1899), who described cases of long-standing hypertension without clinical evidence of renal disease. There thus arose the conception of an essential or primary hypertension to be distinguished from the secondary hypertension due to chronic renal disease. Recently the experiments of Goldblatt *et al.* (1934) who produced sustained hypertension in dogs by means of screw clips on the renal arteries, have led some students of Bright's disease to return to the old belief that all hypertension is renal in origin. The proponents of this argument usually suggest renal arteriosclerosis, with resulting diminished blood-flow through the kidney, as the responsible mechanism in essential hypertension. There are strong arguments against a too hasty acceptance of this thesis. It is, of course, quite possible that essential hypertension is due to some extrarenal factor—endocrine or other—which causes a functional diminution of blood-flow through the kidney, but the evidence at present available suggests that renal arteriosclerosis is the result not the cause of the hypertension. For we see: (1) that broadly speaking the longer the duration of the hypertension the greater the degree of renal arteriosclerosis (Turnbull, 1915); (2) that in malignant hypertension the arteriolar lesions in the kidney are minimal in the early stages and severe in the later stages (Ellis, 1938); (3) that if hypertension is produced in a rat by clamping one renal artery, arteriolar lesions will appear in the unclamped kidney due to the hypertension but will be absent in the clamped kidney which is protected from the hypertension by the clamp on its renal artery (Wilson and Byrom, 1939).

We must conclude that the evidence that essential hypertension is due to renal vascular lesions is still insufficient. Until further evidence is available it seems to me desirable to retain the conception, which we owe to Allbutt, of essential hypertension in contrast to the hypertension secondary to renal disease.

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Indications (1) With the exceptions mentioned below, all cases of varicosity of the saphenous vein and its tributaries are suitable for the use of 'Ethamolin.'

(2) When the Trendelenburg sign is positive, a combination of 'Ethamolin' treatment with ligation of the saphenous vein is usually necessary, but injection alone has sometimes produced a favourable result.

(3) When ligation has been performed, 'Ethamolin' may be used to occlude any isolated varices or to treat any fresh varices which may appear.

(4) If varicose ulcers are present, healing—after a healthy granulating surface has been obtained—may be accelerated by injecting 'Ethamolin' into the veins draining the ulcerated area. Both treatments can then be carried out concurrently.

Contra-indications to injection treatment Where there is a history of tendency to thrombosis, or when thrombosis has already taken place in the deep veins of the leg; acute phlebitis; when the patient is bedridden; ulceration (see indication 4); marked oedema (which should be

treated by rest in bed with legs elevated; after some days, injection treatment may be possible); marked cardiac or renal involvement; hyperthyroidism; diabetes; acute or chronic skin disease to the area involved—and pregnancy.

Technique The needle should be as fine as possible, short-bevelled for small veins and long-bevelled for large, mobile varices.

Empty vein method This is the method of choice in most cases. With the patient standing, mark with a coloured antiseptic solution the course of the vein to be injected. The patient should then lie down. Apply a tourniquet with sufficient pressure to distend the varicose vein (a sphygmomanometer armlet may be used). Insert the needle through adjacent healthy skin into the distended vein. Release the tourniquet, permitting the vein to collapse before injecting the solution. Apply light pressure to the puncture for two minutes, then a dressing. The patient may then walk about as usual but not take violent exercise for 24 hours.

Full vein method When veins are small, or the above technique is inconvenient, injections may be made directly into full veins without application of a tourniquet.

Dosage Generally the total amount injected at one sitting should not exceed 6 cc. in divided doses. The individual dose ranges from 0.5 cc. to 2 cc. according to the size of the varix. Further injections may be given at 5-7 day intervals. The usual precaution of giving initially a very small trial dose—say, 0.2 cc.—is recommended because hypersensitivity—which occurs very rarely—may result in general reactions.

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morbid anatomist viewing the end-stage has found difficulties of interpretation, or that the pathological classification of Bright's disease has been traditionally confused.

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Dr. Clifford Wilson: *Histological Evidence on the Relation of Hypertension to Renal Disease.*—In the progress of patients with Bright's disease two of the most striking features seem to be: (1) The similarity of the terminal stages in different types of Bright's disease, a similarity which is often reflected in the histological changes in the kidney; (2) the rapid deterioration which often takes place when the blood-pressure rises to levels of 200 mm.Hg or more. Professor Ellis has outlined the conception of a vicious circle which it appears may help to explain these features, the essential aspects of the vicious circle being the production of hypertension following renal ischæmia on the one hand, and the production of obstructive renal arterial lesions by hypertension on the other.

Volhard and Fahr (1914) established the existence of a form of essential hypertension in which death occurs from uræmia. Histologically the condition is characterized by severe acute vascular lesions in the kidney, so-called acute fibrinoid necrosis of the arterioles, and endarteritis of the smaller arteries. Volhard and Fahr termed this condition "malignant nephrosclerosis" and subsequently it became more generally known as malignant hypertension. The clear separation of this malignant form of essential hypertension is the great contribution of Volhard and Fahr to our knowledge of Bright's disease. The importance of maintaining this separation has not been appreciated and in recent years, particularly in America, the term "malignant hypertension" has been applied to cases of chronic nephritis and chronic pyelonephritis with a severe terminal high blood-pressure and clinical features, such as papilloedema, resembling those of malignant hypertension. Thus the distinction made by Volhard and Fahr is in danger of being confused and the true identity of malignant hypertension again obscured. The term "malignant hypertension" should be confined to the malignant type of essential hypertension, thereby maintaining the contrast with the more common benign (essential) hypertension. The terminal hypertensive phenomena in other forms of Bright's disease can if desired be described as a "malignant termination".

It is perhaps desirable first to describe the arterial lesions of malignant hypertension described by Fahr, i.e. fibrinoid necrosis and endarteritis. These occur in the kidney and other organs:—pancreas, intestine, suprarenal, retina, testis, in that order of frequency.

Fig. 1 shows typical examples of acute fibrinoid necrosis of an afferent arteriole to a glomerulus, and of endarteritis, or cellular intimal thickening, of an interlobular artery. It seems probable that endarteritis results from organization of fibrinoid necrosis in the smaller arteries.

Professor Ellis has presented evidence for the view that in malignant hypertension, renal damage is secondary to the hypertension, being minimal in the early stages and increasing as the disease progresses. Figs. 2, 3 and 4 are low-power views of the kidney from cases of malignant hypertension at different stages. Fig. 2 shows almost complete absence of histological change in the kidney; fig. 3 shows a moderate renal involvement, and fig. 4 a severe degree of renal damage, with focal interstitial fibrosis and tubular dilatation. It is this picture which has led to the confusion between malignant hypertension and chronic nephritis and resulted in the inclusion of both conditions under the heading "chronic interstitial nephritis".

The arterial lesions illustrated in fig. 1 are constantly found in malignant hypertension. They are also found, with the same organ distribution, in other types of Bright's disease

This separation of essential hypertension from the hypertension of chronic nephritis was an important advance in our knowledge of "the kidney and hypertension", but it did not satisfactorily solve the problem because, as many observers began to note, certain cases which appeared to be examples of primary hypertension died in uræmia and showed at post-mortem the histological changes of chronic nephritis. These are the cases which are now generally recognized as malignant hypertension (Volhard and Fahr, 1914; Keith, Wagener and Kernohan, 1928; and Fishberg, 1931). The recognition of malignant hypertension was of primary importance in the solution of our problem, but its significance was for long—and generally indeed is still—not appreciated.

In a previous communication (Ellis, 1938) I pointed out that in the stage of malignant hypertension when symptoms first appear clinical evidence of renal impairment is often lacking or minimal, and that histological examination of the kidneys of patients dying in this stage shows little or no evidence of nephritis, arteriolar necrosis being apparently the earliest histological change. We have now studied 103 examples of malignant hypertension. Albumin was absent from the urine of 10 when first seen. In 26 there was only slight albuminuria and the renal function tests were normal. We have examined the kidneys in 12 cases dying from a non-renal cause such as cerebral or subarachnoid hæmorrhage, cardiac infarction or failure. None of these kidneys showed extensive damage, the renal parenchyma was well preserved; in some the lesions were so scanty that only careful search revealed them. The earliest lesions appeared to be arteriolar necrosis. Fahr was the first to appreciate the significance of these arteriolar lesions, he considered them the cause of the glomerular lesions. He attributed them to various toxins—syphilis, lead, rheumatism and various other unknown agents. Volhard considered that both arteriolar and glomerular lesions were due to vascular spasm. We thought that the glomerular changes were due to the arteriolar lesions and that these were a direct result of the hypertension. The production by Wilson and Byrom, in designed experiments, of these arterial and glomerular lesions would seem definitely to establish their hypertensive origin. Furthermore, the histological picture in the rat with experimental hypertension closely resembles "chronic interstitial nephritis" in man. This conception that hypertension can produce changes in the kidney formerly regarded as chronic nephritis is essential to an understanding of chronic Bright's disease.

A major difficulty in the understanding of Bright's disease is that different renal disorders may, in their late stages, show a very similar clinical picture consisting of hypertension, renal failure, and hypertensive retinitis with papilloedema. Histologically the same difficulty occurs, the kidneys showing a similarity of histological change which has made their differentiation difficult. This end-stage is often designated "chronic interstitial nephritis". Most close observers of Bright's disease have long recognized that "chronic interstitial nephritis" is not an entity but includes a number of different conditions: chronic nephritis, malignant hypertension, chronic pyelonephritis, the late stage of a toxæmia of pregnancy which has not cleared up, the "chronic interstitial nephritis" of lead workers, even the amyloid kidney may terminate with the clinical and histological picture of "chronic interstitial nephritis" superimposed on amyloid disease. All these conditions are associated, when this syndrome develops, with hypertension. We believe that the syndrome and its associated histological picture are the result of this hypertension. This is the explanation for the similar end-stage of such otherwise varying conditions. We envisage the common factor to be renal ischæmia. The progressive course of the condition we attribute to the vicious circle demonstrated by Wilson and Byrom (1941) in rats—renal ischæmia leads to hypertension which in turn gives rise to destructive vascular lesions, these lead to further renal ischæmia and so augmented hypertension—a progressive process.

When the occurrence of these common hypertensive lesions is appreciated the histological differentiation of the underlying conditions is simplified and with the assistance of the clinical history a separation of the various conditions is usually possible. The term chronic interstitial nephritis should therefore be dropped. Chronic hypertensive Bright's disease is composed of a number of entities including chronic nephritis, benign and malignant hypertension, chronic pyelonephritis, the hypertension following toxæmia of pregnancy and various other odd conditions. The separation of these various entities is most easily achieved by careful observation of the natural history of their development. Since hypertension is common to them all it is not surprising that the

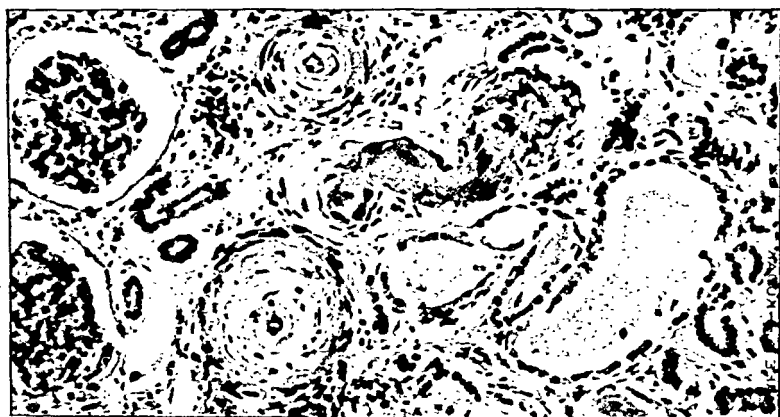


FIG. 1.—Kidney, malignant hypertension. Endarteritis of interlobular artery and fibrinoid necrosis of afferent arteriole. (Hæmatoxylin and eosin. $\times 150$.)

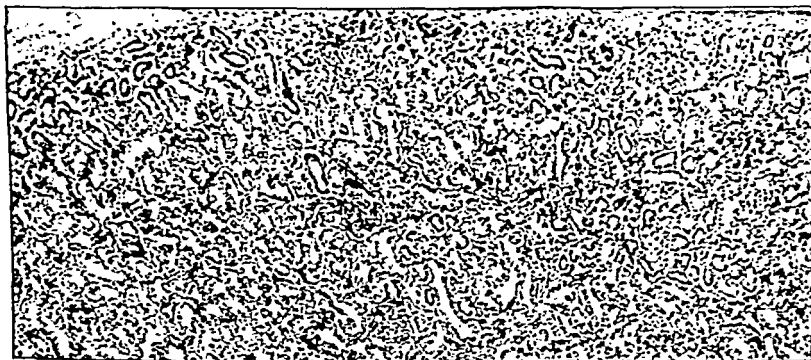


FIG. 2.—Kidney, malignant hypertension. Early stage showing apparently normal kidney. (Hæmatoxylin and eosin. $\times 44$.)

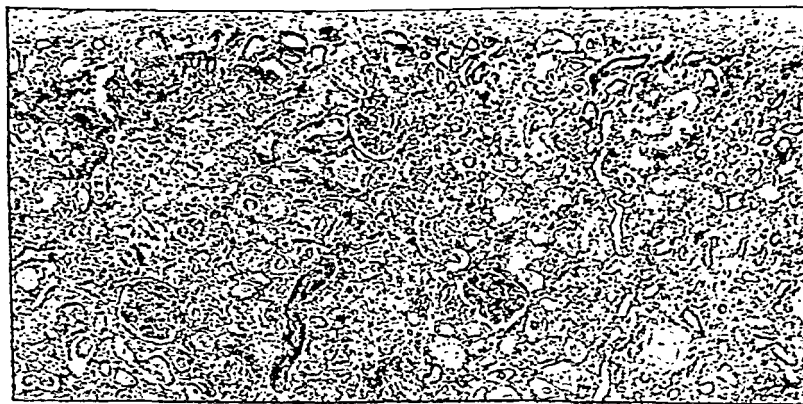


FIG. 3.—Kidney, malignant hypertension. Moderate renal damage. (Hæmatoxylin and eosin. $\times 44$.)

when severe hypertension has been present. This has not only led to difficulty in the histological diagnosis of malignant hypertension but has been the main obstacle to the recognition of this condition as a separate disease by the pathologist. From fig. 2 it will be obvious that in the early stages there is no danger of confusing the histological picture with that of chronic nephritis; on the other hand it may be difficult, and very occasionally it is impossible, to distinguish between the kidney in the advanced stages of malignant hypertension (fig. 4) and in a long-standing case of chronic nephritis in which endarteritis and fibrinoid necrosis of arterioles are present. (By "long-standing" chronic nephritis I mean those cases in which the disease has been present for many years or even decades, and in which the kidney shows the typical focal fibrosis.) The histological differentiation between malignant hypertension in the late stages and long-standing chronic nephritis is based on the following considerations: (1) Renal contraction is usually much more marked in chronic nephritis; (2) the glomeruli are greatly reduced in number in chronic nephritis but only slightly reduced in malignant hypertension; (3) a large number of glomeruli, often the majority, appear normal in malignant hypertension—relatively few in chronic nephritis; (4) the abnormal glomeruli show a greater variety in size and in type of lesion in chronic nephritis; the "type lesion" being a relatively large glomerulus showing increased cellularity, loss of lobulation and more or less extensive capsular adhesions. In malignant hypertension acute glomerular necroses are more common, and form the predominant lesion.

These histological differences between malignant hypertension and chronic nephritis are explained by the different natural history of the two diseases. In malignant hypertension we have a severe initial hypertension, acute vascular lesions dominate the picture, and the course is rapid so that death usually occurs before renal destruction is far advanced. In chronic nephritis severe hypertension is usually a late feature, whilst the renal damage has progressed slowly over many years and the resulting renal contraction is usually severe.

Having discussed the differences between malignant hypertension and chronic nephritis, let us again consider the vicious circle which plays such an important part in both diseases. The evidence which I shall present is derived from the study of experimental hypertension in animals.

Wilson and Pickering (1938) and Goldblatt (1938) noticed that when hypertension was produced in rabbits and dogs respectively, by clamping both renal arteries, these animals often developed hæmorrhages in the gut. Histologically, arterial lesions were found closely resembling those of malignant hypertension. These arterial lesions were not found in the kidneys, whence it was inferred that they were caused by hypertension, from which the kidneys were protected by the clamps on the renal arteries.

About this time, Byrom and I devised a method of determining the blood-pressure in the rat, which made it possible to follow the course of experimental hypertension in this animal (Byrom and Wilson, 1938). Following a technique similar to Pickering's, we were then able to produce hypertension by applying a silver clip to the renal artery and found that in the rat, in contrast to the rabbit and the dog, a permanent hypertension frequently resulted when one renal artery was partly occluded, the other kidney being left intact. It was thus possible to study the effect of experimental hypertension on an intact kidney.

(1) We discovered that when a sudden rise in blood-pressure to a high level, e.g. over 200 mm.Hg followed renal artery compression, acute vascular and glomerular lesions were produced in the *unclamped* kidney within a few days. No such lesions were present in the clamped kidney, which appeared in most cases histologically normal. These vascular and glomerular changes show a close resemblance to those of malignant hypertension in man (Wilson and Byrom, 1939).

It appeared, therefore, that lesions of the renal glomeruli and arterioles which had previously been thought characteristic of nephritis could be caused by hypertension. Since one kidney was intact at the onset, renal failure could not be a causative factor (confirmed by blood-urea determinations) and since the lesions were absent from the clamped kidney, a circulatory toxin or chemical agent appeared to be excluded.

(2) The next step was the finding in rats with sustained hypertension lasting many months, of chronic renal changes closely resembling chronic interstitial nephritis in man (Wilson and Byrom, 1941). These apparently represented the healed or healing stages of the acute lesions but the renal damage was more extensive, and acute and chronic lesions were often present together. These changes also were confined to the unclamped kidney, the clamped kidney appearing histologically normal. The lesions affected glom-

tension or there may be some individual susceptibility in the blood-vessels. A similar failure to develop renal vascular lesions was observed in a number of rats with very severe, sustained hypertension.

(3) We might expect lesions of this extent to cause sufficient ischaemia in the unclamped kidney to give rise to hypertension, thereby providing the second step in the vicious circle. To test this assumption the clamped kidney was removed in some 27 animals. In two-thirds of these the operation was followed by some degree of persistent hypertension. A typical example is shown in the accompanying chart. In this case hypertension persisted for twenty-one weeks after removing the clamped kidney.

It appears, therefore, that the hypertensive lesions in the unclamped kidney were themselves capable of causing a sustained hypertension.

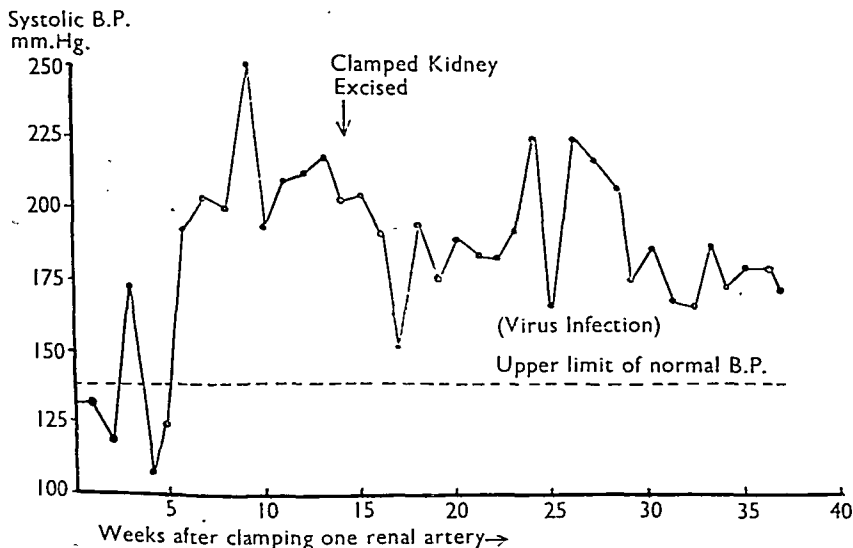


Chart to show persistent hypertension after excision of clamped kidney.

(4) Lastly, to make the vicious circle complete, it follows that these animals in which the clamped kidney has been removed should eventually develop uræmia owing to progressive vascular lesions in the unclamped kidney. Our experiments were cut short by the outbreak of war and we were unable to observe this occurrence. In several animals, however, with sustained hypertension recent acute vascular lesions were found in the kidney and in other organs a considerable time after the clamped kidney was removed: this suggests that the residual hypertension is capable of producing further renal vascular lesions.

In conclusion, if this conception of a vicious circle is substantiated it will explain the rapidly progressive terminal stages both in malignant hypertension in other forms of hypertensive Bright's disease. It is a common observation that in chronic nephritis, once the blood-pressure rises over 200 mm. the patient's condition deteriorates by what may be termed "geometrical" progression. Further it will explain the confusing similarity, both clinical and histological, between the various types of hypertensive Bright's disease in their terminal stages, and finally if, as appears possible from recent work, a reliable method of reducing the blood-pressure becomes available, the existence of this vicious circle will provide a rational basis for attempting to lower the blood-pressure in these conditions.

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cruli, tubules, interstitial tissue and arteries, and were focal in distribution, producing the familiar picture of focal fibrosis and tubular dilatation (fig. 5).

In view of the focal distribution and the close relation of the parenchymal changes to the arterial lesions it seems justifiable to conclude that this picture of chronic interstitial nephritis is a purely vascular process, resulting from high blood-pressure. Why all patients with hypertension fail to develop this picture in the kidney is not clear. Its production may be related to sudden development or to exacerbations of the hyper-



FIG. 4.—Kidney, malignant hypertension. Late stage showing focal fibrosis and tubular dilatation. (Hæmatoxylin and eosin. $\times 44$.)

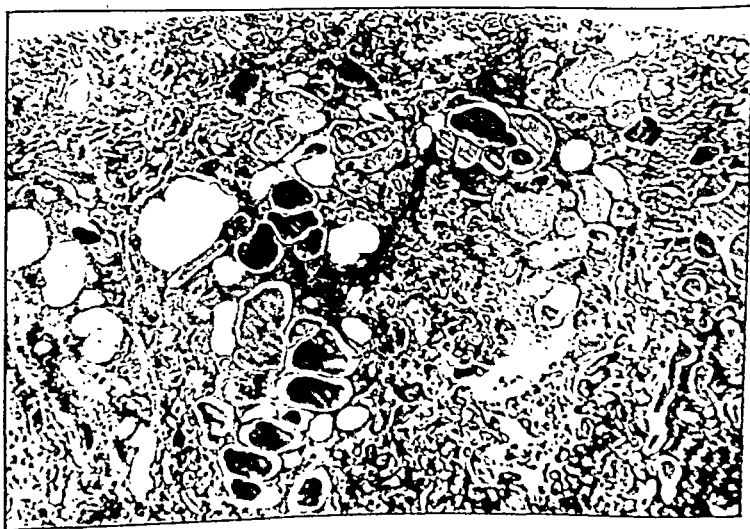


FIG. 5.—Unclamped kidney of rat with chronic hypertension. Showing similar extensive fibrosis and focal tubular dilatation. (Hæmatoxylin and eosin. $\times 39$.)

to be renin in the renal vein of animals with experimental hypertension (Munoz, *et al.*, 1940; Page, 1940). Some evidence has also been presented that hypertensin may be detected in the circulating blood—but this is less convincing. Extensive assays conducted by Prinzmetal, Kelsall and myself have yet to be gathered together and analysed with regard to the renin content of the kidney in experimental hypertension.

Attempts are now being made, particularly in North and South America, to detect renin or hypertensin in blood obtained from the renal vein and the general circulation in patients with hypertension. The technique for detecting these substances is complicated and there is no wide adoption of a method of biological assay. Without further comment I would refer readers to a recent paper by Page in which he claims that hypertensin is detectable in plasma obtained from the peripheral circulation in patients suffering from benign, malignant, and nephritic hypertension.

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Lieutenant-Colonel Robert Platt: I shall confine my remarks to one of the most interesting corollaries to the recent work just described, namely the question of unilateral renal disease and hypertension.

It has been known for some time that in the human subject unilateral disease of the kidney may be associated with hypertension of the malignant type. For instance, Schwartz in 1924 described three fatal cases of malignant hypertension in children. Only in one was a post-mortem examination made, and a condition of hypoplasia of the right kidney was found. This kidney weighed 6 g. and the left kidney 150 g. The left or larger kidney is said to have shown the histological changes of glomerulo-tubular nephritis, but in view of our present knowledge of the subject one wonders if those changes were really secondary to the hypertension induced by a small ischaemic kidney on the opposite side.

Ask-Upmark (1929) recorded six cases of malignant hypertension in children with unilateral renal hypoplasia, and mentions several others described in the literature. Fishberg (1939) in his monograph states that hypoplasia of one kidney is not a rare finding in a young patient who has succumbed to malignant hypertension.

Saphir and Ballinger (1940) record three cases of unilateral stenosis of the renal artery with hypertension, in two of which post-mortem revealed changes characteristic of malignant hypertension in the opposite kidney. Thus has Nature imitated the experiments of Wilson and Byrom (1939).

The commonest pathogeny of hypertension and unilateral renal disease is either hypoplasia of one kidney or unilateral pyelonephritis. Weiss and Parker (1939) record several examples of the latter type but the two conditions often co-exist, as the hypoplastic kidney appears to be especially liable to infection.

In actual fact the underlying cause of the hyperplasia may be lack of blood supply owing to a congenitally small renal artery, in which case the hypoplastic kidney will of necessity be also an ischaemic one and therefore a potential cause of hypertension.

Since the work of Goldblatt *et al.* (1934), clinicians interested in renal disease have been on the look-out for cases of hypertension with evidence of unilateral pathology in the hope of restoring the blood-pressure to normal by removal of the faulty kidney. The first successful case was that of Butler (1937). This was a boy of 7 who developed a unilateral pyelonephritis following the removal of a renal calculus. The blood-pressure, which was normal at first, gradually rose to 160/105 and at the age of 8 nephrectomy was performed. The blood-pressure fell to 100/70 and the urinary infection cleared up.

Professor G. W. Pickering: In the commonest form of hypertension, benign or essential hypertension, it is still uncertain whether the renal lesions are the cause or the result of raised arterial pressure. The problem of whether or not any hypertension is renal in origin will probably be solved when it is possible to demonstrate the mechanism of renal hypertension in man.

The only form of experimental hypertension closely resembling persistent hypertension in man is that produced by reducing the blood-flow to the kidney, for example by renal artery constriction (Goldblatt *et al.*, 1934). Such animals, in addition to hypertension, show cardiac hypertrophy and a normal skin circulation (Pickering and Prinzmetal, 1938*b*). If the constriction is mild, renal function may remain relatively unaffected, if severe, renal failure occurs and in such instances retinitis and arteriolar necroses in the organs are found (Goldblatt, 1938; Wilson and Pickering, 1938). Experimental renal hypertension is chemical and not nervous in origin for it can be produced in the sympathectomized animal; and by constricting the artery supplying the kidney separated of all its nervous connexions through being transplanted in the neck. Recently it has been shown by Fasciolo, Houssay and Taquini (1938) that the arterial pressure of a nephrectomized dog under chloralose anaesthesia is raised by transplanting into its neck an ischaemic kidney from a hypertensive dog, but not by transplanting a kidney from a normal dog.

The idea that hypertension might arise from the release of a pressor substance from the kidney is very old. As long ago as 1898 Tigerstedt and Bergman described the pressor action of renal extracts and the chief properties of the active substance which they named renin. Despite its discovery by a famous physiologist but four years after that of adrenaline, only Bingle and Strauss (1909) had published any clear confirmation of the existence of renin before 1936, when work began in a number of places which has subsequently demonstrated unequivocally the existence and chief properties of renin. That this substance had proved so elusive was due to its instability, to the difficulty of separating it from depressor agents, and finally to the inhibitory effect of most anaesthetics and particularly of urethane on its action (Pickering and Prinzmetal, 1938*a*). Renin has only been extracted from the cortex of the kidney and exists in large quantities in rabbit, pig, dog and cat, but only small amounts can be obtained from human kidney, and Prinzmetal and I failed to find it in sheep, horse, cow, or whale. It is a protein separating with the globulin and particularly the pseudoglobulin fraction; it is destroyed at 60° C., by strong alkali and acid; and by alcohol and acetone at room temperature though not at -10° C.; in these solvents it is insoluble. Injected intravenously into the rabbit renin produces a rather slow rise of arterial pressure which lasts for half an hour or more in the unanaesthetized animal. In the unanaesthetized rabbit it produces fairly constant responses provided that sufficient time elapses between injections for the arterial pressure to return to normal, and this fact provides a basis for biological assay. But in the anaesthetized animal repeated injections at short intervals have long been known to produce decreasing responses. Renin produces vasoconstriction in the rabbit's ear perfused with blood but not in the rabbit's ear perfused with Ringer. These curious facts have been explained by the discovery, made independently by Page and Helmer (1940*a* and *b*) and Braun-Menendez, Fasciolo, Leloir and Munoz (1940), that renin only produces vasoconstriction in the presence of a constituent of normal blood which is gradually used up when renin acts: this substance is found in the pseudoglobulin fraction of the plasma proteins. Renin reacts with this *in vitro* and produces a third substance, angiotonin or hypertensin, which on intravenous injection gives a quick rise of arterial pressure. Hypertensin is thermostable, diffusible, and soluble in alcohol and is thus totally unlike renin chemically, from which indeed it is thought to arise by the enzyme-like action of renin on a plasma precursor. Finally there is evidence that hypertensin is itself destroyed by another enzyme which is present in many tissues and in normal kidney. Thus the present view is that renin, reacting with the precursor in plasma, liberates hypertensin which is subsequently destroyed by another enzyme system. We may ask, then, whether experimental renal hypertension is due to the release of renin from the ischaemic kidney? The present evidence suggests that it is. Thus Miss Hill and I (1939) were able to produce prolonged hypertension in the anaesthetized rabbit by prolonged infusion of renin and to show that this hypertension resembled experimental renal hypertension in the normality of the ear circulation, and in the similar duration of the hypertension following the cessation of the infusion in the one case and removal of the kidney in the other. More direct evidence is the detection of abnormal quantities of what appears

the operation. She was then apparently well and had had no headache or vomiting since the operation. Mr. Nutt reported that the retinal œdema was already improved. The blood non-protein nitrogen was 37.2 mg. per 100 c.c. and the urine still contained a trace of albumin.

Macro- and microscopic examination of the specimen showed evidence of chronic healed pyelonephritis in a congenitally hypoplastic kidney. There are apparently four very small arteries running into the hilum of the kidney, with no sign of a main arterial trunk. There is gross hypertrophy of the ureter. There are patches of atrophy with fibrosis of glomeruli, colloid casts in the tubules and endarteritis obliterans. Other (small) areas are apparently healthy. There is little evidence of the effects of hypertension, as one would expect. These may, of course, be present in the opposite kidney which we have not been able to examine.

She was seen again on July 30, two months after the operation. She was then apparently quite well and had had no return of symptoms. Her complexion and colouring, which had previously been sallow and unhealthy, were strikingly altered. The facial palsy had improved. The blood-pressure was 140/90. The retinal picture was much improved although there was still a little œdema of the upper and inner part of the right disc.

When again examined on October 15, four and a half months after operation, she was in excellent health. Her parents say she gets up in the morning singing instead of vomiting. She never refuses breakfast, and has no headache. Blood-pressure now 125/70. The sounds being much clearer and the diastolic pressure definite. The facial palsy is much improved. Urine Sp. Gr. 1018. No albumin. No pus or casts. The retina presents an extraordinary picture. The vessels appear quite normal, there is no swelling of the discs or œdema of the retina; yet there are extensive shining-white exudates, obviously not of recent origin, the only remaining indication of her extremely serious condition a few months ago which, but for the operation, would I am sure already have proved fatal.

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Dr. Geoffrey Evans showed in the form of a graph blood-pressure readings taken regularly over a period of nineteen years of a case of benign hypertension. The patient first consulted Dr. Evans in 1922 on account of his having been told that he was suffering from high blood-pressure, and was unfit to occupy an important position in his company on that account. His blood-pressure at that time was 190/116. During the succeeding nineteen years the man had occupied the position of managing director of a large company for twelve years and afterwards became chairman of the company. During these years he had four hypertensive attacks, two of which were subacute and were controlled by conservative treatment, two attacks were acute and were controlled by intensive treatment. The highest blood-pressure readings in the acute attacks were 220/116 in one attack and 220/130 in the other. The last readings were 176/90, and these figures were the lowest for the past eight years.

Dr. Evans emphasized the importance of knowing the natural history of benign hypertension, and described four types of the condition. This case illustrated the type in which there is persistent hypertension with short phases of exacerbation and long periods of remission. He suggested that a phase of hypertension is the clinical counterpart of a

Twenty months after operation the blood-pressure was 115/75 and the child remained well. In 1938, Leadbetter and Burkland recorded the case of a boy of 5½ with an ectopic hypoplastic kidney. He had been known to have hypertension for five years, that is since he was 6 months old. The blood-pressure fell after nephrectomy to 125/72 and later came down to 96/70. On examination of the kidney after its removal, a plug of smooth muscle tissue was found partially occluding the main renal artery.

Barney and Suby (1939) record a successful nephrectomy in a girl of 10. For two years she had had pyuria, and intravenous pyelography showed a functionless right kidney. The blood-pressure, which was 190/120, came down to 110/70 within twenty-four hours of the operation. The affected kidney was atrophied, probably as the result of healed pyelonephritis, and showed thickening of its arterioles. Twenty-one months after operation the blood-pressure was 92/60.

Striking though they are, none of these three cases showed the changes of malignant hypertension, and if the results are to be successful it is obviously important to perform the nephrectomy if possible before the onset of the malignant phase, because as Wilson and Byrom (1941) have shown, the vicious circle will sooner or later appear, with secondary changes in the opposite kidney.

Nevertheless, some successful cases have now been recorded in which nephrectomy was performed at apparently a late stage of the disease.

Boyd and Lewis (1938) describe the case of a man of 31 with headache and blurred vision; blood-pressure 200/120, retinal œdema and macular exudate. In this case a large infarct was discovered in the right kidney and after nephrectomy the blood-pressure returned to normal and the eye symptoms cleared up. The blood-pressure is known to have remained normal for six months.

Schroeder and Fish (1940) and Nesbit and Ratliff in the same year record a number of adult cases of hypertension in whom nephrectomy had been performed for various reasons. Most of these cases were not completely successful though improvement in the blood-pressure was often observed. Sometimes the results were only temporary. In many of these cases there was evidence of disease in the opposite kidney and so their suitability for operation may be questioned. Patch *et al.* (1940) have recently reported another case and collected altogether 23 in the literature.

My own case is a little girl of 8 who came to see me on May 22, 1941. The complaint was of severe headache and vomiting which had been troubling her for over six months. Recently the vomiting had been persistent every morning on waking. The previous history was important. A year before, she was investigated rather thoroughly for recurrent pyrexia. A *Bacillus coli* infection of the urine was found and an intravenous pyelogram had been performed which showed a functionless left kidney. The right kidney appeared to be normal. This was confirmed by cystoscopy and injection of indigo-carmin. Retrograde pyelography had failed to outline the left renal pelvis, the iodide solution flowed straight back along the ureteric catheter. There was no note of the blood-pressure at that time. My examination, which was just twelve months after this investigation, revealed a blood-pressure of 200/120. The heart was somewhat enlarged. Neither kidney was palpable. Both fundi showed papilloœdema with advanced retinitis. The blood non-protein nitrogen was 52.3 mg. per 100 c.c. The urine—an early morning specimen—showed specific gravity 1010. Trace of albumin. No pus or casts.

An interesting feature of the case was that four times during the last few months she had had attacks of facial palsy, and when I saw her there was an incomplete paralysis of lower motor neurone type on both sides.

On May 29 she was admitted to a nursing home. The blood-pressure was then 220/120. My colleague, Mr. Nutt, reported that there were 2 dioptries of swelling in each disc, with small hæmorrhages, a good deal of retinal œdema, and some powdery white exudate around the maculæ.

The following morning Mr. Lytle did a cystoscopic examination which showed that indigo-carmin was still excreted by the right kidney within five minutes, though none at all came from the left. An anæsthetic was therefore administered and an incision made to expose the left kidney. A small hypoplastic kidney about 1½ in. in length was removed. At the end of the operation the blood-pressure was 190/120. That was at 1 p.m. At 5 p.m. the pressure was 160/128 although there was no shock or vomiting from the operation. The next day it was 155/95. The diastolic pressures throughout were difficult to take as the sounds were very faint. The blood-pressure remained at about this level—sometimes as low as 145 until the child was discharged ten days after

Section of Surgery

President—E. ROCK CARLING, F.R.C.S.

[December 3, 1941]

DISCUSSION ON THE EFFECTS ON THE KIDNEY OF TRAUMA TO PARTS OTHER THAN THE URINARY TRACT, INCLUDING CRUSH SYNDROME

Dr. E. G. L. Bywaters: Impairment of urinary excretory function after severe injuries is not uncommon, but little is known about how it is produced. It seems likely that several different mechanisms are involved; with more complete investigation of these cases, it may be possible to sort them into several classes, each group depending on the particular type of functional lesion involved and on the particular aetiological agent (Table I).

TABLE I.—IMPAIRMENT OF RENAL FUNCTION AFTER TRAUMA TO DISTAL PARTS.

A. *Functional azotæmia* due to decreased glomerular filtration or increase in formation of waste products.

Examples: Oligæmic shock, including hæmorrhage.
Dehydration or electrolyte loss.
Pericardial tamponade and vascular stasis.

B. *Organic changes.*

Examples: Crushing injury, burns, some types of obstetric shock, traumatic liver necrosis, intravascular hæmolysis, tubular or ureteric blockage due to sulphapyridine.

After severe traumata with hæmorrhage and laceration, including those inflicted by the surgeon, oliguria is usually noted; this may sometimes be accompanied by a faint trace of albumin and hyaline casts in the urine and, more rarely, by a small rise in blood-urea concentration. This type I have classified as functional impairment of excretion, because renal tubular function itself is seldom, if ever, permanently affected; complete recovery ensues. A similar type of functional impairment, that following gastro-intestinal hæmorrhage, first observed in 1934 by Sanguinetti, has received much critical attention recently; there appear to be at least four factors involved.

(a) Increased protein breakdown in the small gut gives rise to increased urea formation in the liver [14]: this increase depends directly on the rate of amino-acid absorption and on adequate de-amination by the liver.

(b) External hæmorrhage itself, according to Black, working on dogs [3], does produce a very slight impairment of excretory function, as shown by the delayed excretion of ingested urea. This is probably due to diminished blood flow resulting from decreased

pathological state which he called arteriolosclerotic disease. He suggested that if such a phase of active disease is not controlled it may develop into so-called malignant hypertension. In such a case as that recorded he suggested that the development of recurrent hypertension was comparable to an attack of congestive heart failure in chronic myocardial disease.

Dr. Wilfrid Oakley: During an investigation on the sedimentation rate in renal disease, it was found that normal readings were obtained in cases of benign essential hypertension, but that in malignant, the sedimentation rate was definitely increased. In one case in a child in which a benign stage developed into the malignant, a change in sedimentation rate was noted; but it could not be concluded that this preceded such clinical evidence as hypertensive retinopathy.

Professor R. V. Christie: Professor Pickering has mentioned that extracts of kidney have been used in the treatment of hypertension, and I should like to ask him his opinion of the therapeutic results obtained. Both Page [1] and Grollman [2] and their co-workers have claimed results which are quite spectacular.

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Professor Pickering (in reply to Professor Christie): I am not entirely convinced by the evidence submitted and prefer to reserve judgment on this extremely important question.

Professor H. P. Himsworth: Whenever the results of experimental investigations appear to have a clinical bearing there will not be wanting zealous clinicians who will claim to have discovered the clinical counterparts of these experiments. The modern views on the role of chronic unilateral pyelonephritis in producing hypertension are perhaps another example of such uncritical zeal. The view is now widely publicized that chronic urinary infections often produce infection of one kidney, that the inflammatory process in this kidney affects the renal vessels so as to diminish the blood supply to that organ, and that the resulting renal ischaemia leads to hypertension. Proof of the correctness of this hypothesis appears to be lacking and the theses published in its support seem, in many instances, to be little more than circular arguments. Examination of material from cases of unilateral chronic pyelonephritis reveals that in a large number of these cases there exists some gross abnormality of the renal vessels which might well in itself lead to renal ischaemia. Lieut.-Colonel Platt's patient is a case in point; the affected kidney was supplied by four renal arteries, and it is quite usual in cases of so-called "hypertension due to unilateral chronic pyelonephritis" to find hypoplasia of the renal artery, or partial obstruction of the renal artery by an atheromatous plaque or some similar lesion which will mechanically reduce the renal blood-flow. An explanation, at least as tenable as that now fashionable, is that the primary defect in these cases is a vascular lesion causing renal ischaemia which in turn causes, not only hypertension but a diminished rate of urine formation in the affected kidney; that the consequent relative stasis in urine flow favours the occurrence of urinary infection in the renal tract of the affected side, and that as a result unilateral chronic pyelonephritis is established. This view has, incidentally, the merit of providing a reasonable explanation of the fact that the pyelonephritis in these cases is unilateral.

Dr. Paul Wood reported the result of an experiment in which a pint of blood was removed from the renal vein of a man of 21 with malignant hypertension, and transfused into an anaemic boy aged 8. No rise of blood-pressure in the boy could be detected. Details of this observation will be published in due course.

Dr. Horace Evans: Malignant hypertension as we understand it is a disease which manifests itself suddenly, usually without any indication of pre-existing high-blood-pressure, and indeed may occur in relatively young people. Although a malignant type of termination is sometimes seen after years of benign hypertension, this is rare, and it would therefore surprise me very much if this occurred in Dr. Geoffrey Evans' patient.

In regard to the association between the contracted kidney and hypertension, I have always found it difficult to understand the state of affairs as it exists in most cases of renal rickets. These patients have hypoplastic fibrotic kidneys, but there is really no hypertension.

by fig. 1, a case which I saw through the kindness of Mr. Riddell and Dr. Dow at St. George's Hospital. The patient showed no pallor or sweating or fall of blood-pressure, yet, as calculated by venous hæmoglobin and hæmatocrit readings, she had lost over a litre of plasma into the tissues, without either external hæmorrhage or any more œdema than a few square inches over one hip; she passed myohæmoglobin in the urine with casts. Probably many cases go through this first phase of shock without its being noticed. That this was a case of "crush syndrome" was established by the finding of myohæmoglobin and creatine in the urine, sure indices of muscle damage, and by the presence of albumin and casts, with a depressed urinary urea concentration and azotæmia, pointing to parenchymatous renal damage.

The two most interesting biochemical features in our own cases were those relating to renal function and those relating to tissue breakdown. For the former, comparison of

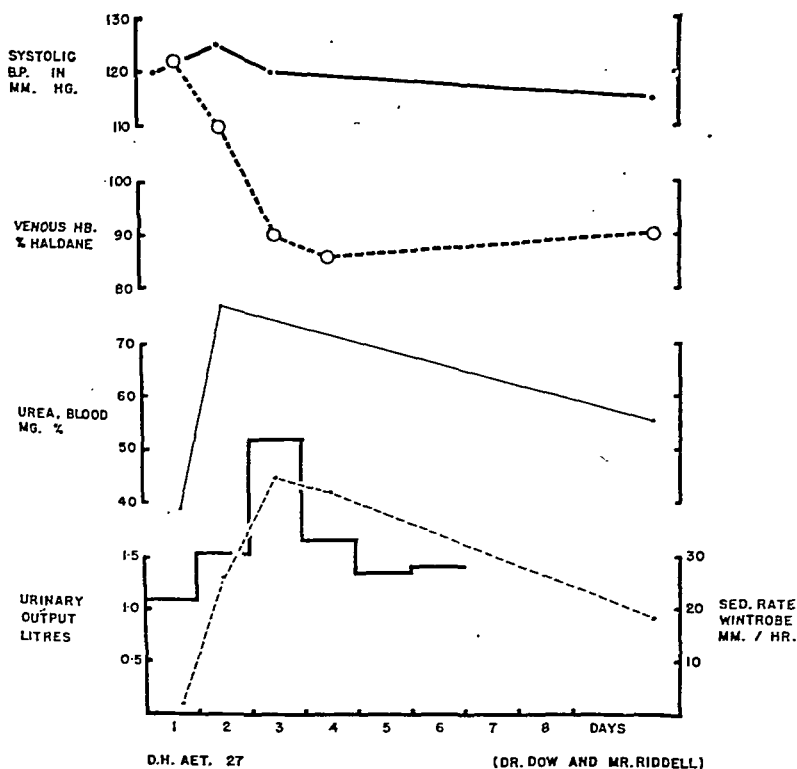


FIG. 1.—Mild case of crushing injury.

Note hæmoconcentration on entry, with normal blood-pressure, and rise in urine output, blood urea and sedimentation rate. She showed myohæmoglobinuria and creatinuria with a urinary pH of 4.6, responding rapidly to alkali therapy.

blood and urinary figures showed that tubular function was impaired, there being failure to concentrate urea and failure to reabsorb chloride: in addition, there was leakage of glomerular filtrate back into the blood-stream. There may be biochemical evidence of recovery of renal function, even though death occurs at the usual time (seventh day) and in the usual way [2]. Secondly, there was creatinuria and myohæmoglobinuria [6], which must have come from damaged muscle: this tissue autolysis probably explains also the

blood volume. In addition, dehydration and electrolyte loss due to vomiting, which is not an infrequent clinical complication, will further reduce excretory power owing, perhaps, to a similar decreased renal blood flow. Davis [10] has shown a non-protein nitrogen rise of about 100% following anhydræmia in dogs produced by subcutaneous injection of 25% saline.

(c) Decreased renal reserve may be a factor particularly in the elderly: Wallace and Schafer [21] have shown that, after venesection of a litre of blood, the blood urea rises only in patients with hypertension and renal sclerosis.

(d) The oliguria accompanying the systolic pressures below that necessary for glomerular filtration to occur may produce a more lasting effect on the kidneys, as is recorded by Stafford [20], after a stab wound of the heart, where pericardial tamponade developed and the blood pressure remained at or below 60 mm.Hg for fifteen hours; there was oliguria for eight days, with blood casts and albumin, a blood urea rising to 220, hypertension and an excretory function, measured by serial phenol-red excretion tests, temporarily decreased to zero.

These four factors, increased protein breakdown, decreased renal blood flow, decreased filtration pressure, and decreased renal reserve, may all possibly play some part in actual clinical cases, both of gastro-intestinal hæmorrhage and also following trauma. Generally, the renal lesion is functional and not structural.

As elsewhere, however, there is no sharp dividing line between functional and organic change except on paper (Table I). Structural changes are sometimes seen following burns (although even in severe cases it sometimes happens that neither functional nor organic change is found), in some types of obstetric shock (Young and McMichael, 1941 [22]) after septic abortions (Bratton, 1941 [4]) and in the so-called "hepato-renal syndrome" described after traumatic liver necrosis (Furtwaengler, 1927 [12]). I am not discussing either these or the anuria sometimes seen in cases of severe trauma due to sulphapyridine blockage or mismatched transfusion, because I want chiefly to deal with "crushing injury". The Medical Research Council has collected details now of over 70 cases; it is on this group that the following remarks are based.

It was not until after the first cases had been published that we discovered that this "new" syndrome, occurring in patients pinned for hours under the debris of bombed houses, was not new at all. It was recognized in Germany during the 1914-1918 war [11], mentioned in official books on military surgery [15], and worked up pathologically [19]; indeed, it had only been missed by a week in 1909 following the Messina earthquake: there, the German relief hospital arrived on the fourteenth day and observed only the muscle and skin gangrene in recovering cases [9]. Why it has not been remarked on since 1923, or at all in Anglo-Saxon literature, would, perhaps, be understandable if it occurred only with bombs and in earthquakes, but it does occur in civil practice following mining accidents, as McClelland has recently recorded from Canada [17], and also, as will be detailed later, following traumatic injuries to main vessels.

CLINICAL ACCOUNT

A typical severe case, having a history of being buried for several hours with masonry resting across a limb, will be shocked on admission or soon after, with a fall in blood-pressure following a preliminary period of apparent well-being. During this initial phase, hæmoconcentration occurs (and compensatory vasoconstriction) from loss of plasma into the injured part, which becomes swollen and hard. There is also loss of sensation and power and the skin shows whealing and, later, blisters: arterial pulsation distally may be impaired. After restoration of blood volume by transfusion of blood or serum, the patient recovers, but it is noticed that the urine is blood-stained and contains albumin and pigmented granular casts. In the next few days the urinary output decreases and becomes clearer: the blood-pressure rises to a high level: there is a progressive increase of blood urea, phosphate and potassium, and a decrease in alkali reserve. The patient becomes alternately drowsy and apprehensive: death occurs suddenly about the seventh day, and is occasionally preceded by abnormalities of cardiac action similar to those produced experimentally by potassium intoxication.

An increasing number of mild and surviving cases are being recognized and published, often as showing the beneficial results of treatment. Most of these show no clinical (hypotensive) "shock", but this is not to say they are not shocked, as may be illustrated

(2) In other cases, the whole muscle may be necrotic, irrespective of skin pressure areas. These are muscles contained within tight fascial sheaths, and it seems probable that rise of intrafascial pressure may be responsible for the ischæmic necrosis seen.

(3) Finally, in occasional cases, isolated fibres show necrosis. This is shown in our own Case I [5], where we believe the ischæmia was in large degree vasospastic in origin, due to perivascular hæmatoma. Cohen [8], has recently emphasized this possibility. This type of case may be characterized by lack of progressive swelling of the leg, and by little in the way of hæmoconcentration. The same isolated fibre necrosis is seen, too, in cases with arterial rupture and limb ischæmia, without any prolonged crush, such as may occur after an automobile accident (fig. 3). These cases, of which seven have been recently recognized, as well as the two originally recorded by Husfeldt and Bjerring [13], closely resemble cases of crushing injury clinically, biochemically and histologically, as will be brought out later in the discussion, but (except for a forty-five-minute crush case of Mr. Belsey and Dr. Miles, where we were able to identify myohæmoglobin) it is still uncertain, in this type of case, which has often received massive blood transfusions, whether the

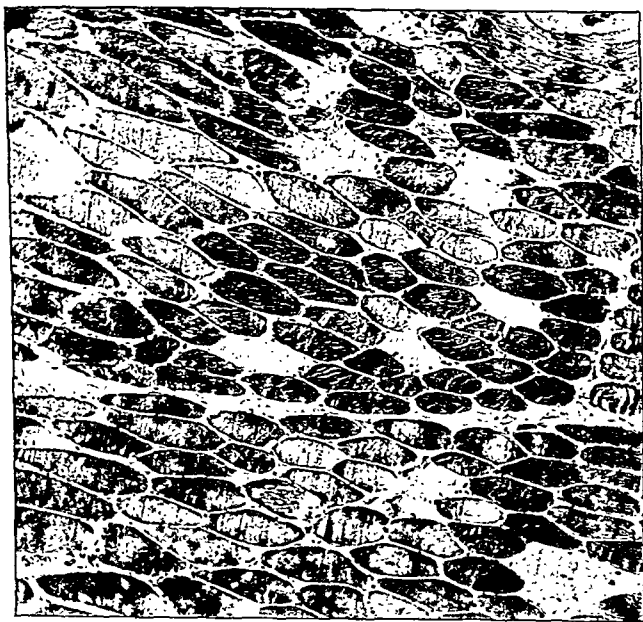


FIG. 3.—Muscle from ischæmic limb below site of rupture of popliteal artery (Mr. Graham's case) showing loss of staining ability and early vacuolation ($\times 80$, hæmatoxylin and eosin).

pigment excreted in the urine is hæmoglobin or myohæmoglobin; it is often obviously a very important point. Such cases must occur frequently, and it is to be hoped that with the knowledge gained from the four cases described later, they will be more frequently recognized and investigated (see figs. 6, 7, and 8).

In any given case of crushing injury, then, there may be ischæmic muscle necrosis due to any or all of these processes.

PATHOGENESIS

Although muscle ischæmia and necrosis are thought to play important parts in the genesis of the renal lesion, it is still uncertain how they act. Obstruction of tubules by

acidosis and the very low urinary pH almost invariably seen as soon as circulation to the damaged part is re-established. Whether the phosphate and potassium increases can in part be thus explained, is still uncertain.

PATHOLOGICAL FEATURES

If death does not occur before the usual time from other causes, the kidneys are swollen and wet. The most obvious feature microscopically is the presence of pigment casts in the collecting tubules similar to those seen in the urine. More important, we think [7], is the severe tubular damage seen in the distal convoluted tubules and Henle's loop of the boundary zone—often with complete necrosis—and discharge of hyaline casts into the interstitial tissue and surrounding cellular reaction (fig. 2). This corresponds with the biochemical indication of tubular dysfunction and leakage.

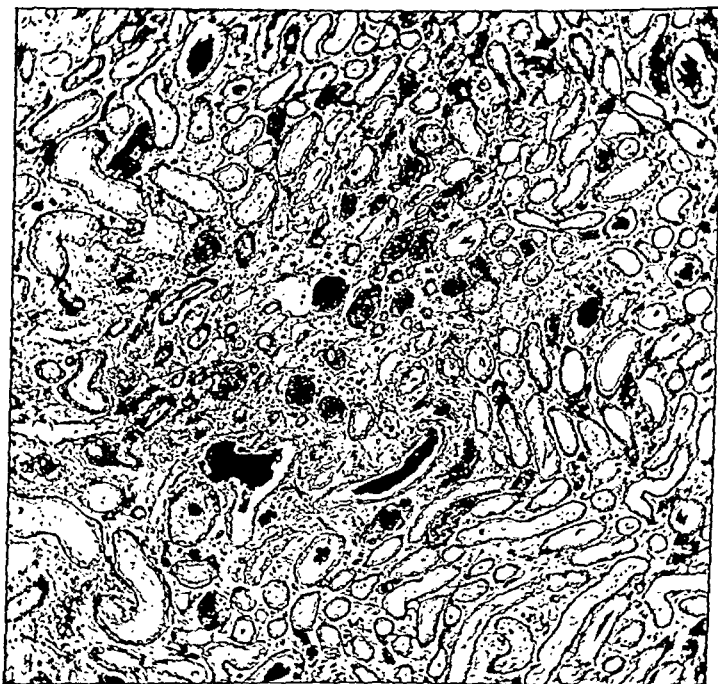


FIG. 2.—Boundary zone from D.W. ($\times 80$, Macgregor's Azan Carmine) showing focal area of necrosis, and on its margin, the extrusion of hyaline cast into oedematous interstitial tissue from partially necrotic tubule.

This histological change is not entirely specific, since we have seen a very similar picture in kidneys from some wasting conditions, from a case of paralytic myohæmoglobinuria and from mismatched transfusion. However, the pigment excreted in the urine in these cases is not hæmoglobin but myohæmoglobin, whose renal clearance is twenty-five times that of hæmoglobin, thus accounting for the lack of plasma colour and the absence of jaundice. It may be distinguished in the Hartridge reversion spectroscope by the different position of the bands.

Myohæmoglobin, along with other autolytic products, comes from muscle, which shows necrosis, loss of staining ability, and reactive changes such as cellular increase, oedema and calcification. There are three main types of muscle lesion:

(1) Part only of the muscle is necrotic, and there is a sharp boundary line between living and dead, corresponding exactly with the pressure marks seen on the skin. This is the commonest type of change, and, we think, is due to direct pressure ischæmia.

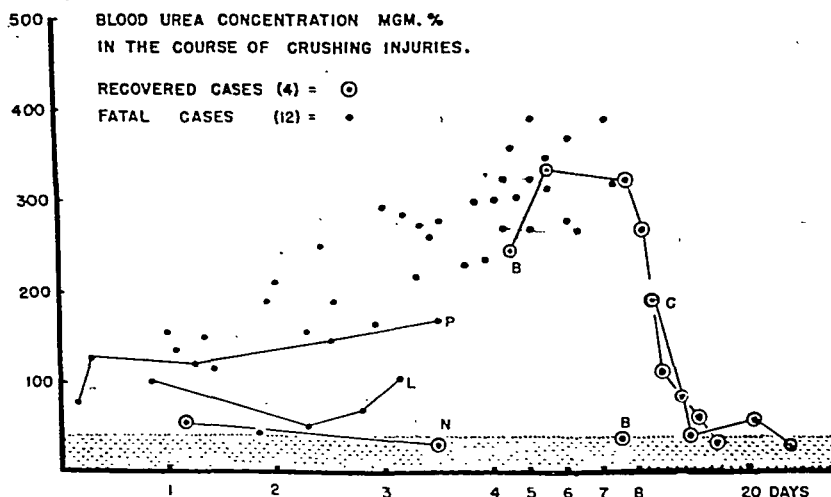


FIG. 4.—Blood-urea concentration in crushing injury. Composite chart, using data previously recorded (*Brit. M. J.*, 1941 (i), 427, and (ii), 475, *London Hospital Gazette*, June 1941, 44, 126) as well as hitherto unpublished cases through the kindness of Dr. Warner and Dr. Horn.

Dr. Pochin's figures (P) are of non-protein nitrogen (mg. %).

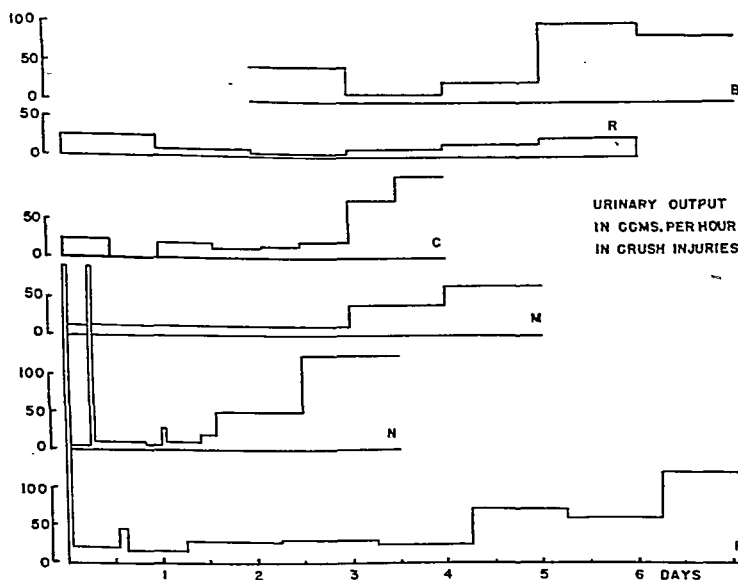


FIG. 5.—Urinary output in crush injury. 5 recovered cases, N., M., C., B., and P. (data respectively supplied by Mr. Neligan and Professor Ellis, Mr. Naunton Morgan, Professor Christie, Mr. Blackburn and Dr. Kay and Dr. Pochin) and one fatal case, Mr. Romani's (R).

casts containing myohæmoglobin derivatives certainly occurs, especially in acid urine as Baker and Dodds [1] pointed out for hæmoglobin, but there may be in addition another factor concerned in the formation of the casts, either of a toxic nature producing catarrh of the lining epithelium, or some such factors as decreased urine flow due to shock, or endocrine disturbances, to cite only two possibilities. Furthermore, obstruction by casts must be widespread to be effective, and what urine is secreted would be of normal composition. It seems, therefore, to us that another mechanism besides obstruction must be involved, perhaps a toxic process producing epithelial necrosis, perhaps tubular ischæmia due to the rise in intrarenal pressure consequent on obstruction, perhaps something else. Professor Shaw Dunn has previously drawn attention to the similarity of the necrosis produced experimentally by phosphate (which is increased in the blood in these cases). Experimental work in progress on dogs by Professor Winton and his associates, and on rabbits at the Postgraduate Medical School indicates that there is at least one other factor necessary besides tubular blockage. Thus the pathogenesis of this condition is still as much a matter of speculation as the similar problem of renal failure following mismatched transfusion.

PROGNOSIS

The critical period is towards the end of the first week; most cases lasting to the eighth or ninth day will recover. The surviving cases (which amount to about one-third of the total collected) have been, in general, less severely injured than the fatal cases. The severity of the injury must be judged by the amount of tissue rendered necrotic, and hence, when the circulation is re-established, by the amount of hæmoconcentration produced by loss of plasma into the damaged area. The severity of the renal lesion must be judged by the rise in blood urea and blood-pressure, by the urinary output, and by tubular function as measured by the percentage concentration of urinary urea compared with that in the blood. A good prognosis may be given with small injuries, and a relatively good one with injuries so severe that there is no re-establishment of the circulation to the part. A good prognosis may be given with a falling blood-urea level or, since there is a progressive rise in most fatal cases, a blood-urea figure below the expected level for the time after injury. To illustrate this, in fig. 4 I have charted blood-urea levels from 16 patients (data sent to the Medical Research Council through the kindness of the physicians and surgeons concerned). The fatal cases show a straight line curve rising steadily to death on or about the seventh day. The surviving cases (circles) may follow this curve and then fall, or they may fail to rise as much and fall earlier. The fall is associated with a diuresis, as is shown in fig. 5 (where five recovering cases are contrasted with the case recorded in the original publication [2] which died). At the same time the hypertension disappears. There is as yet no absolute proof that prognosis as regards renal failure depends on the treatment administered, since not only is prognosis at the beginning of treatment uncertain, which invalidates many of the "success" claims, but treatment is seldom adequate or begun early enough, and this invalidates most of the conclusions regarding failure. However, it appears probable that renal damage is produced as soon as absorption of autolytic products from the limb occurs, that is along with the re-establishment of the circulation. This is the critical time: unless the kidneys are adequately protected, perhaps by diuretics such as fluids by mouth or vein and sodium sulphate, perhaps by alkali, it seems possible that measures directed towards increasing the circulation to a necrotic limb, may do more harm than good. Casualties rescued from collapsed houses have usually been first treated for shock, then the circulation to the injured limb has been re-established and the kidneys have not been considered until the fourth day, but—if instead of this procedure, these cases were first of all treated for renal failure, then a good alkaline diuresis established as soon and as rapidly as possible, and finally the shock and local lesion treated—I think the mortality figures would improve. Prophylactic fluid intake during a raid is, of course, an individual affair, but one of Dr. Grant's cases who recovered had had two pints of beer just before burial.

In conclusion, I want to re-emphasize that we know very little as yet about the syndrome; further accurate and detailed observations relating to renal function are urgently needed both in this type of injury (particularly of that period immediately before and after release from the debris) and also in various other types of trauma.

able by asphyxia, and resulting in an increase in subfascial tension which may be complicated by a recurrence of the vascular spasm.

We do not yet know: (1) How long a period of ischaemia or pressure is necessary to produce the changes in muscle that are interpreted as necrosis. The average period of burial or crushing has been about eight hours, but we have recently seen a case in which the patient was trapped for only forty-five minutes at most; a reflex vascular spasm may have been active for a much longer period following the patient's release; (2) whether toxin production continues after restoration of limb circulation, and whether it is influenced by the metabolic activity of the tissues; (3) at which stage the renal damage occurs, whether it is an early irreversible change, or whether progressive damage takes place during the period of survival of the patient; (4) if muscle necrosis is the sole cause of the renal damage.

The search for collateral evidence that might throw light on the pathology of the crushed limb, and point the direction in which the therapeutic solution lies, has been without success.

In Volkmann's contracture we have a condition of acute muscle necrosis, in which, according to Griffiths, there is evidence that the ischaemia is due to vascular spasm. Whether the resulting changes in muscle are comparable to those occurring in crushed limbs remains to be demonstrated, but as yet we have been unable to find any mention of renal impairment or the changes in clinical condition that accompany the latter injury. Granted that the volume of muscle involved in the typical Volkmann complicating injuries to the elbow is small, it is conceivable that minor degrees of renal impairment may have failed to attract attention.

A survey of the case records of patients with limb fractures admitted to a general hospital during the last ten years, has again elicited no reference to complications at all comparable to those under discussion.

Cohen has recently reviewed the literature on acute traumatic arterial spasm and stresses its frequency, but again the incidence of ischaemic muscle necrosis seems never to have aroused any suspicion of renal damage.

The work of Andrews on experimental uraemia has excited comment during the discussion of the crush syndrome, but with little justification. Andrews ligated the limbs of dogs for varying periods sufficiently tightly to produce oedema of the limb. Following release of constriction in animals which were receiving intravenous hypertonic saline, Andrews observed an immediate fall in the output of urine, a lowered alkali reserve and ultimately a clinical condition suggestive of uraemia, terminating in death. However, he could demonstrate only slight changes in the convoluted tubules, not extensive enough to cause blocking, and concluded that the kidneys were not responsible for the suppression of urine.

Allen ligated limbs of rats and rabbits with rubber tourniquets to stop all circulation, and observed that release after five hours was invariably followed by death of the animal from a condition he describes as "secondary shock" but does not define. The condition was temporarily relieved by transfusion with blood from normal or equally shocked animals. Death could be prevented by amputation of the limb within ninety minutes of release of the tourniquet, by the early intravenous administration of saline from the time of release, or by the refrigeration of the ischaemic limb at temperatures of 1° or 2° C. during the period of constriction. This last observation may be significant and relevant to the present discussion.

Before attempting to assess the influence of surgical treatment on the course of recorded cases of the crush syndrome it is essential that we establish certain definite criteria of diagnosis, prognosis and clinical response, and unless these criteria are conscientiously observed, no conclusions can justifiably be drawn. The problem is rendered much more difficult by the fact that several forms of treatment have been used concurrently, and as no strict attention has been paid to the chronological sequence of therapy and response, it has been impossible to determine their relationship.

In future there must be more control of and attention to the time factor. We now know that spontaneous recovery can occur, and difficulty arises from the fact that treatment, to be of any avail, will probably have to be started very early, before it becomes obvious from the rapidly diminishing urine output that the case falls into that group that inevitably dies between the sixth and ninth days with complete anuria.

If every case is adequately investigated and reported it may eventually be possible to

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Mr. Ronald Belsey: Only by detailed observation, recorded at the bedside, and not from memory when the excitement has evaporated, shall we obtain the vital pathological data without which there can be no rational approach to the problem of treatment. When several hundred cases of this condition have been fully investigated, clinically and biochemically, then will be the time to get dogmatic about treatment. For the present let our attention be directed to the problem of deciding what the surgeon is trying to do, rather than how he is going to do it.

In the crushed limb the following changes have been observed: First, necrosis of muscle, patchy in distribution and rarely corresponding to areas of damaged overlying skin such as would suggest that the necrosis is due to direct pressure. The muscle is pale and mottled. Hæmorrhage into the muscle or tissue planes is not constant. There is œdema of the limb and an increase of tension in the tissues within the fascial compartments of the limb. During life there is frequently evidence of impaired peripheral circulation and sometimes absent pulsation, but at autopsy there is rarely evidence of thrombosis or gross damage to the main arteries of the limb, although in a few instances rupture of a major vessel has been found. Thrombosis and mural necrosis have been demonstrated in some of the smaller muscular arterioles, and in the veins. Intense arterial spasm would appear to be the common cause of the ischæmia and has been demonstrated at operation. Clinically, peripheral arterial pulsation is sometimes absent when the case is first seen; in other cases an initially weak pulse becomes progressively obliterated with increasing œdema of the limb during the first day or two. But in at least two recorded cases the pulse has been normal, and in one was always stronger than in the uninjured limb.

Loss of nerve function has been recorded in the majority of cases, and has commonly involved all the nerves of the limb, a distribution suggesting that the failure of conduction is due to ischæmia and asphyxia rather than direct pressure. In the cases that have survived the recovery of nerve function has been steadily progressive over a period of six to twelve weeks, and usually complete.

Simple fractures may be present, but evidence of gross trauma to the limb is remarkable by its absence. The overlying skin seldom shows more than slight grazing initially, but with the increasing œdema may show bleb formation in places where there has never been any evidence of direct bruising of the skin.

So much for the facts already observed, on the basis of which we assume that there occurs in the crushed limb ischæmic necrosis of muscle, due probably to traumatic arterial spasm, possibly to direct pressure also; that with restoration of the blood supply to the limb there occurs a flooding of the circulation with a hypothetical toxin, and an outpouring of fluid into the tissues of the limb from vessels rendered abnormally perme-

appear to be any indication for surgical treatment; it would have taken a bold man to amputate the left leg through the thigh within Allen's experimental time limit of one and a half hours following release.

CASE II.—A girl, aged 18, had both legs crushed for nine hours. Shock was not severe but intense oliguria was present throughout, the maximum measure obtained being 7 oz. on the fourth day. No intravenous fluid therapy was given and for the first five days this patient received the rest and quiet, the lack of interference, that have been advocated so strongly by the non-interventionists. Clinically the patient resembled a case of terminal uræmia. The swelling of both legs was intense, peripheral pulsation was diminished but present, till the fifth day, when the pulse was hardly perceptible in the right leg. Under general anaesthesia, the right leg was decompressed from the middle of the thigh to the ankle. Oedema fluid poured from every tissue plane and the subfascial tension was very high. The femoral artery was exposed in Hunter's canal and found to be in a state of intense spasm, about 3 mm. in diameter and without visible pulsation. There was extensive muscle necrosis and contusion. The patient died suddenly with complete anuria on the next day. Oligæmia and dehydration probably contributed to the death of this patient. Unfortunately a full biochemical investigation was not possible as the hospital had just been bombed again. Amputation was out of the question in this case, and the extent of the injuries were such that no treatment would probably have been of any avail.

CASE III.—A man, aged 36, was pinned for eight hours with his right arm above his head. On admission there was localized swelling of the right arm in the region of the biceps muscle, slight swelling of the left forearm, and bilateral incomplete median and ulnar nerve lesions. The pulse was normal at both wrists. The urine showed the typical changes: blood, albumin and pigment casts. Next day the whole of the right arm was tensely swollen, the pulse had disappeared, the blood urea was 52 mg.%, but there was no oliguria. Under general anaesthesia the right arm was decompressed by slitting the fascia from the upper third of the arm to the middle of the forearm. The subfascial tension was greatly increased, all layers were oedematous and the biceps and brachialis muscles showed the typical necrotic changes. The brachial artery was identified with difficulty, owing to its intense spasm, surprisingly small size, and complete absence of pulsation. The outer coat of the vessel was dissected off for a distance of about 1 in. at a point high in the arm, the wound was left wide open, and the limb elevated on an aeroplane splint. By the time the patient had returned to the ward peripheral pulsation at the wrist was normal. Next day the blood urea rose to 68 mg.% but the urine measure was satisfactory. Albumin and pigment casts were still present. At no time did the general condition of the patient give rise to any anxiety. Secondary suture of the wound was done two weeks later and the patient made an uninterrupted recovery. Nerve function has returned and the only residual deformity is limitation of extension of the right elbow, due to fibrosis of the biceps and brachialis muscles. In this case surgical treatment was directed entirely to the preservation of the limb.

CASE IV.—Was of unusual interest and will delight the hearts of the non-interventionists. A boy, aged 14, was pinned by the shoulders and both legs below the knees for seven hours. He reports that his left arm went numb very quickly. On admission to St. Thomas's Hospital he was not considered to be shocked and was transferred to Botleys Park Hospital on the same day. There was swelling of the left arm, marked in the region of the elbow, patchy anaesthesia of the hand, wrist drop, a diminished pulse at the wrist, slight oedema of both legs below the knees and bilateral foot drop. His urine was muddy brown, contained red cells, methæmoglobin but no albumin. The general condition remained fairly good for the next four days, with a daily urine measure of about 20 oz., but there was progressive oedema of the left arm and right leg. The pulse was never obliterated. On the fifth day the patient was drowsy and began to vomit; urine output dropped to 7 oz. and the blood urea was 120. By the eighth day the general condition was poor, blood-pressure was 150/70, the urine excretion had risen to 72 oz., but the blood urea was 460 mg.% and the urea clearance 9%. From then on the patient began to recover, but as the oedema in the left arm subsided, so the muscles began to contract, flexors more than extensors, and a Volk-mann type of contracture appeared imminent. By intensive physiotherapy and the use of splints at night the contractures were controlled. Four months after the injury, urea clearance was 108%, blood urea 17 mg.% but the urine still contained occasional non-pigmented hyalo-granular casts; nerve function was completely restored and the only residual disability was slight limitation of extension of the fingers of the left hand and dorsiflexion of the right foot. At no time did the urine contain any albumin.

The main interest in this case lies in the extreme degree of nitrogen retention, the almost complete recovery without specific treatment, and the fact that at one stage amputation of the arm was strongly advocated.

infer from the extent and distribution of the crush lesions whether any given case is likely to survive. The tendency to record only those cases that recover, and suppress the failures, cannot be too strongly deprecated.

There can be no excuse now for failure to foresee the advent of this state in any case of massive tissue trauma before the patient becomes uræmic.

To obtain the prognostic criteria that presage complete failure, only those cases dying with anuria or intense oliguria between the sixth and ninth days should be used. This selection of material will tend to eliminate cases dying of secondary shock with its attendant renal impairment, or later septic complications.

Little in the way of operative treatment has been employed as yet, and probably quite rightly. Primary amputation has been performed in three cases, two of which were fatal. In the first case recorded by Bywaters and Beall, a female aged 17, whose left leg had been crushed for nine hours, amputation through the thigh and apparently above the level of tissue damage was done at the thirty-sixth hour, on account of incipient gangrene of the limb. There was no evidence of damage to other limbs. Death occurred on the sixth day with almost complete suppression of urine.

Decompression of the limb by incision of the deep fascia has been done in seven cases, of which only three have been recorded so far. Three cases died and four survived. Bywaters and Beall recorded two cases: in the first, a male aged 34, with injuries to all four limbs, decompression of the left arm was performed on the second day, on account of the local condition of the limb; the hand was described as "blue, cold and pulseless". Pulsation returned following this operation. In this case the lowest recorded urine output was 20 oz., but was probably higher as incontinence was present. The blood urea rose to 345 mg.% on the seventh day, when the patient died with evidence of staphylococcal pyæmia. No change in the general condition of the patient was observed following operation.

In the next case, a male aged 16, with a crush injury of the left leg, decompression was performed on the third day owing to failure of the peripheral circulation and threatened gangrene; pulsation was restored but the patient died suddenly eight hours after operation. At autopsy the muscles of the leg showed the typical patchy necrosis, but the kidneys were not examined microscopically. This case must be rejected as far as the present survey is concerned on account of the absence of any record of the urine output or blood chemistry.

The best recorded case of survival following limb decompression is that reported from the London Hospital of a girl, aged 18, whose left leg was crushed for fourteen hours. Again we are confronted by the suspicion that this case would probably have survived without operation in view of the fact that the urine output never fell below 37½ oz. and the maximum blood urea recorded was 54 mg.%. The fate of the limb without decompression is another matter. But in the case of a girl, aged 17, buried for thirteen hours, decompression of the left thigh on the third day failed to save the limb, which was eventually amputated below the knee for gangrene. Decapsulation of the kidneys has been performed in one case with established renal failure, but the patient died.

There emerge two distinct problems: first, to save the patient's life, and secondly to save his limb. The latter problem would appear to depend upon our ability to relieve intense spasm of the peripheral arteries, the primary problem is how rapidly this can be achieved. The two aims may be mutually antagonistic in that restoration of the circulation to the crushed limb may lead to a sudden flooding of the body with toxin. The sudden unexplained collapse of Bywaters' fourth case eight hours after limb decompression may be significant in this respect.

I shall now describe five cases that have been admitted to St. Thomas's Hospital, London, and a St. Thomas's Sector Hospital. Full case reports will be published in due course.

CASE I.—A man, aged 20, has already been reported. The region of his left knee was crushed for ten hours. On admission to hospital he rapidly recovered from shock. The local damage to the left leg appeared relatively slight: the œdema was confined to the region of the knee, and pulsation in the dorsalis pedis and posterior tibial arteries was stronger than on the normal side. By the fourth day the urine output had dropped to 3 oz., the blood urea was 165 mg.%, the patient vomiting and drowsy. The urine excretion began to improve, but the blood urea rose to a maximum of 393 mg.% on the eighth day, when the patient died suddenly. Autopsy revealed muscle necrosis in the calf and lower thigh, and typical renal changes. At no time did there

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Dr. J. McMichael: We may safely disregard transfusion and bacterial toxæmia as causes of the renal failure. There is little doubt that breakdown (? autolytic) products from dead or dying muscles are the main factors. Professor Winton and his colleagues at University College have shown that two main effects are produced in the kidney by crushing injuries: (1) poisoning of tubule cells, and (2) tubular blockage, the latter possibly resulting from the precipitation of myohæmoglobin casts in the tubules.

While no final conclusions can be stated regarding therapy, I think we may now approach the problem of treatment on a rational basis. Instead of waiting two or three days to find out whether or not kidney damage is manifest, we must think of the kidney from the outset. It should be impressed on rescue parties that the real danger in people with crushed limbs is that of renal failure. Before the patient is released, therefore, every effort should be made by means of abundant warm drinks to ensure a good urine flow. The advantage of a good diuresis may be: (1) the dilution of chemical poisons within the kidney tubules, and (2) the prevention of precipitation of myohæmoglobin casts.

It is important to realize that the kidneys are only damaged when the circulation is re-established through the crushed limb, i.e. after release. It would be advantageous, therefore, to apply a tourniquet to the proximal part of the limb before the weight is taken off, and this should be kept in position until the patient is in hospital. After this some control should be exercised on the restoration of circulation. The tourniquet may be replaced by a sphygmomanometer cuff which is deflated slowly below the systolic pressure. By this means a sudden flooding of the circulation with toxic products may be prevented. During this phase no attempt should be made to warm the limbs, and the injured limb should, in fact, be kept cold. Cooling with ice packs might have the effect of slowing the circulation and decreasing the rate of tissue autolysis.

Alkalinization of the urine seems to be advantageous, and it is certainly a rational procedure to give alkalis, as the alkali reserve of these patients is often markedly reduced. Sodium bicarbonate and sodium citrate should be given by mouth (30-60 gr., 2-3 hourly), from an early stage. Other diuretics may be tried, but there is little evidence as yet to favour any particular one. Mersalyl has been a failure more often than it has succeeded, and as this drug may be a tubular cell poison, it is safer to avoid it.

It will be impossible to maintain a good diuresis in the presence of circulatory collapse, and shock must therefore be treated by transfusion, usually of serum or plasma because of the frequent hæmoconcentration.

Professor J. Shaw Dunn, restricting his remarks to the renal pathology in cases of crush syndrome, agreed with Dr. Bywaters in finding in the cases which he had examined important damage of renal tubular epithelium in addition to the presence of brown casts in the lumina. The lesion could be described as an acute tubular nephritis, while its accompanying functional disturbances of anuria or oliguria, marked loss of concentrating power, and retention of urea could readily be paralleled in mercurial nephritis of man and in experimental tubular nephritis of animals. In the present cases the lesion showed the remarkable feature that it was restricted to the lower segments of the nephrons, viz. the ascending limbs of Henle's loop and second convoluted tubules, whereas almost all the many and varied substances which, in natural disease or experimentally caused tubular nephritis, affected the upper segment, or first convoluted tubule. Selective damage of the lower nephron segments appeared to have been little known in human disease, and few substances employed experimentally have had this effect. It might be significant, however, that two substances which occurred naturally in the human metabolism, namely uric acid and phosphoric acid, had been shown capable of this effect. The former injected intravenously in the rabbit had been shown to produce not only this renal lesion but also the effects of temporary oliguria, loss of concentrating power and rise of blood urea. It has been suggested that the special localization of effects of these agents is due to acidification of the renal filtrate in the tubular segments concerned, and it

CASE V.—A man, aged 37, was crushed for only forty-five minutes by a falling crane in a munition factory. He sustained multiple compound fractures of the right leg, a fractured pelvis, complete rupture of the membranous urethra, and a fracture dislocation of the left ankle-joint. Shock was severe and when controlled by multiple blood transfusions, the right leg was amputated through the thigh, the wound of the left ankle excised, and a suprapubic cystostomy performed. On the second day the left showed all the characteristic crush changes, including bleb formation of the skin, anæsthesia, and muscle paralysis. The blood-pressure was 170/100, blood urea 209 mg.%, the urine contained pigment casts, albumin, red cells and myohæmoglobin, but the measure never dropped below 18 oz. On the third day vomiting was profuse, the patient was drowsy and the blood-pressure had risen to 200/100, the blood urea to 238 mg.%. Death occurred on the fourth day, from traumatic paralytic ileus with renal failure a contributing factor. At autopsy the muscles of the left calf were necrotic, and the kidneys showed the typical lesions in the tubules.

In this case the interest centres on the short period of forty-five minutes during which compression can have occurred. Any surgical attack on the left leg which showed the crush changes was contra-indicated by the severity of the co-existing lesions.

And so the picture yet remains far too confused to justify any conclusions upon treatment for the local condition of the crushed limb. In none of the published case reports is there evidence that operative surgical treatment has played any part in saving the life of the patient. The case for amputation remains unproved. Ablation of crushed arms is probably never indicated, but in view of the poor prognosis with extensive crushes of the leg, the application of a tourniquet to the limb before, or at the time of release, by a competent doctor attached to the rescue squad, followed by early amputation as soon as shock has been controlled, would appear to be rational. Many limbs might be sacrificed unnecessarily, and until the crush syndrome can be reproduced experimentally, and the fatal renal complications averted by early amputation, such reasoning should be applied to the case of the human being only with extreme caution.

Limb decompression is indicated as a means of saving the limb in those cases where peripheral pulsation has completely disappeared, but apparently has no influence upon the patient's general condition. The use of drugs, of which alcohol in large doses is probably the best, to relieve peripheral arterial spasm, has not been tried in this condition to my knowledge. Splintage and refrigeration of the damaged limb with ice-bags, as a method of reducing the metabolism of the limb during the period of ischæmia, and possibly also of diminishing toxin production, would appear to be rational if commenced early, in view of the experimental work of Allen, and certainly demands a carefully controlled clinical trial.

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Histological examination of the kidneys revealed no sign of antecedent renal disease. The glomeruli were normal. The renal tubules contained many eosinophilic granular casts. These were most numerous in the medulla but a few were seen in the second convoluted tubules in the cortex and in the tubules of the medullary rays. In the tubules containing casts there was some necrosis of epithelial cells and some evidence of epithelial regeneration. The interstitial tissue and blood-vessels were normal.

Comment.—It is true that the possibility of the case being one of ordinary transfusion reaction cannot be excluded as the man received a quantity of stored blood. However, the lack of lumbar pain or rigor at the time of the administration of the blood, and the lack of jaundice subsequently argue against this, as does the fact that Group "O" blood was given. The case, therefore, is believed to be one of crush syndrome.

Mr. W. H. Graham: The following case I encountered recently is of interest in association with the pathogenesis of renal damage in crushing injury.

A man, aged 18, was injured when the lorry in which he was riding came into collision with a concrete structure. He was extricated almost immediately and received in the casualty ward within thirty minutes of the accident. At that time he was found to be shocked (B.P. 80/50), cold, clammy, pulse 120, temperature 97° F. (fig. 6). There

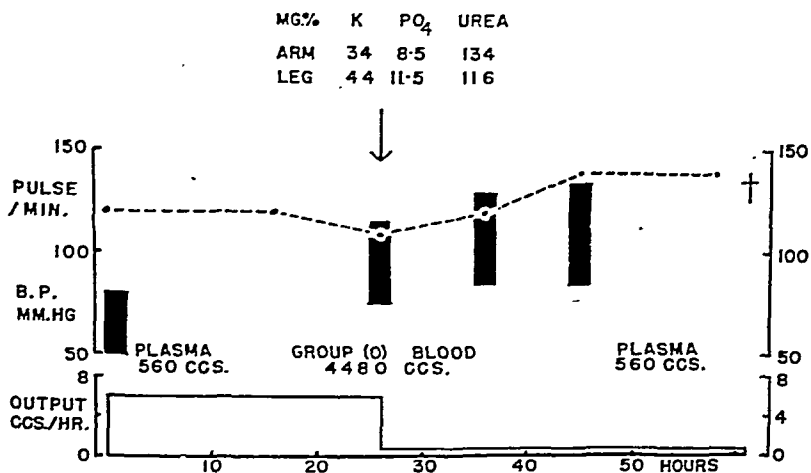


FIG. 6.—Chart showing course in Mr. Graham's case.

[KEY: Pulse—dotted line; blood-pressure—solid rectangle; intravenous fluid given; urinary output. Note oliguria.]

was a compound fracture of the right tibia and femur, and a simple fracture of the upper third of the left tibia in good position. Blood was issuing from the wound in the right thigh and from a deep laceration behind the left knee. Anti-tetanic and anti-gas-gangrene serum was given, the wounds were bandaged and splinted, and plasma (560 c.c.) and Group O blood (1,120 c.c.) given by intravenous drip. Sulphapyridine (2 g.) was given intravenously, followed by 1 g. intramuscularly every four hours.

Sixteen hours after admission the left leg was anæsthetic, cold, blue and pulseless, and the left thigh swollen and tense. Slight oozing only was seen from the wounds. 1,400 c.c. of Group O blood were given. The hæmoglobin was 51% (Haldane), pulse 120. At twenty-six hours, seen in conjunction with Dr. Bywaters, he was pallid, sweating and vomiting: B.P. 115/75, pulse 112. The left leg was warmer and of a better colour. Catheterization produced 150 c.c. of brown smoky urine which contained 60 mg.% albumin, 540 mg.% urea, and 85 mg.% NaCl, and gave a strongly positive benzidine reaction. A brown deposit formed, consisting of amorphous pigment granules: there were no casts, corpuscles or cells. Filtered urine gave a negative benzidine reaction. Venous blood samples were obtained at this time both from the arm and from the left foot (*see* Table I). The serum was not increased in colour.

seemed probable that the localization of lesion in the kidneys of crush syndrome was also contributed to by acidification of the filtrate in addition to the usual factor of concentration. The presence of the brown casts of myohæmoglobin in the tubules was at any rate confirmatory of the view that the renal change was caused by a positive factor derived from muscle and carried to the kidneys in the blood-stream, rather than to loss of something from the blood into the tissues. From observations on experimental tubular nephritis, Professor Shaw Dunn agreed with those speakers who had recommended the early administration of abundant fluids by the mouth, with alkalization, as a possible means of forestalling or lessening the damage to the kidneys.

Mr. V. H. Riddell: I have only two observations to make: (1) If a large number of casualties is admitted to hospital it is very easy to miss the early crush syndrome unless the casualties are systematically examined. Such a patient often looks very well and for this reason may temporarily be passed over in favour of examining someone in a neighbouring bed who appears to be more dangerously ill. The only way to avoid this and other mistakes is to examine each patient in turn, however rapidly, from top to toe, and then, in the case of the crush syndrome the tell-tale wheals or simply a localized erythema of the skin will draw our attention to the lesion. The nurse in charge can then be warned as to what is likely to happen, the first urine can be saved and not thrown away and the investigations begun at the earliest moment.

(2) We all know that the crush syndrome is associated with impairment of renal function, but it is perhaps not so well known that other injuries, including operative shock, may also be associated with depression of the renal function. The impairment of renal function following injury varies from a temporary anuria of twenty-four hours' duration to a permanent and fatal variety.

Attention has been drawn to this oliguria or anuria in different ways. At two of the hospitals I visited for the Medical Research Council, ordinary rooms had been converted into resuscitation wards and special sluices rather than hand-basins fitted for dealing with the large number of urine bottles which it was supposed would be used. After admission of several batches of casualties the ward sister noticed that these utensils were only rarely asked for by air-raid casualties.

At another hospital a physician on the staff had been investigating cortin excretion in the urine of air-raid casualties and found difficulty in obtaining the necessary volume of fluid for his estimations. At St. George's we noticed that male patients who are normally fairly free in their use of the bottle did not call out for it in the same way as they do in the more convalescent wards.

Grant, of the Clinical Research Unit of Guy's, has also pointed out that if the first specimen of urine in the shocked patient is examined, it will often be found to contain red cells and albumin. I have confirmed this, and am at present investigating the first urine specimen passed after all big operations to see how consistent a finding this is.

Captain D. Magner, R.C.A.M.C.: The following case is of interest in that the muscle damage was not due to prolonged compression, but rather to sudden severe bruising suffered in a motor accident.

A British soldier, aged 26, was struck a heavy blow by a lorry. On admission there were signs of damage to the left side of trunk and left thigh, and multiple fractures of the pelvis. The blood-pressure was 100/60.

On the next morning, as he had not voided, he was catheterized and the 8 oz. of urine were found to show traces of albumin and small numbers of red blood cells. The blood-pressure had fallen to 78/58. He was given a transfusion of stored Group O blood. The patient's blood was Group B. No untoward effects were noted during the transfusion. His general condition improved, but he did not void. Daily catheterization yielded only small quantities of urine which never showed hæmoglobin or pigmented casts. The blood urea rose progressively to 328 mg.%, on the day of death—seven days after the accident.

Autopsy revealed subcutaneous extravasation of blood in the left flank, and swelling of the left thigh, multiple fractures of the pelvis, and considerable retroperitoneal hæmorrhage in the pelvic tissues. There was also hæmorrhage beneath the sheath and into the fibres of the left iliopsoas muscle. This muscle, those of the posterior abdominal wall on the left side, and those of the left thigh, were moist and oedematous.

The kidneys appeared normal except for some cortical swelling. The rest of the autopsy was negative except for a patchy bronchopneumonia in lower lobes of both lungs.

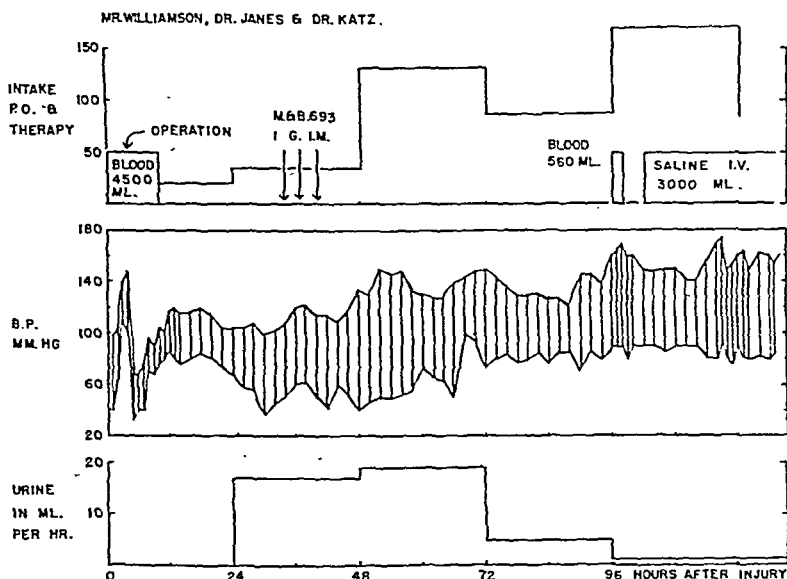


FIG. 7.—Chart showing course in Case I. Note increase of blood-pressure accompanying oliguria.

and viable though pale. There was dry gangrene of the right middle shin, plantar skin and terminal phalanx of the fifth toe, as well as of the skin and terminal phalanges of all the toes on the left side. The kidneys were swollen (7 oz. each) and stripped easily. The cortex was pale; the interlobular arteries were distended with blood. Microscopically the kidneys were similar to those seen in the crush syndrome.

CASE II (fig. 8).—This man was crushed momentarily between two cars, and on admission was found to be very shocked. There was a compound fracture of the left femoral shaft, and a simple fracture of the right pubic ramus. After resuscitation, a Steinmann pin was inserted through the tibia, and after excision of the wound, the limb was put up, with a loose plaster over the wound, on a Thomas' splint in extension. Next day he was conscious, and vomited 45 oz. The left leg and foot were white (except for petechial hæmorrhages and purple areas), insensitive to just above the knee and immobile. The muscles felt unduly hard. 8 oz. of thick brown-red urine were obtained by catheter (Table II). As there was no improvement, the limb was amputated at mid-thigh under gas and oxygen anæsthesia. Some of the muscle appeared to be greyish brick colour and non-contractile. The femoral artery at the upper end of the wound was pulsating, but, although intact throughout the lower part, it was inactive. Both it and the femoral vein were found to contain ante-mortem clot. Later that night a further urine specimen was obtained. Anti-gas serum was given. On the third day he was better; he vomited 8 oz. and excreted 8 oz. of urine, but by the fourth day his condition had deteriorated: the urine output was 4 oz., vomit 12 oz. The dosage of pot. citrate was maintained. Blood-urea was found to be 248 mg. per 100 c.c. He died on the morning of the fourth day, in coma, with a blood-urea of 288 mg. per 100 c.c. and a blood bilirubin within normal limits. Post-mortem, 6 oz. of urine were found in his bladder. There was no obvious sepsis or evidence of muscle necrosis. The femoral vessels were normal. The kidneys were swollen (12 oz. and 6 oz.) with some petechiæ in the pelves, and a pale swollen cortex. Microscopically, they resembled the kidney of crush syndrome.

Comment.—In such cases, if it is thought that renal damage is produced by absorption from the damaged limb, the practical question arises whether early amputation will be more effective: both cases presented evidence of muscle ischæmia, and in each there was a definite vascular lesion to account for it.

TABLE I.—CHANGES IN BLOOD FROM ARM AND FROM BELOW LESION.

(Bioch. Dept., British Postgraduate Medical School.)

| Venous blood : mg. per 100 c.c. | | Arm | Foot |
|---------------------------------|-----|-------|-------|
| Urea | ... | 134 | 116 |
| Plasma phosphate (as P) | ... | 8.5 | 11.5 |
| Plasma chloride (as in NaCl) | ... | 512 | 489 |
| Total plasma protein | ... | 6,500 | 6,500 |
| Potassium | ... | 34 | 44 |
| Hæmoglobin (% Haldane) | ... | 55.9 | 54.5 |

A further 1,400 c.c. of Group O blood were then given. Thirty-six hours after admission, he was of a good colour, dry and warm, B.P. 130/85, Hb. 56%, pulse 120. By forty-five hours he was alternately drowsy and restless, B.P. 135/85. The temperature had risen slightly to 99°, pulse 140. The thigh and leg were now less tense, he was drinking copiously, and vomiting a little. Further Group O blood (560 c.c.) and plasma (560 c.c.) were given. During that night he complained of pain in the loins; no urine was obtained on catheterization. His pulse became weaker and more rapid (140), temperature 101°, and he died sixty-one hours after admission. There was no terminal leakage from the leg wounds.

Post-mortem (seventy-two hours after death) by Dr. Skene Keith, showed slight œdema of the lungs and small petechiæ on the posterior surface of the heart. The kidneys were much swollen (the right kidney weighed 176 g.); the cortex was very pale and bulged considerably on section; the medulla showed many dark streaks. The left femoral vein and artery and hamstrings were completely severed at the top of the popliteal fossa, and a large fibrin clot was found here. The muscle above this level was normal. At the site, it was pale, and below of a somewhat rusty colour. That in the opposite leg appeared normal. The bladder contained 25 c.c. of brown urine giving a very strong benizidine reaction. There were many brown pigmented casts containing, rarely, cuboidal epithelial cell remains.

Microscopically, large pigmented casts were seen filling a large number of tubules. There were foci of œdema and cellular infiltration surrounding the ascending limb of Henle's tubule in the boundary zone, which, becoming necrotic, had liberated into the interstitial tissues hyaline casts. The muscle fibres from below the lesion showed patchy loss of staining ability and occasional vacuolation (see fig. 3). There was no inflammatory reaction and no arteriolar damage, but considerable œdema and, here and there, small hæmorrhages. Above the lesion the muscle appeared normal.

Comment.—Despite the resemblance to crushing injury, there was no prolonged pressure in this case, which appears, therefore, to fall into the group just mentioned by Dr. Bywaters, where muscle is injured by ischæmia, due, not to direct pressure, but to interference with blood supply.

[Thanks are due to the Chief Medical Officer of London County Council for permission to record this case.]

Mr. J. C. F. Lloyd Williamson and Dr. L. R. Janes: We have seen two cases recently where death from renal failure occurred following severe trauma to the limbs with compound fracture and vessel trauma.

CASE I.—A man, aged 24, was pinned for five minutes beneath an overturned Bren gun carrier. On admission one hour afterwards, he was found to be very shocked (fig. 7) and had compound fractures of the lower ends of both femora. After resuscitation, the wound was excised, and it was found that both the popliteal artery and vein were divided in each leg. Both limbs were packed with gauze and sulphanilamide powder, and enclosed in loose plaster. Next day there was some cyanosis of the feet, not improved by cutting the plaster, and some vomiting. Hæmoglobin was 100%, 2 oz. of urine obtained in the morning were clear, but 12 oz. later in the day were red-brown and contained casts but no red cells. On the third day he was restless, some sensation returned below the knees, and 16 oz. of clear urine were passed. Hb. 60%. On the fourth day, he passed 4 oz. of clear urine. Some mottling of the lower legs and feet, with red patches, was seen. On the fifth day, 4 oz. of urine was passed. He was not so well on the sixth day: urine (1 oz.) showed a faint trace of albumin and blood urea was 560 mg.%. He died in the early hours of the seventh day in coma, and was found to have 1 oz. of brown urine in the bladder. Post-mortem showed no jaundice: there was œdema of the legs, and a pallid necrosis of the lower end of the right vastus externus (5 cm. in diameter) was seen. The muscles of the legs and feet were œdematous

subsequent changes in renal function. During and immediately after the operation, a marked drop in blood-pressure was accompanied by polyuria while the urine at this time showed a high urea concentration, indicating normal renal function. Later on the rate of urinary output increased still further and numerous hyaline casts were present in several urine specimens. These we must assume had accumulated during the earlier phase of decreased renal function. After blood and serum transfusions amounting to a total of 5 pints the blood-pressure rose to normal and no further changes were observed. Combined clinical and biochemical studies in such cases will no doubt reveal many examples of such early changes in renal function.

Mr. Guy Blackburn: Sodium sulphate is undoubtedly of value in these cases. A good parallel could be drawn from its use in calculous anuria and after urological operations. Similarly both sodium chloride and sodium bicarbonate intravenously could be useful, the latter especially in raising the alkali reserve. This has been particularly evident in a case recently reported by me in the literature (*Brit. M. J.*, 1941 (ii), 475).

I feel that one should be quite clear as to the distinction between inability to pass water and suppression of urine in dealing with air-raid casualties; Munro's tidal drainage is a useful therapeutic measure with the first of these. Chemotherapy can be dangerous in crush injuries, where deposition of crystals in the tubules can easily enhance the degree of obstruction already present.

[January 7, 1942]

The Value of the Urinary Diastase in the Acute Abdomen

By E. B. C. HUGHES, F.R.C.S.

SUMMARY

SOME time before this war, an investigation was begun in an attempt to clear up the uncertainty surrounding this test. Some of the findings are reported here.

Lewison (1941) has presented results in some 700 cases of various abdominal diseases, and has summarized the literature. His conclusions give a very fair picture of the present beliefs concerning this test. He states in conclusion that: "Deviations from the normal (blood) amylase value occur infrequently in diseases other than pancreatitis. The range of these aberrations is restricted and unlikely to be a source of diagnostic error." Although the number of cases in this investigation is small, certain facts emerge which seem to contradict this statement, and to be of sufficient importance to justify their presentation.

The urine amylase has been estimated owing to the ease with which specimens could be obtained, and also in order to obtain some idea as to the value of a single urine estimation in the diagnosis of acute abdominal conditions.

The method used has been to incubate the urine with starch solution and to estimate the disappearance of the starch by means of iodine. Particular emphasis must be laid on keeping the temperature, pH value and quantities constant in each reading.

A large number of estimations were done, and amongst these were estimations on forty cases of acute peptic ulcer perforation. In these forty cases, four were found in which the urine diastase was over 300 units, and of a level previously thought diagnostic of pancreatitis. In addition three of these cases had areas of fat necrosis, and in these the pancreas seemed normal at operation. The fourth case had no fat necrosis present. At post-mortem the pancreas was found to be normal. In two of the cases the diastase level fell rapidly after operation to normal limits. In two the post-operative levels were not done. All were duodenal perforations in the anterior wall.

Three suggestions are made as to the possible cause of this finding. First, these cases may have had a separate area of pancreatitis. This seemed an unlikely combination, and neither the rapid return of the diastase to normal, nor the negative findings in the one post-mortem would seem to support this. Dr. Hamilton Patterson (1939), however,

- MR. WILLIAMSON AND DR. JANES

E.W., AET. 22 YEARS

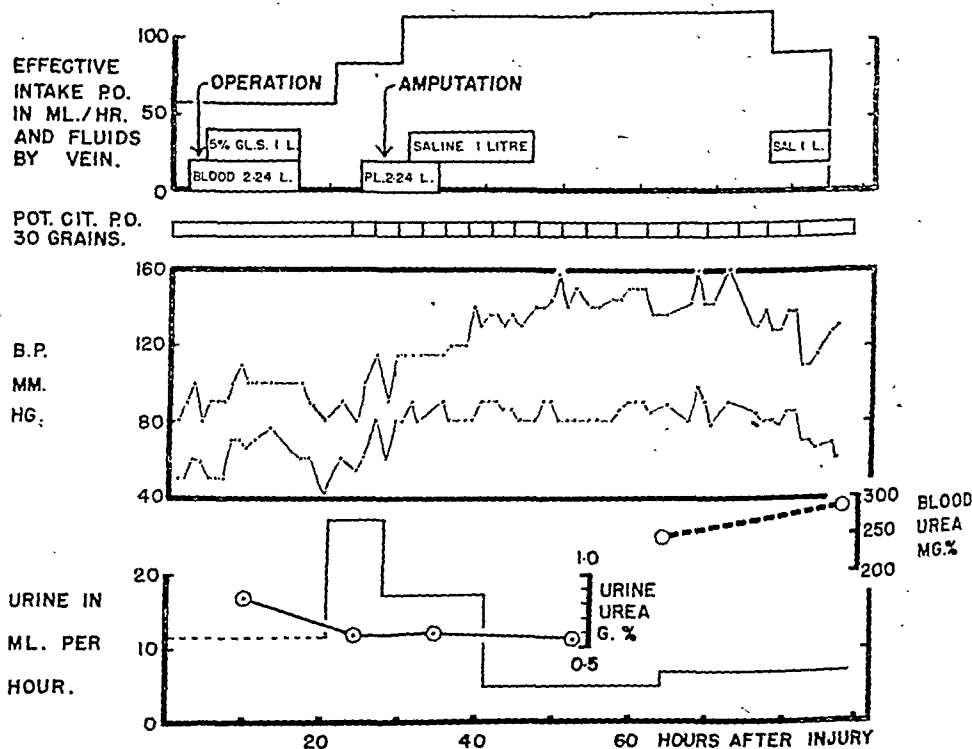


FIG. 8.—Chart showing course in Case II. Note increase of blood-pressure accompanying onset of oliguria together with poor urea concentration in the presence of raised blood urea indicating tubular impairment.

TABLE II.—URINE FINDINGS, CASE II (WILLIAMSON AND JANES).
(Department of Biochemistry, British Postgraduate Medical School.)

| Time | Urea | Alb. mg./100 c.c. | Crea-tine | pH | Supernatant fluid | Deposit | |
|--------------------|------|-------------------|-----------|-----|-------------------|------------|--------------------------------------------|
| | | | | | | Colour | Micros. |
| 2nd day 3.30 p.m. | 830 | 200 | 0 | 4.7 | Brown | Black | Pigment granules and epith. cells |
| 2nd day 11 p.m. | 590 | 160 | 0 | 5.3 | Yellow | Dark brown | Pigment granules; no casts |
| 3rd day 12.20 p.m. | 590 | 180 | 0 | 5.5 | Yellow | Dark brown | Pigment granules, red cells and leucocytes |

Each deposit gave a positive benzidine reaction and showed the presence of acid hæmatin. The supernatant of the first specimen showed faint acid hæmatin bands. No hæmoglobin, methæmoglobin or myohæmoglobin bands were seen.

Mr. Ruscoe Clarke: There can be little doubt that minor degrees of renal disturbance following trauma, are frequently missed on account of their transitory nature. The following is a case of a major crush involving all the tissues of the leg resulting from an automobile accident. Shock and hæmorrhage necessitated the administration of blood and serum. The leg was amputated and a severe scalp wound explored. Recovery was uneventful. The renal changes were detected as a result of careful biochemical studies. Prior to operation marked oliguria accompanied a normal blood-pressure. This was presumably due to incipient shock. Its pre-renal origin is suggested by the

back. The wound was evidently regarded as less severe than many others which were being admitted from Dunkirk and Belgium at that time, as it was not excised until the morning after admission, and he was not given a prophylactic dose of A.T.S. as supplies then available had been expended on apparently more serious cases. About seven days after the wound he developed severe tetanus, beginning with stiffness of the jaw, and died on the eleventh day after injury.

He received much A.T.S. (although the exact amount is not recorded) after the onset of the condition. Further, unlike the majority of soldiers, he had not been actively immunized against tetanus, possibly because he belonged to one of the first groups called up.

CASE IV.—Female, aged 55. Injured in a bombed house at Abbey Wood on 7.9.40. 3,000 units A.T.S. were given on that day at the local hospital. Her injuries were: A small wound of the neck opening the trachea; a small frontal scalp wound and perforations, which became infected, of both ear drums. On the fourteenth day severe generalized tetanus developed. She was successfully treated with 200,000 units A.T.S. and paraldehyde and avertin narcosis.

This patient's daughter, who was injured in the same house, had received a wound of the temporal region—a pebble had passed through the temporal muscle into the brain and later an intracranial abscess formed. The daughter also suffered from severe spasm of masseters and temporal muscles on both sides and may have been another case of tetanus, but I have not included her as the injury to the temporal muscle and the subsequent infection could have caused spasm.

CASE V.—Female, aged 39. She was thrown out of her car near Dorking on 10.8.41, sustaining a simple dislocation of an elbow; cut eyebrow; slight grazes on the knuckles and a wound of one popliteal fossa extending down to the muscles. The head and leg wounds were excised seven hours after their infliction and the latter was loosely sutured after the local application of sulphathiazole. 3,000 units A.T.S. were given at the time of the operation.

On the seventh day a serum rash developed and on the eighth at 9.0 p.m. she complained of shivering and stiffness of the back and shoulders which she had noticed for three hours. She was thereupon examined and found to have stiff jaws and brisk reflexes. At 9.30 p.m. a severe spasm of face, neck and upper spine occurred, and 20,000 units A.T.S. were given intravenously. At the end of this injection she collapsed and her blood-pressure readings were 65/50! Throughout that night her systolic blood-pressure never rose above 80 and during the intervals of consciousness she complained that she could not see—possibly from cerebral anæmia. No further generalized spasms occurred but a risus sardonicus persisted throughout that night and the next day. Her treatment was most difficult because her general condition had been so profoundly affected by the, presumed, anaphylactic shock. Later rectal paraldehyde was given as her condition improved. She recovered after 120,000 units of A.T.S. had been given. She also received sulphathiazole orally as well as locally. The leg wound was never more than very mildly infected and the scalp wound healed by first intention.

The symptoms of tetanus appeared early—eighth day—and the generalized spasm was separated from the initial symptoms of stiffness by only three hours. Such a case with short incubation time and very short period of onset is usually fatal. She recovered after receiving what may be considered a moderate dose of A.T.S. for so severe a case. One wonders whether the severe anaphylactic shock might have had a beneficial effect. At one time, I am informed, severe specific fevers were treated by protein shock.

CASE VI.—Female, aged 45. On 11.5.41 she was buried in the ruins of a bombed house where she lay pinned beside a hot stove. Her injuries were: Extensive superficial burns of one arm and thigh and extensive bruising without open wounds. She was given 2,000 (? 2,600) units A.T.S. on the day of injury at hospital. On the tenth day spasm of the jaw was noticed and despite large doses of A.T.S., avertin and sodium amylal narcosis she died on the twelfth day.

CASE VII.—Male, aged 45. Injured by a bomb explosion in a Surrey town on 16.11.40. His injuries were: Concussion with a scalp wound and a compound fracture of the fibula. 3,000 units of A.T.S. were given at the first-aid post but no surgical treatment was carried out for fifteen hours. The leg wound was clearly severely infected, for much secondary hæmorrhage took place, and his general condition was made worse by a lung infection and probable pulmonary infarct. On the twelfth day a stiff jaw was noticed and next day he died with arched back, locked jaws and spasms, despite much A.T.S.

informs me that he has performed a post-mortem on such a case. Secondly, these cases may have had other peptic ulcers on the posterior wall of the viscus, penetrating the pancreas and giving rise to a local area of pancreatitis. The previous history, the rapid return of the diastase to normal, and their uneventful progress does not seem to favour this possibility. Many such cases, however, have been described, particularly by Probststein, Wheeler and Gray (1939), and by Meyer and Amtman (1936). The third suggestion is, that at the time of perforation the viscus is filled with pancreatic juice and this is discharged from the duodenum, and hence adsorbed from the peritoneum.

It is believed that these facts are of some importance in view of the modern tendency to treat pancreatitis conservatively. It is possible that this diagnostic error may be avoided by estimating the diastase at higher levels, and preferably in the blood. A quick and reliable method has recently been described by Somogyi (1938) in which the estimation may be done in a few minutes without the necessity of incubating large numbers of tubes. This process is now on trial for these estimations in acute abdominal disease.

Estimations have been done on samples from many different hospitals and from many individual surgeons. I am, however, especially grateful to the staffs of University College Hospital, and of the Royal Sussex County Hospital for their help.

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[This investigation is still in progress. The amplified findings will be published later.]

Tetanus in a London Sector in Two Years of War

By E. J. RADLEY SMITH, M.S.

AN effort was made to collect cases of tetanus occurring in Sector 9 by making them notifiable to the Group Officer. In the first two years of war seven, or possibly eight, such cases have been noted and some interesting features emerge from a study of their immunological and other characters.

CASE I.—Female, aged 50. Injured on 19.9.49 when the windows of her house in rural Surrey were blown in by blast while she was looking out. Her injuries were slight: cuts on the face and one hand. The cut hand became very mildly infected but when she was discharged from hospital on the ninth day all wounds were healed and she was quite well. 3,000 units anti-tetanic serum had been given at the first-aid post. She returned to hospital on the fourteenth day after injury complaining of stiffness of the jaw. There was great spasm of the masseters but apart from this the only other sign of tetanus which ever developed was that described by Gordon Holmes—the extension of the knee when the plantar response is elicited. She was successfully treated by 140,000 units of A.T.S. intramuscularly and by paraldehyde medication.

CASE II.—Male, aged 45. An air-raid casualty from South London, injured on 10.9.40. He was treated at the local hospital where 3,000 units A.T.S. were given on the day of injury. It is not known whether subsequent prophylactic doses were given at the two hospitals where he was subsequently treated. He had multiple severe injuries: laceration of left foot; perforating wound of left knee-joint; compound fractures of one metatarsal and phalanges of right foot and compound fracture of right radius. Gas gangrene developed in the right foot. His wounds were clean and healing well in December and his general condition was such that he was allowed home in plaster. On 6.1.41—three and a half months after the receipt of his injuries—he returned complaining of stiff jaws, back and neck. In a few days the condition had extended to clonic spasms of legs and abdomen and these continued up to 19.1.41. He was successfully treated with 660,000 units A.T.S. intravenously, intramuscularly and intrathecally and with sedatives.

CASE III.—A young soldier wounded in France a few days before his admission to hospital on 27.5.40 with a slight soft tissue gunshot wound of the lower part of the

and in all but one of them the three doses could not have been given as the incubation period was too short.

It is clear that one dose of 3,000 units A.T.S. given soon after the wound does not prevent tetanus. Further, it does not guarantee that should tetanus arise it will be local, delayed or mild, as is illustrated by Cases IV, V, VI, VII. This has already been pointed out by Cole (*Lancet*, 1940 (i), 167). In one case the tetanus was localized and in another long delayed, and this might be ascribed to the effect of the prophylactic dose. On the other hand Aldren Turner has recorded a case of chronic tetanus which had never received serum (*Proc. Roy. Soc. Med.*, 1939, 32, 1413 (Sect. Neurol., 99)). Instructions are not likely to reach every doctor who may treat a casualty, and will not be remembered in detail, so that the optimum prophylactic treatment is almost certainly not applied in many cases.

The number of cases of tetanus here recorded is small, but they have been drawn from a large number of casualties, and it does appear that active immunization with tetanus toxoid, as practised by the R.A.M.C. is of great value. As this can be easily, and even advantageously, combined with anti-enteric inoculation (Maclean and Holt, *Lancet*, (ii), 581, and Ramon and Zoelter, *Ann. Inst. Pasteur*, 1927, 41, 83), it would appear that the advantages of such active immunization should be laid before the public. The inquiries which the compilation of this brief report made necessary and other conversations with medical men have made it abundantly clear that many doctors still do not realize that every casualty, whatever the type or severity of wound, should receive a prophylactic dose of A.T.S. at once, and that this should be repeated on two further occasions. This state of affairs after more than two years of war may perhaps strengthen the case for active immunization.

The Immediate Replacement of Avulsed Soft Tissues

By P. H. JAYES, M.B., B.S.

A. C., aged 13, a gipsy boy, was bitten by a horse on December 15, 1941. He sustained a severe wound of the thigh as a result of being seized by the horse and tossed into the air. His mother, who witnessed the accident, discovered that a large piece of skin was missing from just above the knee. A few minutes later, she found the piece of flesh lying on the ground close to the horse. She picked it up, put it back on her son's leg, and bandaged the limb with boracic lint.

The accident occurred at about 12.30 p.m. and the boy was admitted to hospital the same afternoon at 3 o'clock. His general condition was good and soon after admission he was anesthetized. It was found that a piece of skin and subcutaneous tissue had been completely severed from the lower part of the right thigh. The detached portion of skin and fat was lifted off the raw area and washed thoroughly in warm saline. Although it had contracted and at first seemed to be much too small, it actually represented the whole of the missing part. Except for one abrasion the skin appeared to be in perfect condition. It was pinned out on a board and all the subcutaneous fat was carefully dissected off its deep surface. In this way the avulsed tissue was converted into a full-thickness skin graft (Wolfe graft).

The wound measured about 7 by 3½ in. and extended down to the deep fascia. Its edges, which had retracted, were absolutely clean cut, as if with a scalpel. The quadriceps muscle was not damaged, but the upper half of the patella was exposed at the lower end of the wound.

The raw area was washed thoroughly with saline and hæmostasis obtained. Sulphanilamide powder was sprayed on to it and then the "graft" was applied. It fitted perfectly into the wound and it was sewn in position with fine silk sutures. A number of tiny slits were made in the "graft" with the point of a scalpel so as to provide an exit should any blood or serum collect beneath it.

Great attention was paid to the fixation of the "graft". A roll of wool was soaked in flaine and paraffin emulsion and teased out into fine layers. These were applied to the "graft" and built up to form a thick, even pad. Gauze and wool were put on over this and a crepe bandage applied with firm pressure. The knee was immobilized by a plaster back-splint.

Convalescence was uneventful. The temperature rose to 99° F. on several occasions in the first few days, but the leg was comfortable and the dressings were not disturbed.

| Case | Type of tetanus | Physical injuries | Locality of receipt of injury | Presumed incubation period | Prophylactic dose of A.T.S. | Curative dose of A.T.S. | Active immunization | Result |
|------|------------------------|------------------------------------------------------------------------------|-------------------------------|----------------------------|-----------------------------|-------------------------|---------------------|-----------|
| I | Local | Clean cuts from glass | Chipstead, Surrey | 14 days | 3,000 u. | 140,000 | None | Recovered |
| II | Delayed | Compound fractures with gas gangrene | Peckham, S.E. | 3½ months | 3,000 u. | 660,000 | None | Recovered |
| III | Generalized and severe | Slight soft tissue wound of back | France | 7 days (appr.) | Nil | Amount unknown | None | Died |
| IV | Generalized and severe | Small wound of scalp and trachea. Perforated drums | Abbey Wood, S.E. | 14 days | 3,000 u. | 200,000 | None | Recovered |
| V | Generalized and severe | Slight scalp wound and grazes. Simple dislocation. Mildly infected leg wound | Dorking, Surrey | 8 days | 3,000 u. | 120,000 | None | Recovered |
| VI | Generalized and severe | Extensive burns and bruises but no other wounds | Sydenham, S.E. | 10 days | 2,000 u. (? 2,600) | Amount unknown | None | Died |
| VII | Generalized and severe | Comp. fr. of fibula with severe infection | Purley, Surrey | 13 days | 3,000 u. | Amount unknown | None | Died |

COMMENT

Only in Cases II, VII, and possibly V, where the injuries were severe, deep and involving muscle, did conditions arise which are usually considered favourable for the development of tetanus. In Case VI there were no wounds other than superficial burns, and in Case I only a few clean cuts caused by window glass which had all healed in less than nine days.

Seven, or possibly eight, cases have been recorded in Sector 9. All but one have occurred in civilian casualties and the only soldier who contracted tetanus had escaped the customary active immunization. Is this not significant? These cases have occurred in one Sector in two years of war. The figures for this Sector, available at the moment for only one year, show that 4,562 civilian air raid and about 6,500 Service casualties have been treated. Both civilian and Service casualties receive prophylactic A.T.S., but the troops have been actively immunized as well (Reg. Med. Serv. Army, 1938; appendix 10, para 6v) and amongst these so immunized no case of tetanus has been noted, although the most favourable circumstances for its development were surely to be found in the severely wounded from Dunkirk and Belgium who had often been virtually untreated for some days and of whom a considerable number did develop gas gangrene. Several Service casualties, other than battle casualties, e.g. motor-cycle accidents, injuries sustained on manoeuvres with A.F.V., &c.—have contracted gas gangrene often in a severe form.

In all but one case prophylactic serum had been given soon after the wound had been inflicted. As far as is known this initial dose had not been repeated. E.M.S./Gen. 246 not only advocates a single dose of prophylactic A.T.S. of 2,600 units, but actually explains why no further doses should be administered. Although this has been amended by E.M.S./Gen./333, which is still current and which advocates 3 doses each of 3,000 units at weekly intervals, it is to be feared that the earlier instruction still lingers in the mind since not one of the cases is known to have received a second prophylactic dose. Perhaps the type of wound did not suggest, in some of the cases, that tetanus was likely to develop,

and in all but one of them the three doses could not have been given as the incubation period was too short.

It is clear that one dose of 3,000 units A.T.S. given soon after the wound does not prevent tetanus. Further, it does not guarantee that should tetanus arise it will be local, delayed or mild, as is illustrated by Cases IV, V, VI, VII. This has already been pointed out by Cole (*Lancet*, 1940 (i), 167). In one case the tetanus was localized and in another long delayed, and this might be ascribed to the effect of the prophylactic dose. On the other hand Aldren Turner has recorded a case of chronic tetanus which had never received serum (*Proc. Roy. Soc. Med.*, 1939, 32, 1413 (Sect. Neurol., 99)). Instructions are not likely to reach every doctor who may treat a casualty, and will not be remembered in detail, so that the optimum prophylactic treatment is almost certainly not applied in many cases.

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Sulphanilamide 1 grm. four-hourly was given by mouth for four days as a prophylactic measure. On the seventh day the bandage was removed and the upper and lower edges of the "graft" inspected. The appearance was satisfactory and the crepe bandage was reapplied without disturbing the dressings as a whole. On the tenth day all the dressings were removed. About 80% of the "graft" had taken well, the small area of loss being at the site of the original abrasion. This area is now clean and epithelializing rapidly from the edges. Healing should be complete in about six weeks from the date of injury.

COMMENT

In such cases, where the surgeon is presented with the avulsed soft tissues the possibility of immediate replacement should always be considered. The severed tissues must be treated in exactly the same way as any other graft. It would be futile to replace a piece of skin with a large quantity of fat attached; the fat should therefore be removed and the skin alone grafted back into place. The graft must be handled very gently and complete hæmostasis obtained before sewing it into position. Lastly, firm contact between the whole graft and its bed should be maintained during the period of healing.

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★ Extract from "Brit. Med. J.," 1940, i, 631:

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Section of Ophthalmology

President—A. J. BALLANTYNE, M.D.

[November 14, 1941]

Angiomatosis Retinæ. Account of a Case, Including the Histological Results of X-ray Treatment

By Professor A. J. BALLANTYNE, M.D.

(From the Tennent Institute of Ophthalmology, The University and Western Infirmary,
Glasgow)

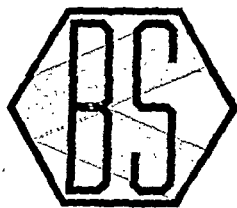
THIS communication deals with a case of angiomatosis of the retina in which the large tortuous pathological vessels ended in an ophthalmoscopically invisible "capillary nævus" at or near the macula, no "massive exudate" having formed at the point of junction of the vessels. Fundus drawings show the appearances before and after X-ray treatment, and the histology of the retina and its vessels is shown in photomicrographs of sections obtained after excision of the eye, which was demanded on account of intra-ocular hæmorrhage followed by acute secondary glaucoma. The fundus appearance in this case closely resembled that of a case described by Mr. A. S. Philps at a meeting of this Section on February 10, 1939, and it was referred to in the discussion on Mr. Philps' case. My fundus drawings were reproduced in the *Proceedings*, 1938-39, 32, 1261.

The patient, a young man, aged 19, was first seen by me at the Tennent Institute on December 4, 1936, having been referred by Dr. J. Pendleton White for an opinion regarding possible treatment.

Defective vision in the right eye was discovered after an attack of influenza about September 1936, and he had recently suffered from epistaxis.

Vision in the right eye: $+ 0.5$ D sph./ $+ 2.5$ D cyl. axis $90^\circ = \frac{6}{18}$; in the left eye: $+ 2$ D cyl. axis $90^\circ = \frac{1}{12}$.

Ophthalmoscopic examination of the right eye (fig. 1) showed great enlargement and tortuosity of the lower temporal artery and vein, which met and formed a twisted loop



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BRITISH SCHERING

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A systematic general investigation was negative. There was not, nor has there ever been, any evidence of intracranial disease nor of hæmangiomata in any of the organs.

On the assumption that a hæmangioma was situated at the confluence of the two large vessels, four radon seeds giving a total of 4,000-5,000 r were placed on the sclera at the situation of the macula. A month later there was no change in the fundus picture, and radiograms showed that the seeds had not remained fixed in their original positions. He was dismissed, the vision of the right eye being $\frac{6}{36}$ (1 letter). Six weeks later he was readmitted, and X-ray treatment was begun on April 20, 1937.

The X-ray applications were made at three points: above the right eyebrow, near the outer canthus, and in the malar region, directed in each case towards the posterior pole of the eye. There were 14 treatments spread over three weeks and the total dose delivered at the treated zone was 4,750 r.

He was dismissed on May 8, 1937, with a fairly well-marked local reaction, which reached its peak, with redness of the lids and conjunctiva, corneal œdema and some turbidity of the aqueous, about six weeks after commencement of the X-ray treatment.

Eleven weeks after the beginning of the X-ray treatment the first ophthalmoscopic signs of a retinal reaction were seen in the shape of a white lateral sheathing of the central loop of the large vessels. Visual acuity in the right eye was now $\frac{6}{60}$. In about a fortnight a constellation of white spots, like those constituting a "macular star" appeared above the macula, and there was a progressive increase in the extent and width of the vessel sheathing, which throughout the whole period of observation of the fundus, was confined to the large abnormal vessels in the lower half of the fundus (fig. 2). The white spots varied from time to time; disappearing almost entirely for a period. At a later date patches of woolly-looking exudate appeared near the disc. Small patches of pigment on a pale background were also seen in the lower central area.

During the succeeding twelve months he suffered a good deal from recurrent neuralgic pain in and around the right eye, and this was relieved by alcohol injection of the trigeminus. Vision deteriorated and it became more difficult to obtain a good view of the fundus owing to œdema of the cornea, folding of Bowman's membrane, K.P., flare in the aqueous, and opacities in the lens. Other by-effects of the X-ray treatment were: loss of eyelashes, depigmentation of the skin of the lids, telangiectasis of the eyelids and conjunctiva bulbi, closure of the lachrymal puncta and a general rigidity of the eyelids.

On April 2, 1939, two years after the X-ray treatment, there was sudden loss of vision in the affected eye, due to a gross intra-ocular hæmorrhage, and the tension rose to 30 mm.Hg. This increased, and the eye became painful, with hyperæmia of the iris, as well as of the conjunctiva.

The eye was excised, and both the local anæsthetic injection and the removal of the eye were impeded by the very rigid state of the eyelids, the conjunctiva and the orbital tissues.

An artificial eye is now worn, but the shrinking of the skin and orbital tissues gives a sunken appearance to the right eye, and the inelastic condition persists.

There has been no pain during the last two years.

After fixation in formalin the eyeball was bisected in an equatorial direction, and the posterior half examined with the corneal microscope (fig. 3). The main inferior temporal vessels were seen to be represented by apparently solid white bands. One or two others of the inferior branches had a similar appearance while others showed a very narrow blood column heavily sheathed in white. The vessels in the upper half of the retina presented mainly a reduction and variability of calibre with a minor degree of sheathing.

To a large extent the central area of the fundus was occupied or covered by hæmorrhages of various types. Some of these were obviously in the anterior layers of the retina, some in the deeper layers, and others of more massive character either subhyaloid or under the internal limiting membrane. At two points there was a large oval mass of greyish colour with some resemblance to a cyst. This was afterwards found to be a collection of serum, separated from a massive hæmorrhage.

in the midst of an ill-defined, dusky, reddish-grey area as big as the disc and embracing the macula. The two large vessels were similar in colour and calibre, and could not be distinguished as artery and vein. The other retinal vessels were also somewhat dilated and tortuous, but arteries and veins were easily distinguished. The disc and neighbouring retina were œdematous and the disc of somewhat florid colour. There were no hæmorrhages or exudates. The left fundus was normal.

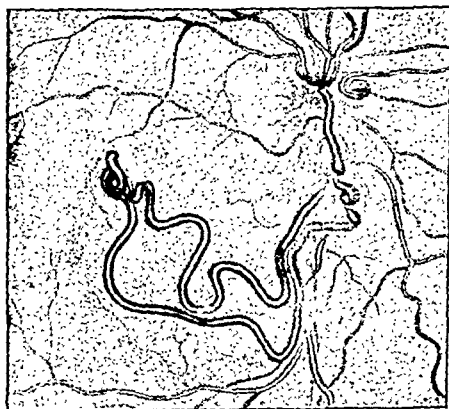


FIG. 1.



FIG. 2.

FIG. 1.—Angiomatosis retinæ. State of the right fundus before treatment. The pathological vessels are confined to the lower half of the retina.

FIG. 2.—State of the retinal vessels five months after X-ray treatment. The abnormal vessels in the lower half show thickening and opacity of their walls, and narrowing of the lumen.



FIG. 3.—State of the fundus in the eye two years after X-ray treatment. (Binocular microscope $\times 7$.) More extensive changes in the retinal vessels, especially in the lower half. Massive retinal hæmorrhages which had caused secondary glaucoma and led to excision.

and, indeed, so long as the fundus was visible there was no *apparent* change in the other retinal vessels. Twelve months later, however, inspection of the excised eye with the corneal microscope showed opacity in the walls of some of these, while sections revealed that there were practically no normal vessels in any part of the retina. The vascular changes, however, were notably more advanced in the lower region. Even the vessels on or near the disc show this contrast between those belonging to the lower and those belonging to the upper parts of the retina (fig. 4).

The clinical and pathological findings make it evident that the pathologically distended vessels were more radio-sensitive than the normal ones, and suggest that a smaller dose of the radiation, or similar doses spread over a longer period, might have produced the desired obliterative change in the big vessels without affecting the normal retinal circulation or producing the other harmful by-effects seen in this case.

One naturally hoped to find some histologically normal vessels and to be able to trace the sequence of changes from these to the completely obliterated vessels; but this was rendered difficult if not impossible by the radical differences between the vessels in the upper and lower halves of the retina above referred to.

It will be remembered that the accepted description of the vessel changes resulting from exposure to radium or X-rays (Colwell and Russ, 1934) is that there is a swelling of the collagenous elements in the vessel walls, proliferation and degeneration of the endothelial cells causing obliteration of the lumen, splitting of the elastic fibres and hyaline degeneration of the muscular coat.

If we examine some of the least abnormal vessels in the upper part of the retina (fig. 5), we find the walls thickened, and already showing a feebleness of the eosin staining, and a reduction in the number of nuclei. A single layer of endothelium is present, and the lumen of the vessels shows little, if any, distension or contraction. Only in a few instances can we find a thin layer of subendothelial tissue inside an internal elastic lamina. At this stage the Van Gieson and Mallory stains give the characteristic reactions of connective tissue; but Weigert's elastic tissue stain shows an increase of darkly staining elastic fibres throughout the whole thickness of the wall (fig. 6). Contrasting this with the vessels of similar size from the lower part of the fundus we find, in the latter, a similar weakness of the eosin staining, the zone nearest to the lumen being paler and poorer in nuclei, while there is a great reduction in the diameter of the lumen (fig. 7). Weigert's elastic tissue stain brings out the fact that the narrowing of the lumen is brought about by the presence of a thick, homogeneous feebly staining layer, within a coat composed of elastic tissue fibres similar to that described in the upper group of vessels (fig. 8).

It would appear then, that at this stage we see a notable swelling and degeneration of the collagenous elements of the outer coats of the vessels mingled with an abundance of elastic fibres, while in the pathologically distended vessels, associated with the angiomatous lesion, there is a greatly thickened intima, already reduced to a hyaline state.

The description already given applies to most of the vessels of the first group; but some of them show a reduction in the fibrillary character of the wall, and a feebler reaction to the Weigert stain, so that the wall throughout its thickness assumes a pale purplish colour and a structureless appearance (fig. 9). The lumen still remains comparatively open and there is no suggestion of the inner coat seen in the vessels of the second group. Among the latter, we can trace a sequence of more and more complete degenerative changes (fig. 10). The two coats remain distinguishable almost to the last stage, the outer one preserving a certain fibrillary structure, while the inner, sometimes separated from the outer by a sinuous refractile line, remains amorphous. Both become progressively paler, nuclear staining disappears from the outer coat, and the increasing thickness of the inner wall causes extreme narrowing of the lumen until we finally get a shrunken solid, hyaline, structure without a lumen and giving merely a pale purplish reaction to the Weigert stain.

There is little or no abnormality of the vessels in and around the optic nerve after it has left the eyeball.

I have made no attempt in this description to differentiate arteries from veins, as it seems to me impossible in these pathological vessels to do so with any certainty.

When the serial sections reach the macular region the retina is naturally greatly dis-

The two portions of the eye were embedded in celloidin and serial sections cut in an approximately vertical direction.

Interest naturally centred for the most part in the condition of the retina and of the retinal vessels; and especially in the nature of the reaction of the pathological vessels and the angioma to the X-ray application.

The histological picture was inevitably disturbed by the gross hæmorrhage, the more so as there was an interval of two months between the occurrence of the intra-ocular hæmorrhage and the excision of the eye. Moreover, for about a year before the excision



FIG. 4.—Section of retina adjacent to nasal disc margin. Shows contrast between inferior vessel with relatively thick hyaline wall and small lumen, and superior vessel with thick but less degenerate wall and relatively wide lumen. [Low power / Obj. 1". Oc. 2.]

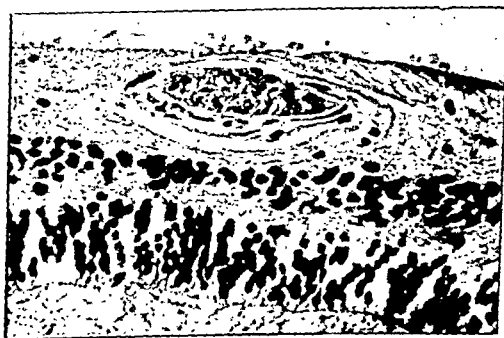


FIG. 5.—A vessel in the upper part of retina; hæmalum and eosin. The wall is thickened but the lumen is wide. [High power / Obj. $\frac{1}{2}$ ". Oc. 2.]

it was impossible to get a clear view of the fundus, on account of changes in the cornea and lens. But, at the same time, the picture seen in the excised eye by the corneal microscope, helped to bridge this gap, having a sufficiently close resemblance to the ophthalmoscopic drawing, made two years earlier, to enable us to correlate the clinical and the pathological changes.

Perhaps the most interesting and important clinical observation was the fact that the earliest changes seen with the ophthalmoscope occurred in the large pathological vessels;

turbed by the bulky and extensive hæmorrhages and exudates; but we find here a compact mass of fibrous tissue covering an area about 2 mm. in diameter and occupying the nerve fibre and ganglion cell layers: with a maximum depth of about 0.5 mm. In this compact tissue, which stains to a brownish-red colour with hæmatoxylin and eosin, and which appears to be composed of glia, are found many irregularly shaped blood-filled spaces, as well as greatly distended capillaries and small vessels with more or less thickened walls. It seems fairly certain that this highly vascular area represents the angiomatous growth which forms the link between the large tortuous retinal vessels in the lower part of the fundus.

At a point in the lower half of the retina, a considerable distance below the macula, there is a small cluster of greatly distended capillaries in the nerve fibre layer. It is possible that this represents a small rudimentary angioma.

Throughout the greater part of the fundus included in the specimen, that is to say, over an area about 20 mm. in diameter, the retina presents a greater or lesser degree of disintegration. The changes are irregular in their distribution.

On the whole there is relatively good preservation of the retinal layers in the area below the disc and macula—the region of the pathological vessels. The upper half of the retina where the vessels were more normal, was the site of the first exudates (the constellation of white spots) and in the microscopic sections shows greater disturbance of the retinal layers.



FIG. 9.—The most advanced change in vessels of the upper half of the retina: a thick hyaline wall with a relatively large lumen. [High power; Obj. 1". Oc. 2.]

Œdema of the nerve fibre layer is widespread. Within and somewhat beyond the area disturbed by the hæmorrhage there are round or oval cavities in the inner molecular layer, in the inner nuclear layer and in the outer molecular layer. Those in the two inner layers are for the most part empty in the sections, but those in the outer molecular layer, which may break through into the inner and outer nuclear layers, are in many places filled with red blood corpuscles, albuminous exudate or masses of fibrin, a picture resembling that seen in so-called albuminuric retinitis (fig. 11). This type of lesion is most fully developed in the lower central part of the fundus, where the cavities in the outer molecular layer are very large and are separated from one another by stout columns of hyaline looking material (fig. 12). These spaces break through the inner nuclear layer into the inner molecular, and the nuclei belonging to the former are found scattered irregularly along the columns separating the cavities.

Exudates of albuminous character are found between the internal limiting membrane and the nerve fibre layer, or even extending deeply enough to impinge on the external



FIG. 6.—The same vessel: Weigert's elastic tissue stain. Proliferation of elastic tissue throughout the whole thickness of wall. No intimal thickening. [High power / Obj. $\frac{1}{2}$ ". Oc. 2.]



FIG. 7.—Vessel in lower half of retina: haemalum and eosin. Great thickening of whole wall and very narrow lumen. [High power / Obj. $\frac{1}{2}$ ". Oc. 2.]

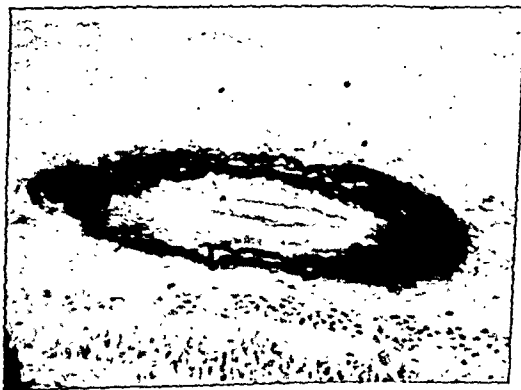


FIG. 8.—The same vessel: Weigert's elastic tissue stain. The wall consists of two portions: an intima, greatly thickened and completely hyaline, and an adventitia composed largely of darkly stained elastic fibres. The lumen is very narrow. [High power / Obj. $\frac{1}{2}$ ". Oc. 2.]

nuclear layer. Similar exudates are found between the rod and cone layer and the pigmented epithelium.

Hæmorrhages are a very prominent feature of the sections throughout the central area of the fundus, and they occur at all depths in the retina. Most prominent of all are those covering the macular region, which the microscope shows to be situated for the most part

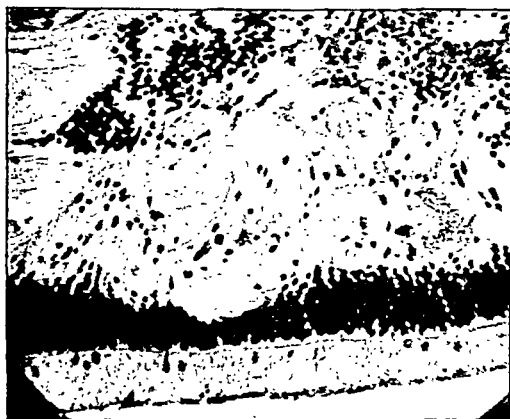


FIG. 11.—Disorganization of the retina, with formation of cavities in all layers, some containing masses of whole blood, serum or fibrin. The external nuclear layer is fairly well preserved but the internal nuclear is broken up and scattered. [Low power / Obj. 1". Oc. 2.]



FIG. 12.—Large cavities occupying the external molecular, the internal nuclear and the internal molecular layers. [High power / Obj. $\frac{1}{4}$ ". Oc. 2.]

under the internal limiting membrane. In two situations coagulation has squeezed out a serum which lies in front of the massive hæmorrhage (fig. 13) and is represented in fig. 3 by the grey cystic-looking masses with a delicate striation apparently due to traction folds.

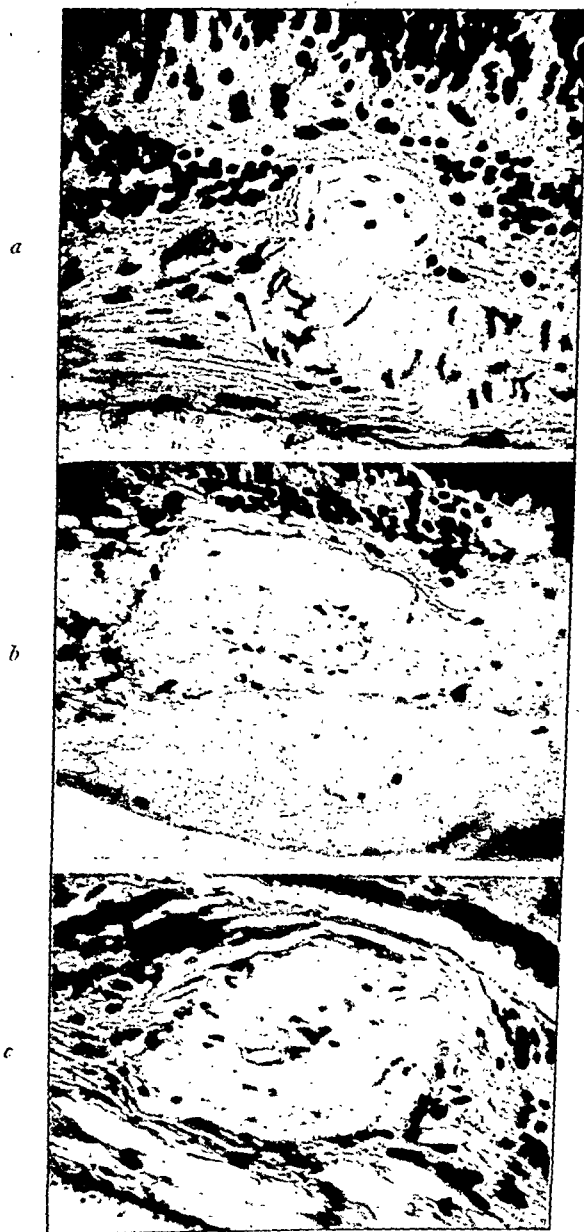


FIG. 10.—Three vessels, *a*, *b* and *c*, from lower half of retina, showing progressive changes. The adventitia, as well as the intima, becomes completely hyaline, the lumen diminishes and finally closes. [High power / Obj. 1". Oc. 2.]

In addition to these intraretinal hæmorrhages there are hæmorrhages under the retina (between the rods and cones and the pigmented epithelium) as also in front of the internal limiting membrane. There was, of course, gross hæmorrhage into the vitreous, and a layer of blood corpuscles is found on the anterior surface of the retina in most of the sections. The least disturbed part of the retina is the external nuclear layer which, for the most part, presents a fairly normal and uniform depth throughout the sections. The rods and cones are well preserved in some places, destroyed in others. The ganglion cells in general are either scanty or entirely absent. The retinal pigment epithelium is intact in most of its extent.

It is difficult to judge the condition of the choroid in these formalin-fixed specimens, but it shows variations in thickness and apparently in vascularity from one point to another. In one area about the temporal limit of the large hæmorrhages, and some six millimetres below the macular level, the choroidal vessels have thickened hyaline walls, and over the same area (some 6 mm. in extent) the pigmented epithelium is of very irregular thickness, the retina is thin, with loss of its rods and cones and partial destruction of the other layers, and there is migration of pigment into the atrophic retina.

Far forward, behind the ora serrata, some sections show a collection of "ghost cells", mingled with pigmented cells (no doubt shed from the hexagonal pigment layer) lying between the rods and cones and the pigmented epithelium.

The optic nerve shows some atrophy of the nerve bundles and thickening of the fibrous septa. In the pial sheath there are many dilated blood-vessels, some with thickened walls.

In sections of the anterior segment of the eyeball, there are pathological changes in the cornea, iris and ciliary body. The corneal epithelium varies in depth and there is some desquamation of the superficial cells. At the limbus there is vascular pannus and round cell infiltration of the cornea and episclera. Over a small area in the lower part of the cornea there is destruction of Bowman's membrane. Descemet's membrane and the corneal endothelium are intact; but there are a few K.P. The ciliary body and processes show some atrophy. There is partial closure of the angle of the anterior chamber. In some of the iris vessels there is a thickened hyaline wall with narrowing of the lumen.

COMMENTS

The occurrence of a capillary angioma of the retina in or near the macula is unusual if not unique. Junius (1930), describing a case in which the angioma was peripherally situated, but caused a central scotoma, remarked that up to that date no case of angioma at the macula had been reported: and I have failed to find any reference to such a case subsequent to the date of Junius' contribution.

It is unusual also for a case to come under observation before the angioma has become visible either as a red vascular nodule or as a pale globular tumour—an angiogliosis of the retina. It is noteworthy that in the present case, even after the vessels had reacted by showing thickening and loss of transparency of their walls there was no visible tumour mass at the macula.

Another case which was exceptional in this respect was that reported by Worms and Pinelli (1930) in which one of the large vessels returned to the papilla and the other seemed to sink into the retina, no angiomatic nodule being visible.

The difference in the reaction of the pathological and the normal vessels to the X-ray application is a point of interest and importance. While a few vessels in the upper half of the retina showed a very thin subendothelial internal coat, there is a very striking difference between this and the greatly thickened and degenerate intima seen in the vessels of the lower half; and it is difficult to believe that the first type of reaction was simply an earlier stage of the second. If we compare vessels from the upper and lower halves whose walls have reached the stage of complete hyaline degeneration we see that the first have still only one distinguishable coat, and an open lumen (figs. 5, 6, 9), while the second have two distinct coats and a more or less complete obliteration of the lumen (figs. 7, 8, 10). The latter condition seems to be that described as an endarteritis (or

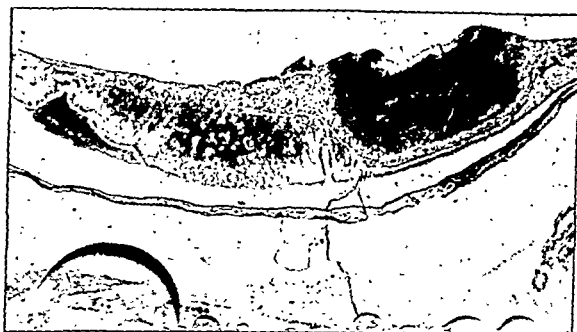


FIG. 13.—Section through the area of massive hæmorrhage, which also contains the angioma. Hæmorrhages are pre-retinal, intra-retinal and sub-retinal. The large dark hæmorrhage is within the angiomatous growth and lies entirely anterior to the inner molecular layer. [Low power / Obj. 1". Oc. 2.]



FIG. 14.—Dilated, thin-walled capillaries surrounded by retinal hæmorrhages [High power / Obj. $\frac{1}{8}$ ". Oc. 2.]

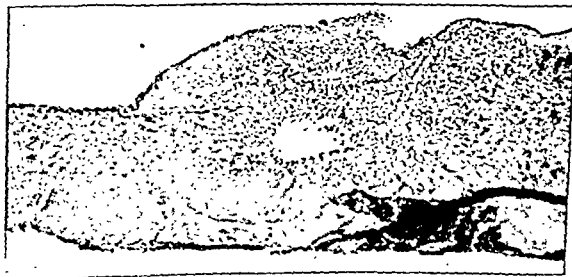


FIG. 15.—Site of the retinal angioma, which is situated within the nerve fibre and ganglion cell layers. Rupture of internal limiting membrane with escape of hæmorrhage into vitreous cavity. [Low power / Obj. 1". Oc. 2.]

a case as this, a globular mass would have developed in the course of time in a similar way. Moreover, one of the early results of experimental radiation of vascular tissues is destruction of the endothelium of the vessels. And the integrity of the walls of the capillaries in this case would be further damaged by the anoxia resulting from impeded circulation in the partially obliterated vessels.

In the presence of hæmorrhages and exudative changes closely resembling those found in hypertensive retinitis it is surprising that there is almost complete absence of the so-called "gangliiform degeneration" of the nerve fibre layer.

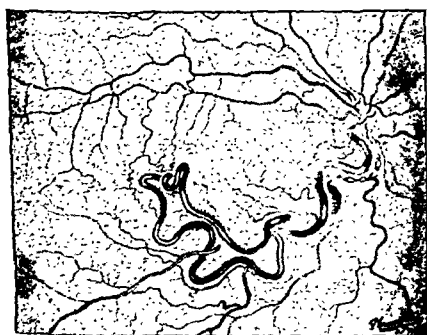
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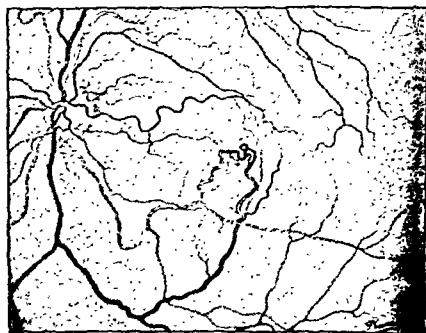
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 WORMS, G., and PINELLI, J. (1930), *Bull. Soc. d'ophth. de Paris*, 42, 335.

Discussion.—Dr. BALLANTYNE in reply to the Chairman said that originally the tension was 30 mm. in the affected eye, but it went up after that. The tension digitally, as compared with the other eye was very high—probably in the region of 40 or 50.

Mr. A. SEYMOUR PHILPS showed two coloured drawings (fig. 1) of the eyes of a patient which had been shown at a meeting of the Section in 1939 (*Proceedings*, 32, 1260). She was a woman of 35, with an arteriovenous communication in each eye. She complained very much of headaches and at a later stage of noises in the head. When the case was



R.E.



L.E.

FIG 1.—Right and left fundi showing arteriovenous communication in each.

shown to the Section some members recommended diathermy, but others urged that it be left alone. The case was left alone, and the vision, which had been $\frac{5}{60}$ in each eye, became $\frac{5}{60}$. It was almost impossible to distinguish the arteries and veins except in so far as the branches of the vessels were not affected. In one eye the condition was not quite so marked as in the other at that time, but six months later she came back because vision in that eye had gone down suddenly; it was in fact $\frac{5}{60}$ and there was white exudate at the arteriovenous communication. No treatment was given, the patient was told to go home and rest. The vision improved to $\frac{1}{2}$ and there it remained, he believed, at the present time. The patient still complained of rhythmical noises in

endophlebitis) obliterans. The former does not fall into that category, and the sections do not show any transition stages between the one and the other.

The vessel changes in this case do not tally in all respects with those usually described as resulting from experimental exposure of healthy vessels to X-rays. Where cellular and nuclear staining is still present there is no evidence of proliferation of the endothelium. In the vessels of the lower retinal region the thickening which is causing narrowing and obliteration of the vessels is probably a proliferation of the subendothelial intima. Even in the capillaries of the angioma itself, there is no endothelial proliferation.

The demonstration of the histological explanation of the ophthalmoscopically "sheathed" vessels is of some interest. In a joint paper with Michaelson and Heggie (1938) it was shown that a white sheathed vessel, identified microscopically, presented atheromatous thickenings of the intima which had undergone a lipid degeneration. It was pointed out that one would not be justified in assuming this to be the condition in every case of vessel sheathing, all that could be claimed was that the appearance of sheathing demanded the occurrence of a qualitative as well as a quantitative change in the vessel wall. In other words, a retinal vessel may be represented by a thread-like blood column although the vessel wall is ophthalmoscopically invisible, so long as the wall is thickened by mere increase of its normal structures. "Sheathing" only makes its appearance when these structures undergo hyaline, lipid or some other form of degeneration. In the present case there is no patchy thickening of the vessel wall, no eccentricity of the lumen, and no sign of a lipid change. We find another kind of qualitative change, namely a hyaline degeneration of the whole vessel wall. It is interesting again to note that in many vessels of the upper part of the retina there was sheathing but no thickening of the intima, merely a hyaline degeneration of the thickened adventitia.

While it is possible, in microscopic sections, to identify the angioma at the macula, the extensive hæmorrhage makes it difficult to study this satisfactorily. No clear view of the fundus was obtained during the twelve months before excision of the eye, and, although the view of the fundus of the excised eye given by the corneal microscope was in a sense a substitute for ophthalmoscopic examination at that date, the hæmorrhage which had occurred two months earlier made it difficult to say to what extent the other retinal changes were due to the natural progress of the vascular lesion, the X-ray treatment or the damaging effects of the hæmorrhage.

The simple structure of the angioma serves to bring it into line with the view of E. T. Collins, v. Hippel, Leber and others that the primary lesion is derived from angioblastic sources, and is in fact a vascular tumour of congenital origin. The greater part of the tumour mass at this stage is composed of glia cells and fibres enclosing the wide capillaries and irregularly shaped blood-filled spaces, both lined with a single layer of endothelium. The gliosis would appear to be a secondary change due to local tissue reaction.

With regard to the source of the hæmorrhage, there is no direct association of the hæmorrhages with the larger vessels, indeed there is a comparative freedom from hæmorrhage in the lower part of the retina where these vessels are most abnormal.

Possibly the greater part of the hæmorrhage came originally from the angioma itself, and the neighbouring capillaries. There are many of these greatly distended capillaries buried among the hæmorrhages. Their walls seem to consist of a single layer of endothelium incomplete in some places as if from rupture (fig. 14). At one point in front of the angioma there is a rupture of the internal limiting membrane through which blood has escaped and is lying in a compact mass on the anterior surface of the retina (fig. 15).

In every case of hæmorrhage the attempt to explain its occurrence requires the consideration of three possible factors: the condition of the blood, the state of the vessel walls and the height of the blood-pressure. Most, if not all, of the hæmorrhages in this case are seen in similar forms in cases of hypertensive disease, with or without renal involvement; but it is very doubtful if the hypertension *per se* is an important factor. In the present case, there was neither hypertension nor any physical defect in the blood. It seems natural to attribute the hæmorrhage to the state of the blood-vessels. It is generally believed that the glial and connective tissue proliferation which produces the characteristic "massive exudate" in Coats' disease may be the result of sudden or gradual escape of blood from congenitally fragile vessels; and it is not improbable that in such

a case as this, a globular mass would have developed in the course of time in a similar way. Moreover, one of the early results of experimental radiation of vascular tissues is destruction of the endothelium of the vessels. And the integrity of the walls of the capillaries in this case would be further damaged by the anoxia resulting from impeded circulation in the partially obliterated vessels.

In the presence of hæmorrhages and exudative changes closely resembling those found in hypertensive retinitis it is surprising that there is almost complete absence of the so-called "ganglioform degeneration" of the nerve fibre layer.

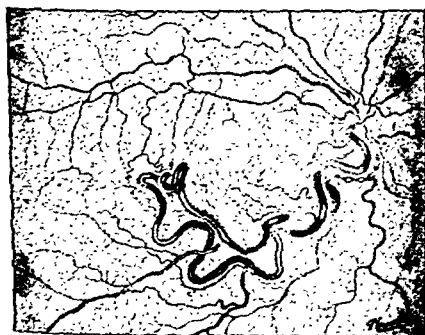
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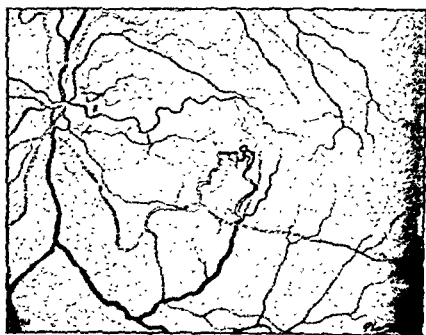
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endophlebitis) obliterans. The former does not fall into that category, and the sections do not show any transition stages between the one and the other.

The vessel changes in this case do not tally in all respects with those usually described as resulting from experimental exposure of healthy vessels to X-rays. Where cellular and nuclear staining is still present there is no evidence of proliferation of the endothelium. In the vessels of the lower retinal region the thickening which is causing narrowing and obliteration of the vessels is probably a proliferation of the subendothelial intima. Even in the capillaries of the angioma itself, there is no endothelial proliferation.

The demonstration of the histological explanation of the ophthalmoscopically "sheathed" vessels is of some interest. In a joint paper with Michaelson and Heggie (1938) it was shown that a white sheathed vessel, identified microscopically, presented atheromatous thickenings of the intima which had undergone a lipid degeneration. It was pointed out that one would not be justified in assuming this to be the condition in every case of vessel sheathing, all that could be claimed was that the appearance of sheathing demanded the occurrence of a qualitative as well as a quantitative change in the vessel wall. In other words, a retinal vessel may be represented by a thread-like blood column although the vessel wall is ophthalmoscopically invisible, so long as the wall is thickened by mere increase of its normal structures. "Sheathing" only makes its appearance when these structures undergo hyaline, lipid or some other form of degeneration. In the present case there is no patchy thickening of the vessel wall, no eccentricity of the lumen, and no sign of a lipid change. We find another kind of qualitative change, namely a hyaline degeneration of the whole vessel wall. It is interesting again to note that in many vessels of the upper part of the retina there was sheathing but no thickening of the intima, merely a hyaline degeneration of the thickened adventitia.

While it is possible, in microscopic sections, to identify the angioma at the macula, the extensive hæmorrhage makes it difficult to study this satisfactorily. No clear view of the fundus was obtained during the twelve months before excision of the eye, and, although the view of the fundus of the excised eye given by the corneal microscope was in a sense a substitute for ophthalmoscopic examination at that date, the hæmorrhage which had occurred two months earlier made it difficult to say to what extent the other retinal changes were due to the natural progress of the vascular lesion, the X-ray treatment or the damaging effects of the hæmorrhage.

The simple structure of the angioma serves to bring it into line with the view of E. T. Collins, v. Hippel, Leber and others that the primary lesion is derived from angioblastic sources, and is in fact a vascular tumour of congenital origin. The greater part of the tumour mass at this stage is composed of glia cells and fibres enclosing the wide capillaries and irregularly shaped blood-filled spaces, both lined with a single layer of endothelium. The gliosis would appear to be a secondary change due to local tissue reaction.

With regard to the source of the hæmorrhage, there is no direct association of the hæmorrhages with the larger vessels, indeed there is a comparative freedom from hæmorrhage in the lower part of the retina where these vessels are most abnormal.

Possibly the greater part of the hæmorrhage came originally from the angioma itself, and the neighbouring capillaries. There are many of these greatly distended capillaries buried among the hæmorrhages. Their walls seem to consist of a single layer of endothelium incomplete in some places as if from rupture (fig. 14). At one point in front of the angioma there is a rupture of the internal limiting membrane through which blood has escaped and is lying in a compact mass on the anterior surface of the retina (fig. 15).

In every case of hæmorrhage the attempt to explain its occurrence requires the consideration of three possible factors: the condition of the blood, the state of the vessel walls and the height of the blood-pressure. Most, if not all, of the hæmorrhages in this case are seen in similar forms in cases of hypertensive disease, with or without renal involvement; but it is very doubtful if the hypertension *per se* is an important factor. In the present case, there was neither hypertension nor any physical defect in the blood. It seems natural to attribute the hæmorrhage to the state of the blood-vessels. It is generally believed that the glial and connective tissue proliferation which produces the characteristic "massive exudate" in Coats' disease may be the result of sudden or gradual escape of blood from congenitally fragile vessels; and it is not improbable that in such

a case as this, a globular mass would have developed in the course of time in a similar way. Moreover, one of the early results of experimental radiation of vascular tissues is destruction of the endothelium of the vessels. And the integrity of the walls of the capillaries in this case would be further damaged by the anoxia resulting from impeded circulation in the partially obliterated vessels.

In the presence of hæmorrhages and exudative changes closely resembling those found in hypertensive retinitis it is surprising that there is almost complete absence of the so-called "ganglioform degeneration" of the nerve fibre layer.

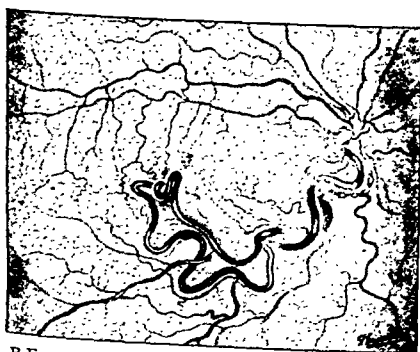
I should like to express my thanks to Dr. J. Pendleton White for his courtesy in sending this case for investigation, and for giving permission for its publication.

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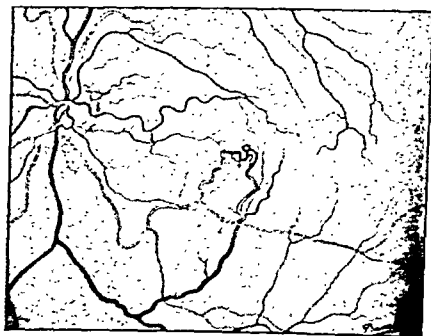
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the head, and had been twice admitted to hospital for a general neurological examination. The surgeon was now of opinion, however, that there was no general neurological lesion. The patient had listened so often to the descriptions of symptoms from which she should be suffering, that she had learnt them by heart.

Dr. BALLANTYNE said the point Mr. Philps raised about the vision having gone down owing to the presence of exudate, and the exudate disappearing, was rather interesting, and it would be useful to know what the precise reaction was, because nothing of the kind had been described so far. In most of the cases hitherto described and illustrated there had been a nodule at the confluence of the two big vessels, which no doubt began with the simple angioma. But what the explanation was of this transient change of the macula it was very difficult to say. Mr. Philps would of course follow up his case, and see what other changes took place.

In reply to Mr. R. H. Bickerton Dr. Ballantyne said that in his case there were quite clearly no similar changes in the other eye.

Section of the History of Medicine

President—J. F. HALLS DALLY, M.D.

[December 3, 1941]

Hospital Building—Past, Present and Future

By CHARLES E. ELCOCK, F.R.I.B.A.

(Abridged)

THE PAST

THERE appears to be reasonable evidence that in Ireland, Egypt and India, houses for the care and treatment of sick people were in existence 300 years before Christ. In Ireland they were called "Houses of Pity", but until the Christian era it would appear that sick people were considered somewhat burdensome and were indeed sometimes made away with if in the interest of the community. With the spread of Christian ideas in Europe it became usual for emperors and others to build and equip centres for the care of suffering humanity. Some of these were remarkable. The Basilias or Hospitium erected by St. Basil in Caesarea in A.D. 369 was a town of healing, separate streets being set aside for different groups of patients, with separate houses for physicians and nurses. In A.D. 400 Fabiola established houses for the sick in every quarter of Rome and there still remains a legend that she founded the existing hospital which bears her name in that city. When Augustine came to this country in A.D. 597 he founded a number of houses for the sick poor, called "bede homes" or "prayer homes", as it was thought that health was controlled by prayer. With the foundation of the great monastic orders in the fifth century and onwards, a cardinal principle was that of caring for the poor and infirm and ministering to their cure.

In A.D. 787 the Emperor Charlemagne commanded that every Bishop should institute a hospital, if possible near to the cathedral. This certainly initiated a development of houses for the special care of the infirm and poor. In England it is probable that A.D. 900 saw the establishment of the first English hospital by the then Archbishop of Canterbury. St. Bartholomew's Hospital in London was founded by Rahere, King Henry the First's jester, turned monk, about the year 1123.

The history of the Royal Hospital of Bethlem or Bethlehem is exceptionally interesting. The Charity was founded by Simon FitzMary in A.D. 1247. The first historical use of the hospital for "distracted persons" appears to be round about 1377. In 1346 the House was taken under the protection of the Mayor and Aldermen of the City of London. In 1403 the Priory of Bethlem had in it "six men who had lost their reason". In 1553 Bridewell Hospital was incorporated by letters patent. In 1676 the second hospital was erected in Moorfields, the architect being Robert Hooke, a genius comparable to Sir Christopher Wren, who was a colleague of Hooke. In 1812 the third Bethlem Hospital was begun in St. George's Fields, Southwark. The architect for this building was E. John Gandy in co-operation with the Hospital Surveyor—James Lewis. Many additions were afterwards made, the most notable being the dome and adjacent buildings which were the design of Sydney Smirke, and were carried out in 1844. In 1926 a magnificent country site of 330 acres was purchased at Shirley, near Croydon, and the fourth Bethlem Hospital was erected and opened in 1930. For this building I had the honour of acting as architect in close co-operation with the excellent surveyor to the hospital, the late Mr. J. A. Cheston. The development of the treatment of mental patients can scarcely be better shown than by a comparison of the plans of the 1676 building and those of 1812 and 1930.

THE PRESENT

Modern hospital planning may be said to have begun round about 1856—the Crimean War period—and was largely assisted by the wise and far-seeing counsel of Florence Nightingale. The Royal Bucks Infirmary at Aylesbury, Addenbrookes' Hospital at Cambridge, Winchester, Guildford, Woolwich, and a host of others were all products of this period, and were, for the most part, the outcome of voluntary and humanitarian ideals. This point requires stressing. It is one of those main historical trends referred to at the outset of this paper, a trend which is a direct development from the old religious foundations.

The passing of the Public Health Act of 1875 ushered in the principle that the health of the nation is the concern of the nation. This Act, with amending Acts—the Isolation Hospital Acts of 1893 and 1901 and others—led up to the Local Government Act of 1929, in which County Councils were required to make a Survey of all hospitals in their area and submit a report to the Ministry of Health. It further did away with the old Poor Law Guardians and required the establishment of Public Assistance Committees which had wide powers of co-operating with suitable voluntary and special hospitals in their areas and were empowered to build and equip further hospitals if found necessary and approved by the Ministry of Health. I was particularly interested in this development being appointed to make the survey and report for the Herts County Council. This Survey and Report was the first to be presented to the Ministry and formed a precedent for many which followed. The increased governmental interest in public health was reflected in the further development of the great and small voluntary hospitals throughout the country.

Within the last thirty years we have seen the development of methods of treatment scarcely apprehended in previous years. This required the hospital architect to devote much attention to a detailed examination of these methods so as to accommodate them with an understanding mind. Diagnostic and therapeutic X-ray departments, radium, sunlight, oxygen treatment, hydrotherapy and many other methods, with clinical, chemical, and pathological laboratories and enlarged out-patients departments and numerous other developments, have all placed the scientific planning of hospitals in the front rank of architectural achievement. The period mentioned has also seen the founding of specialized hospitals and a great development of facilities for the teaching of medical and surgical students. The development of hospitals and centres for treating varying types of mental-disease was not remarkable for the introduction of new ideas in planning during the period under review. But during the last fifteen years great strides have been made, and the modern mental hospitals of this country are worthy of study and are thoroughly up to date.

It still remains an anomaly in our modern British life that the people who could afford to pay a reasonable sum for skilled hospital treatment have the least chance of that treatment and have either to be nursed at home or pay very high fees at private nursing homes. There is a need for a new type of hospital which, fully equipped and planned on modern lines, will serve the needs of what has been called the "white collar" patient. For some time before the war plans had been maturing for a large hospital of this type in London, in which it had been proved by skilled hospital accountants that the charge of £1 a day would cover all hospital costs, leaving the patient in the care of his usual medical adviser, the fees for professional services being paid by the patient as if at his own home. The completion of this project was postponed by the outbreak of war, but it is obviously a development which should not be lost sight of.

The establishment of therapy centres for the treatment of cancer throughout the country and the inclusion of mental treatment in general hospitals are two important trends in present-day hospital building. The maternity and child welfare clinics mark a very important advance in public health.

The actual planning of hospitals has developed from the ward as the central feature. At first large wards were the rule and persisted down to, say, ten or fifteen years ago. In the last fifteen years there has been a tendency to reduce the number of patients in a ward or to segregate them into wards divided by glazed screens, which allow for classification and a lessening of possibilities of cross-infection. In the planning of the extension of the Hertford County Hospital, it appeared to me that it was advisable to design an entirely new type of ward. The increasing appreciation of sunlight and air as valuable therapeutic agencies had made me realize that in our wards we actually used up

70% of the wall surface with bricks, the window openings forming only 30% of the whole. Surely the exact reverse should be the case? Again, the need for sunlight and air had led to the construction of wide and costly balconies which shaded the wards below and required the increased labour of wheeling beds in and out of the wards. Why not turn the wall space into long windows with suitably placed narrow constructional piers and by arranging these windows to slide open we could achieve at one operation the introduction of the maximum of sunlight and the benefits of veranda treatment? This developed the plan of what I have called the "veranda-ward" with the beds grouped between glazed screens, the beds being placed parallel to the wall instead of at right-angles. I have been glad to observe that this arrangement has been followed and copied in this country and all over the world. Australia, Japan, America, Sweden, Finland, and Italy all have examples of this veranda-ward, first introduced at the County Hospital at Hertford, England, in 1929. The plans and details were submitted to the Government Department of Building Research at Watford, who made two models—one of the old type of ward with beds at right-angles to the walls with window between the beds, and the other a model of the new "veranda-ward". These models were placed under an artificial sun which was placed at varying inclinations corresponding to winter, spring, summer and autumn. The Building Research Station prepared the following comparative table of the results which show that at all seasons the new ward gives more than twice as much possibility of sunshine as the old type of ward.

COMPARATIVE TABLE SHOWING VOLUME OF SUNSHINE RECEIVED BY OLD AND NEW (VERANDA-WARD) HOSPITAL WARDS.

| | | Super-feet per day | | | |
|---------------|------------------------|--------------------|----------|---------|---------|
| Old ward type | Direction of ward axis | Jan. 17 | April 15 | July 17 | Oct. 16 |
| | N.-S. | 1,236 | 2,517 | 2,089 | 1,830 |
| | N.E.-S.W. | 1,463 | 2,139 | 2,248 | 1,785 |
| | N.W.-S.E. | | | | |
| | E.-W. | 1,776 | 1,480 | 1,494 | 1,841 |
| New ward type | N.-S. | 2,334 | 5,066 | 5,741 | 3,572 |
| | N.E.-S.W. | 2,873 | 4,264 | 4,522 | 3,524 |
| | N.W.-S.E. | | | | |
| | E.-W. | 3,635 | 2,919 | 2,924 | 3,727 |

I have stressed this matter of the ward unit as it is in the interest of the patient. Similar developments are possible in almost every branch of hospital planning, which will lead to simplification, efficiency and economy in labour and cost. A great deal remains to be done in this matter of hospital planning, as from 1856 up to say 1914, hospital planning with few exceptions became standardized, crystallized and even fossilized. Complacency and tradition make bad masters. The stones they build with have time to gather the proverbial moss.

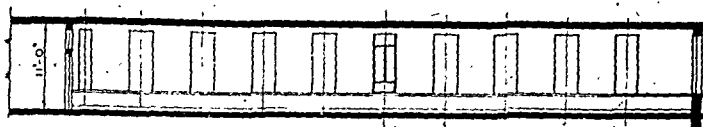
Sufficient attention has not been directed to the extraordinary variations in the cost of modern hospitals. The rough and ready method of assessing the cost at so much a bed has much to be said for it—provided the hospitals to be compared are of similar purpose and equipment. A large voluntary hospital lately erected in London at a cost of over £2,000 a bed is comparable to a somewhat similar hospital lately erected in the provinces at a cost of under £700 per bed. If allowance is made for certain special features in the London Hospital it is still impossible to agree to a figure of over £2,000 a bed. A Cottage Hospital in Wiltshire at under £500 a bed and fully equipped has to be compared with a similar Cottage Hospital in Yorkshire, erected at a cost of £1,000 a bed.

It is evident that there is an enormous divergence in the constructional cost of large hospitals of similar type. These variations include a lack of definite standards in the matter of accommodation, construction and equipment.

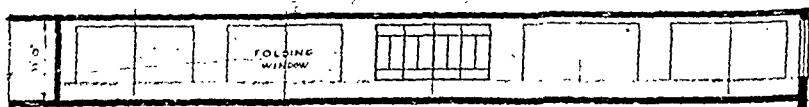
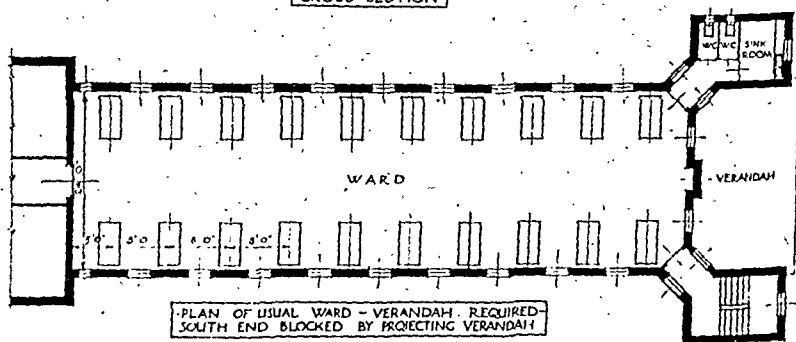
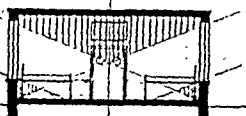
These essentials of modern hospital design should be agreed upon by a conference between medical men, the nursing faculty, hospital administrators and architects.

The architect must try and forget his little architectural tricks and details, and think more on the lines of a scientific "factory for health" rather than an imposing expanse of architectural display. The architectural effect will be gained by the dignity and sincerity of the planning and the arrangement of the masses without the expensive accretions of so-called architectural style, whether classic or modern. By the adoption of the

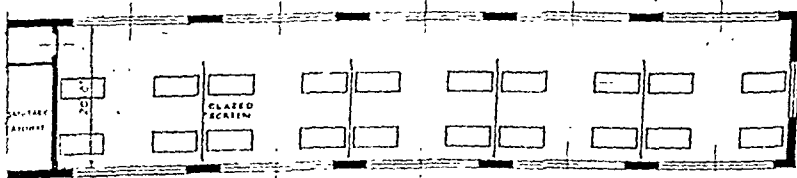
COMPARATIVE · WARDS



ONLY ONE BED GETS COMPLETE INSULATION AT A.M. OR P.M.



BOTH BEDS GET COMPLETE INSULATION AT A.M. AND P.M.



SCALE IN FEET

DESIGNED BY
C.E. RICHARD, F.R.S.D., F.R.C.S.
LONDON & BIRMINGHAM
CONSULTING ARCHITECTS
40, MARK LANE, LONDON, E.C. 3
40, MARK LANE, BIRMINGHAM, W. 2

"health factory" type of plan, with its scientific clarity and flexibility of design, its elimination of all out-moded or unnecessary features, including unnecessary corridors, not only is capital outlay saved but large economies can be effected in annual administrative costs.

My "health factory" consists diagrammatically of a long hall 20 ft. wide placed with the axis east and west. Large folding windows on the south façade come between the supporting stanchions. A typical floor would be divided into units of 30 beds each, and might be three or six units long, and from three to ten floors high, depending on the number of beds required. In the centre of each unit on the northern side are placed the necessary subsidiary rooms, toilet rooms and utility services. Each unit is separated by a cross wall from the adjacent units, but communicates through the escape staircase lobby. Each utility wing on the north side has its own bed and passenger elevators and staircase, but there is no connecting corridor except on the ground floor. All internal walls can be removed without interference with the structure, which allows the utmost flexibility for future needs. All sanitary and main service pipes are in the northern service wings. The main structure would be of steel with thin external walls lined with non-conducting material and noiseproof floors, sanitary apparatus, piping and doors.

The economy of this system of planning is at once apparent when it is shown by this method that a complete General Hospital for 500 patients—without a medical school—fully equipped with all fixed fittings, without furniture but including the nurses' home and all the heating and electrical installation could be erected in this country in 1939 for under £600 a bed.

The elimination of noise is a matter to which considerable thought is being given, both noise from outside and the working noises of hospital administration. Much remains to be done in this direction, and it is obvious more town and city hospitals must remove from their noisy city centres to the more open spaces on the outskirts, with all sorts of consequent advantages. Much cheaper sites could naturally be obtained on the outskirts of large cities and the revenue derived from the sale of valuable sites in congested areas would greatly assist in meeting the cost of the new hospitals.

The Departmental Committee on the cost of hospitals set up in 1933 published its final Report in 1938 and extremely valuable data were registered. The war has prevented its consideration and implementation, but it must be followed up and its findings discussed by the people concerned or these valuable reports may be lost sight of.

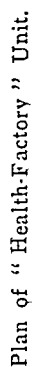
In the opinion of many the main thing which marks the present epoch in hospital history is the so-called "battle" between the voluntary hospital and the Government-controlled hospital. The present Minister of Health last October gave a lengthy exposition which was intended to clarify the position. The main thought underlying this appeared to be stated in the sentence: "It is accordingly proposed to lay on the major local authorities the duty of securing, in close co-operation with the voluntary agencies in the same field, the provision of such a service by placing on a more regular footing the partnership between the local authorities and voluntary hospitals." Whilst nothing definite appeared as to how this co-operation might be secured, it seems to dispose of the idea that this Government has any intention of attending the funeral of the voluntary system.

THE FUTURE

- This pronouncement by the Minister of Health encourages conference, discussion, and co-operation, so that a *via media* to first-class organized hospital service may be discovered. If there is one thing democracy rightly watches with proper jealousy, it is the growth of bureaucracy.

Whilst, however, the growth of bureaucracy must be watched carefully, it should be open to free discussion entirely untrammelled by party politics as to whether it is really true that the health of the nation is the concern of the nation. Whether indeed a nation is of any use unless it is healthful. If these things are really true, a discussion would undoubtedly arise as to whether the health of the people should be a matter of private gain, or public expenditure. It may well be that conference might show that the enlargement of interest in healthful living as one of the first essentials of national well-being might actually bring about a great increase in public officials. If the whole thing had been authoritatively proved, such a bureaucracy would be an essential organ in a healthy national body.

This development might suggest the consideration as to whether an outside Board or



Corporation should not be set up by Royal Charter which should look after the nation's health, and should control all hospitals both governmental and voluntary. Through this Board or Corporation the expenditure of public money would be directly controlled. Money given by voluntary donation or subscription is just as sacred as Government Funds and it would be the sole aim of this Directive Health Board to see that the best use was made of finance, technique and science.

The Directive Health Board might have powers to examine critically all proposals for new buildings or extensions of existing buildings; it might have powers to veto work which did not appear to be in line with modern health proposals, it might have power to arrange areas or regions and obtain surveys of all health work in the areas and see what was redundant and what lacking. In the United States the College of Surgeons maintains a staff of inspectors, who visit each hospital, go over the medical and hospital records, and eventually issue lists of approved hospitals. If a hospital cannot get this approval its appeal for public funds is not certain of public support. All our hospitals should be subject to such examination, and it should be undertaken by this or some similar medical society.

This Directive Health Board would work under the Ministry of Health on somewhat similar lines to the Board of Control of Mental Hospitals, and would thus preserve a continuity of aim in harmony with national democratic methods of government. This whole question of national health and education for healthful living should be untouched by party politics and should be a matter for conference and earnest unbiased discussion—it might assist in ensuring the first essential of any nation—a people who know how to live healthfully.

But whatever is to be done must be attempted as soon as possible—there is no need to wait for the war to stop. Let conference begin, whatever may be the outcome there must be a stop to the amateur development of hospitals which have so frequently been dominated by the well-intentioned whims of the wealthy patrons. The advent of the great Rockefeller Trust of America with its skilled experts to guide its activities showed the way in which great wealth can competently assist in great health movements. The operations of the King Edward VII Hospital Fund and the formation of the Nuffield Provincial Hospitals Trust have already exerted and will continue to exert a profound and beneficial influence in hospital development, provided that those controlling the funds rely upon expert guidance to direct their activities.

And yet—even in the case of the magnificent Nuffield Trust, it might be pointed out that the activities of this Trust might possibly go further than assistance to voluntary hospitals and might with great benefit to the community thrust itself out along other less travelled roads and explore the tangled jungle of our social and industrial conditions. This trust is a great—a magnificent—thing, it may be thought ungenerous to even criticize its operations, but I am convinced the man who has donated to this country these munificent benefactions will be the first to agree that constructive criticism of this noble Trust will be welcomed.

I am emboldened as a layman to emphasize education in healthful living as the first essential of the future of hospital building, because I see this subject referred to so frequently by medical men of national and international reputation. Perhaps it is put very concisely by Dr. Willskey of Boston (U.S.A.) in an Address in 1939 where he said:

“I should like to emphasize the fact that the activities of hospitals in the field of public health keep apace and abreast of the newer knowledge of how disease may be prevented. Hospitals are carrying on many activities which are all-important factors in the control of disease and in the promotion of the public health. One who has a true knowledge of the complete functions of the hospital has an appreciation of the fact that one of the important functions is the constant teaching of people for more healthful living. If we are to prevent disease to its fullest extent, we must acquaint people with those facts which are factors in the reducing of illness to a minimum and in the promotion of health. The greater participation by the medical profession in the field of preventive medicine will call upon the additional resources of hospitals. In the administration of methods and techniques by which disease may be prevented, in the fostering of health habits, and in the carrying on of clinical activities for the conservation of health and the prevention of disease, hospitals, as in the past, will continue their onward march for the betterment of the human race.”

Here we are in touch with something really big. On this high tableland we may feel the stimulus of a new and extended horizon.

What about the committees and medical staff of the future? I think most hospital architects will agree that they have sometimes to discuss the plans of large hospitals with boards and medical staffs and executives who are not well informed as to modern hospital developments. Very few have troubled to visit the United States or the fine new hospitals in Scandinavia or in other parts of Europe. How well-timed therefore was the public-spirited tour of these centres initiated and carried out by the Chairman of one of our newest London hospitals before the plans of the new buildings for the Westminster Hospital were finally settled.

The operations of the new health service will be frustrated if they are not directed by people who are themselves fascinated by the opportunity and thoroughly conversant with what is being done on similar lines in other countries. People who are incapable of being thrilled will be of little value in this crusade. For crusade it will be. All sorts of vested interests pledged to complacent acceptance of things as they are will be arrayed against this attempt towards national healthfulness.

The architects of the future will have more to do than plan hospitals, these committees and medical and administrative executives will be doing much more than working in and administering hospitals. For this wider vision of the people's health service is already here in our midst and has begun to operate, though in detached units.

The Pioneer Health Centre at Peckham was certainly a pioneer, an adventurer into unknown areas. The work of this health centre will in the future be known as a remarkable historic milestone in the endeavour to educate the public towards healthful living.

I venture the suggestion that this preventive work is actually of primary importance, the health being a secondary matter.

But there is a hospital in this country which seems to have struck a new note and is one of the signposts along the health road of the future. The new American Red Cross Harvard Field Hospital Unit in England is actually three services in one group. This Unit is to deal with epidemics, and consists of over twenty buildings. One of the primary services is the mobile field unit of public health doctors and nursing staff ready for service where required in any part of this country. The next service is the pathological and research laboratories, exceptionally well planned and equipped and staffed to undertake work coming to it from all over Great Britain. The third service is that of the very modern hospital for 125 beds fully staffed and now in active work. The whole of the buildings, equipment, heating, cooking, and laundry plant have all been shipped across from the United States, and have been erected ready for service in the course of five months. The whole of the medical, nursing and technical staffs are Americans who have—not without loss of life—risked the journey across here to establish this very modern hospital. Dr. John E. Gordon, the Director, is Professor of Epidemiology at Harvard University, and has been responsible for the arranging and planning. Speaking as a layman, I consider this hospital is one of the most noteworthy developments in hospital service in this country.

But the new movement does not stop here, and it is an active town in Lincolnshire which may possibly have the honour of being the first town in Great Britain to consider health service in the widest sense.

Early in 1939 the Board of this hospital were considering certain extensions and in order to place these correctly in relation to the present and future requirements it was necessary to make a survey of all the hospital buildings and site and plot them to scale and then review the future possibilities for, say, the next thirty years or so. With these plans before them it was asked if certain land should be acquired adjacent to the existing site. It thus became necessary to discuss what might be necessary in a post-war hospital. The Board have realized the development of preventive medicine and with a fine vision have not limited themselves to the mere provision of hospital accommodation, but have gone further. Having in mind the work of the Pioneer Health Centre at Peckham, and other similar ventures, they have now under consideration a scheme which will have education in healthful living as its main incentive. The scheme—which is in its preliminary stages—will certainly mean extension of the hospital, a new nurses' home, kitchens, out-patients' department, maternity unit, modern research laboratories and other hospital necessities, but it will also have as its main feature—preventive and educative therapy in the form of clinics, lecture halls and classrooms, swimming bath, outdoor and

indoor gymnasia, and a variety of other amenities all leading to the enthusiastic teaching of how to live healthfully, both for young and old.

To bring this most advanced project into being, many modifications will be necessary and it can only be attempted in stages.

I have endeavoured in the foregoing to indicate some of the main trends and influences of the Past on the Present of hospital building. Based upon these I have attempted the somewhat thankless task of suggestions for the Future. We must cease to think merely of hospitals as places to educate young doctors, instruct nurses, inoculate guinea-pigs or as elaborate plumbing depots—they must be part of the educational facilities of a community, where education in healthful living is actually the mainspring of their activities. I would suggest that the development of these health centres is something bigger than the provision of mere "health-factories"—health houses or hospitals. To me the wire fence round the cliff top is more interesting than the more spectacular ambulance at the bottom.

Personally, I am getting somewhat tired of the insistence on curing disease. I want there to be very little disease to cure. I visualize the abounding health and happiness of the people as becoming the greatest future glory of civic life.

It may appear a strange thing for an architect to say, but we can attach too much importance to buildings. It is what happens in the buildings that is the really important thing. It is not the great buildings of the Scandinavian countries which mainly arouse our interest—it is primarily the splendid, vigorous health of the people, the buildings being the outcome of necessity and not of mere display.

We have accustomed ourselves to point with pride to our huge medical centres and hospitals, quite forgetting that the greater these are, the greater is the shame that such things should be required. I can see a city with its parks, swimming baths, gymnasia, schools, community halls, hobby and craft rooms, with its museums and lecture halls, in which people can be taught how to eat and drink, how to dress, how to employ their leisure: in short, how to live rightly; and all these linked up closely and intimately with preventive health clinics of all sorts—infantile, adolescent and the adult, and round the corner, somewhat ashamed of its existence, the hospital, to take all those unfortunates whom accident or disease has laid low. Instead of health becoming associated with disease it will thus become associated with healthy living.

Going through the streets of our cities and small towns we see many people standing about at corners, lounging in bars, hump-shouldered and shabby, and in an ignorant effort to pass time away spending their money on "that which is not bread".

Educated and uneducated, we are people of few enthusiasms—not very vigorous in body or mind—nor do we want to be. After this war will come that satanic lassitude—the desire to sit still and let things take their course. This will be the real devil we will be up against. It will take all the enthusiasm we possess to combat this influence which will insinuate itself into tired minds ready for its opiate effect. These community health centres which I have endeavoured to outline would go far to engage the enthusiasm of young and old, would give opportunities to become acquainted with those vigorous and healthy activities of mind and body which would, in a generation, beneficially affect the individual men and women of the future.

This is the Health City of the Future. So we return to that conception of a City of Health which we saw in its initial form in Caesarea in A.D. 369.

Though up the steep paths of the mountain of experience stumbled our ancestors, as we are stumbling now—out of to-day's unparalleled sacrifice of life and treasure—the men and women of the future need not stumble, but at last come out on that fair straight road—whereon the wayfaring man, even though a poor fool, cannot err.

DISCUSSION

Dr. J. Ferguson (Surrey County Council): Mr. Elcock has put forward one conception which I think all of us who are engaged in public health will welcome, and that is the vision of the preventive side of hospital work, making the hospital a preventive centre. That is an idea which I am delighted to hear publicly ventilated.

The 1929 Act really marks a tremendous stage forward in hospital policy, and, therefore, in the hospital designing of this country. Broadly speaking, it brings the public authorities into the very forefront of the picture, and into the forefront of the Bodies responsible for providing hospital accommodation for the country. Since the war of 1914-18, owing to various causes—the changing social habits of the country, the living

in small houses, the shortage of domestic servants, and the increasing complexity of medicine, leading to the difficulty of investigating and treating cases properly at home, there has grown up in accentuated fashion what one might call the hospital habit. For all these reasons the municipal hospitals and the municipalities can no longer be content with providing accommodation for the physical derelicts of the nation; they must provide for the same types of medical and surgical cases as the voluntary hospitals. Therefore, unless you are to do the unthinkable thing of erecting hospital provision only for those who can afford to pay, the public authorities must in the future provide hospitals of the same standards of excellence as the voluntary hospitals. Therefore, one may expect to find a considerable and rapid development of new hospitals in the country as a whole.

I will say nothing about the Nuffield Trust; I will say nothing about the very much debated subject of Regionalization, except this, that large municipal authorities, like the London County Council, like Middlesex, and like Surrey, already have regional schemes in existence, with central purchasing, central direction of the patients to the appropriate hospitals, and so on. So much for policy.

One of the main contributions which Mr. Elcock has made to hospital design is the idea of the parallel bed. It is a very interesting idea indeed. It is an idea which was very fully explored by the Departmental Committee on Hospital Building Costs; but the conclusion which that Committee came to was that the evidence was not at that time great enough to come down whole-heartedly in favour of it to the exclusion of the projecting bed principle. The idea, however, is very attractive. I am going to pass round actual photographs of a hospital which the Surrey County Council has just built, an 860-bed hospital, which will be complete about April 1; and some of which is even now occupied. It is an interesting partial embodiment of Mr. Elcock's idea. A certain number of the beds, 12 in each ward unit of 30 beds, do face south, with beds which are parallel to the southern aspect of the hospital.

The more I see of the building of hospitals the more strongly convinced I am that it is vital to have specialist architects in order to obtain the best results. No doctor can in fact think of all the things which go to the making of a hospital. I urge, therefore, the most complete co-operation between the architect, the doctor and the matron, representing the nursing people, if the best results are to be obtained.

Mr. R. C. Harkness, F.R.C.S.: I would like to mention two aspects of importance. These are size and location. We have voluntary hospitals and municipal hospitals. The latter are a complex set of hospitals. Municipal general hospitals had their origin in the sick wards of workhouses and were at first almost entirely chronic hospitals, but now do much acute work. That has led in some cases to a dividing off of the chronic from the acute work. Other most important groups are the tuberculosis hospitals, which received their main impetus from National Insurance; and the fever hospitals, which were in existence somewhat earlier. The tuberculosis hospitals and the fever hospitals have been managed under public health powers. These sanctions coming at different times are largely responsible for these hospitals being in separate groups. Many consider that something should be done to link the various types of patients in one hospital. There are difficulties in getting the best medical staff, because of the narrow line of specialism and the slowness of promotion. It is sometimes held that medical staffs who treat special forms of disease can do their work better in close contact with those treating other forms of disease. If all these different forms of hospital work are to be brought together we must have large hospitals. Similar considerations arise in another way. Surgery, for example, can only be done to a proper standard if you have enough of it in one place.

The other important subject I want to refer to is the situation. Mr. Elcock has expressed the desire that hospitals should not be built in congested urban districts, but should be put on the outskirts. There are, however, other things which must be borne in mind, especially in big towns. Once when I was superintendent of a hospital I had an inquiry from the Ministry of Health asking: "I want you to explain why it is that your hospital admits x per thousand patients from the population you serve, whereas another hospital admits only half of x per thousand of its population." I replied: "My hospital is in the middle of the district which it serves." The other hospital was a more modern building on the outskirts but the patients tried to get into the hospitals nearer their homes.

Years ago a large voluntary hospital in a provincial town opened a new hospital on the outskirts. It was soon discovered that its patients were going to the voluntary hospital in a nearby municipality: subscriptions were going the same way. That is a thing which has to be remembered, I think. In war-time we can send patients where we like. When we get back to times of peace, I think we may find that it is not going to be so easy. There is a danger that we may build a hospital on the fringe of London and then find that it is impossible to use it for the purposes for which the original hospital was used, because the patients have drifted away.

Mr. W. E. Brooks, F.R.I.B.A. (London County Council): The L.C.C. has about 150 hospitals and institutions, apart from the numerous clinics, nursery and open-air schools under its charge. The hospitals are transferred buildings under the Act of 1929, and a similar survey was taken in London to that which Mr. Elcock tells us he carried out in Hertfordshire.

This survey revealed that many of the buildings might well have been called "Houses of Pity"; but pity, I mean for the doctors, nurses and staffs who had to run them. The state of some of them was very unsatisfactory, but that did not apply to all, for numbers of them were, of course, first-class hospitals, and the bad ones are now very much better. Old wards in many cases have been turned about and divided up, as Mr. Elcock has shown, providing what he has termed "veranda-wards", with their consequent advantages.

Most of the hospital work at the L.C.C. has been in improving the transferred buildings; but many opportunities have arisen where new buildings were required, including new nurses' homes—which have been provided.

One point that interested me perhaps more than any other was in Mr. Elcock's reference to the health services and their effect on the national health, such as health centres, nursery schools, clinics, and other similar services.

We have built many of these smaller buildings: the medical room in the school, the clinics and nursery schools on the housing estates; camps and open-air schools for fresh-air treatment; all in consultation with the Council's medical advisers. In these we hope to teach mothers and children the elements of a healthy state of living. But we cannot stop there. Too many unhealthy and inconvenient houses still exist all over this land, and much of the good work done in the health centres is undone in the home itself.

When the war is finished, we may hope that this last and most important aspect will be tackled with all the energy that we can muster. I can speak with actual knowledge of the large amount that still remains to be done in London. The basis of good health lies in good, clean homes, with modern sanitation and cooking apparatus, together with the means of obtaining and understanding the use and preparation of proper food and suitable clothing.

"Buildings do not create a way of life—they only reflect it", and the work of the architect lies mainly in the interpretation of a developing social order in relation to the time in which he lives. He will provide the buildings, but it is for the doctors, nurses and teachers to educate the people to live in them.

Dr. W. A. Bullough (Essex County Council): I shall refer only to one important matter: the treatment of infectious conditions in general hospitals.

Research has thrown entirely new light on the way such infections should be treated. We have not implemented this knowledge in the design and construction of our hospitals. We have deliberately ignored the fact that persons may be admitted to hospitals in an infective condition or during the incubation period of such infective condition. This mainly concerns infants and children, who from time to time suffer from waves of measles, chicken-pox, whooping-cough, scarlet fever and the like. When these occur among children admitted to hospitals, we have dealt with the situation by various methods, including closing the wards and other stop-gap devices.

The additional expenditure in making the necessary provision will undoubtedly add to the cost of the buildings; but I am sure that in a short time, purely as a financial measure, it will be proved to be very sound. Apart from that, it is our duty to the patients to see that they are not exposed to avoidable infection.

We have in Essex an Epidemiological Committee which meets every month. The members have devoted a great deal of time to this subject, on which they have prepared a memorandum which has been sent to every hospital in Essex. The proposals are designed to prevent the introduction of infection into the wards; and in the case of failure, to control infections which have passed that barrier. They are briefly:

A reception room, with one or more cubicles and facilities for full examination, especially of the throat. The staff in that room would consist of a reception room medical officer, reliable and experienced, who would have full authority to deal with all admissions, under the supervision of the medical superintendent. He must, of course, have bacteriological facilities available.

Next, an observation ward, which should be cubicalized—the method of cubicalization depending on the materials available. Its size would depend on the size of the hospital. Here again, the medical officer would probably be the reception room medical officer, and would be responsible for the cases, which would be allocated to the appropriate physician or surgeon. The nursing staff in the observation ward must be experienced in barrier nursing and the nursing of infectious diseases. Full precautions would be observed re crockery, &c.

An infectious diseases ward and a septic ward, which again must be cubicalized,

would be provided. The size of this ward would again depend on the size of the hospital. It would have a small operating theatre attached to it.

All new admissions would be examined in the reception room by the reception room officer, who would admit to the infectious diseases ward any patient found to have any infectious condition, or any case in which diagnosis of infectious condition is in doubt. Any frank infectious condition would be transferred to the local isolation hospital. Thus the spread of infection would be limited owing to the fact that appropriate cases were either kept in the observation ward or passed on to the infectious diseases ward as required.

It is suggested that the number of beds in the observation and infectious diseases ward should be equivalent to 10% of the total beds of the hospital. In hutted hospitals a single hut might be divided, the two halves being used as an observation and infectious diseases ward respectively. In smaller hospitals side wards, or even cubicles, could be earmarked for these purposes.

These recommendations would have to be supplemented in many other ways to prevent the spread of infection—e.g. supervision over visitors, librarians.

With regard to the adult wards, it would be reasonable to cubicalize 15% of the beds, including the segregation of adults for other purposes. For the children's ward we suggest 30% of the beds should be cubicalized; this is rather a big figure, but from recent experience in Essex we think that nothing less can effectively control infection in general hospitals.

Dr. H. M. C. Macaulay (County Medical Officer of Health for Middlesex): There is one remark which Mr. Elcock made which should be underlined, namely, that the internal walls of his health factory should be capable of re-modelling without interfering with the main structure. It seems to me that the architect, as it were, crystallizes in a static form current ideas of medicine. But medicine is not static; it is vitally dynamic and the hospital which is up to date to-day, may be very far below our expectation in ten years' time.

Mr. Elcock's remark that the hospital of the future should be kept in as fluid and as elastic a shape as possible is one which should be endorsed. If our architect colleagues can give effect to that policy it may have a profound influence, not only on the structure of wards, but on out-patient departments and other buildings; and for years to come the hospital may be able to keep pace with the changes wrought by medical science.

With regard to prevention of disease it is a far easier thing to elicit sympathy for ministering to the care of the sick, than it is to enlist support for schemes of research and prevention. Curative medicine in its human aspect touches a very deep spring in human emotion, whereas the preventive side is a much more cold and calculating business. The cure of a single case of typhoid, by the exercise of medical skill and patient nursing may bring more gratitude than keeping a whole nation typhoid-free. It was with some such thoughts in mind that I listened to the remarks made by Mr. Elcock on the predominant part which, in his view, preventive medicine should take in any scheme in days to come. One is emboldened to hope that his view is shared by other enlightened laymen, and may in time come to be held by the public at large.

We medical officers of health who have had the administration and organization of hospitals placed upon us in recent years should perhaps realize that there is a danger of relegating to the background those preventive methods which are really the primary justification of our existence. Efforts have been made to link up the municipal hospital service with the preventive side of our work: but one of the difficulties with which we are faced is the fact that hospital medical officers are less interested in the preventive than in the curative side. Their training and experience lead them into the paths of the cure of disease. The training of medical students needs radical revision. It is still the usual practice in our medical schools for the whole field of preventive medicine to be covered by a series of perhaps a dozen lectures. Until social medicine is given the place it should have in the medical curriculum, we cannot expect to get the kind of preventive measures which the people of this country deserve.

Mr. C. D. Andrews, F.R.I.B.A.: Mr. Elcock referred to the "white collar" people and I would like to point out the treatment available for the "white collars" of Middlesex. These people have easy access to a most excellent municipal service at a graded fee according to income, the maximum being about £5 5s. per week. This is in hospitals costing £800 to £1,000 per bed.

The veranda-ward calls for a major discussion where many points of view could be expressed.

A previous speaker mentioned the necessity of the specialist architect; there is also the need for the specialist doctor, i.e. one who has studied planning extensively. This

need is reflected in a large number of hospitals which in first cost and in upkeep are unworthy of modern medicine.

Mr. Howard Robertson, F.R.I.B.A.: I was interested in what Dr. Bullough said about hospitals on the perimeter of towns. There is evidence, though this is perhaps a little away from Mr. Elcock's thesis, that there may be many towns, in which should be hospital centres.

One small point has relation to Mr. Elcock's allusion to the "Health Factory". As an architect, I would like to make a passing plea for the health hotel, as opposed to the health factory: because there is a great deal to be done through the form of architecture in the treatment of patients. I think there is a psychological effect of form and shape which we must develop. Perhaps when we have got through the factory phase, we may use architecture almost as a curative medium, quite apart from its functional purpose.

Mr. Duncan Fitzwilliams, F.R.C.S.: All hospitals need recreation rooms where patients can go and sit, read and play games. They should also have dining rooms. All military hospitals in the last war had this accommodation, but the lesson was lost when peace was declared.

The better to illustrate some curious features of hospital life, I am going to suppose that Mr. Elcock is ill and I am going to have him nursed in these pleasant wards which he has designed for the pleasure and comfort of the patient. It is his first night in hospital and he is in strange surroundings. There is a screen around the next bed and the nurses pass hurriedly to and from it. The Resident comes and whispers to the Sister. There is an air of gloom in the ward and the other-inmates are strangely silent. Presently relations arrive and weep and Mr. Elcock realizes that some poor person is dying within four feet of his bed. Sights, horrible sounds, and even more unpleasant things assail his senses, and appal his strained imagination. Few people apart from the medical profession are familiar with death.

If there are any things which are private, birth is one and death is another. The dying person should have been removed to the privacy of a small ward where he can pass beyond the veil with his family around him without harrowing the feelings of twenty other people in a big ward.

All cases on the danger list should be removed from the big wards at once—they require quiet and many other things which can hardly be given without incommoding other patients. There should be no such things as "death wards"; these are only the much-needed facilities for nursing the seriously sick. If they die, they do so decently; if they recover they return to the large ward, which should always be kept bright and cheery.

The next experience is just as repugnant. Mr. Elcock's meal has just been brought him; the kindly nurse coaxes him to eat, but is called away by his immediate neighbour who says something in her ear. A screen is put round the bed and a bedpan is surreptitiously introduced behind the screen. I will leave Mr. Elcock wishing him a good appetite.

Modern hospital beds have large wheels to enable them to be wheeled out on to the verandas, balconies and solariums, which are now considered the normal equipment of hospitals, and which Mr. Elcock has so well elaborated. Those same beds can be wheeled out, just as easily, to the lavatory accommodation which should be provided. In this lavatory these functions can be performed, enemas given and colostomies attended to.

Or again: Mr. Elcock has been admitted for some throat operation and, as he lies in bed, he sees a patient wheeled in from the theatre, still semi-conscious, vomiting and spitting blood into an ill-adjusted bowl which only receives part of what it is intended to contain. The whole spectacle is as disgusting as anything that can be imagined. On inquiry it is learned that it is "only a throat operation".

Recovery wards should be arranged close to the theatres where patients can be housed behind low cubicles so that the nurses passing down the centre can readily keep an eye upon them. Sexes do not matter in these circumstances as patients in this condition are sexless.

Suppose a head accident is admitted, restless, crying out and making such a din that no one can sleep. If the nurse leaves him she is called back by the other sleepless patients as the patient is attempting to get out of bed; or a patient suddenly becomes delirious from illness or alcohol; or a patient may become maniacal. There is little sleep for the sufferers in that ward that night; or possibly on many following nights. No London hospital has a ward where such noisy patients can be accommodated, though in Scotland they have long been considered a requisite part of the hospital.

It is the architects who must devise and carry out these reforms.

Mr. C. E. Kindersley, F.R.C.S.: I should like to emphasize one or two points that Mr. Elcock has brought out in his designs, the first being that as a primary principle a hospital is a building for a scientific purpose and should be designed to that end. Secondly, that the motto of hospital design should be: "that which has to be done most often should be done most easily"—a motto often ignored by architects. To illustrate this I can quote a ward kitchen placed, according to an architect's designs, and even approved by the Ministry of Health, 35 yards from the ward door and 52 yards from the furthest bed, and it took a lot of trouble on the part of the medical staff to have this defect remedied. I have seen a sterilizing room attached to a ward beautifully fitted, but with no space for the sterilizer where it could be provided with the necessary drainage and supplies, and wards designed with nowhere to put the essential basin. These are major crimes. Thirdly, Mr. Elcock mentions an all-important factor in hospital design which he has called "traffics". The whole hospital design should be determined by traffics, and these traffics are not confined to the main corridors only; each department has its own traffics as well. Few appreciate that an operating theatre unit has at least five separate traffics in it—a point too seldom realized by those responsible for its design. A further suggestion he has made is that vertical traffics are better than horizontal traffics whenever possible.

Let a hospital architect attend to his design with the same detail that he would expect of a surgeon operating upon himself. The plea for health factories rather than hospitals should be considered, and conferences concerning hospital buildings and service should be called as soon as possible.

The Chairman: I think that Mr. Elcock has given us a sane, sober and constructive view of hospitals, and a wide vision for the future.

There are three points which struck me about his talk. The first is that the hospital should be a place of prevention. The late King Edward, speaking of tuberculosis, said: "If preventable why not prevented?"

With regard to historical events, Mr. Elcock referred to Rahere as being the reputed founder of St. Bartholomew's Hospital. St. Bartholomew's Hospital has been going since the year 1123. Rahere was prior of the Order of St. Bartholomew. His tomb is near by in the great Priory Church; and probably he had to do with the founding of the hospital.

I think that the hospital wards of the future should be built with sliding windows and veranda-wards and parallel beds.

When the Heart Hospital in Westmoreland Street, was designed, plans were drawn by a colleague of mine, Sir Sidney Russell Wells, afterwards Vice-Chancellor of the University of London, and myself. Those plans went out with the name of an architect on them because we did not want to put on our own names; but they were drawn exactly to our scale plans. We put the hospital kitchen on the roof because we had transport in mind and we also wanted to avoid the smell of cooking.

Post-war planning should begin now with conferences of those who are likely to be actively employed and interested in hospital building and post-war planning in general.

It would also be a useful thing for hospitals to be inspected at regular intervals because they vary a great deal in efficiency.

Mr. Elcock (in reply) stressed the urgent importance of conferences between medical men, the nursing and administrative services and architects, so that certain standards might be arrived at for future hospital and health services planning. This should lead to simple and direct planning with recognized suitable equipment, and economy in capital outlay and in annual administrative costs.

Section of Obstetrics and Gynæcology

President—J. M. MUNRO KERR, LL.D., M.D., F.R.C.O.G.

[January 16, 1942]

Progress in Obstetrics and Gynæcology During the Present Century

PRESIDENT'S ADDRESS

By J. M. MUNRO KERR, LL.D., M.D., F.R.C.O.G.

THOUGH the title of my Address "Progress in Obstetrics and Gynæcology During the Present Century" may appear to be commonplace the subject itself certainly is not so.

THE PAST

At no period in its long history has obstetrics, and its sister branch gynæcology, made as great progress as during the present century. The leap forward, in which I and my contemporaries participated and which a younger generation is expediting, was not due to the efforts of obstetricians alone. It resulted in great measure from assistance we received, *first*, from scientists, (biologists, physiologists, biochemists, pathologists, bacteriologists) and, *secondly*, from the Public Health Service. I propose to develop later in my Address the theme that the closest possible co-operation by obstetricians, scientists and the Public Health Service should be the policy of the future, as only by this means will we ensure that progress in obstetrics and gynæcology does not lose momentum.

Let me briefly summarize the advances made during the first forty years of the present century. We speak of the *science* and *art* of obstetrics and gynæcology—let us take firstly progress in the science of our subject. At the beginning of the century we knew very little about "endocrines". To-day the profound influence their hormones exert on the growth, the nutrition, the mind, and even the character of the individual has in great part been revealed. Naturally obstetricians and gynæcologists are most interested in the influences exerted by the hormones of the gonads, of the pituitary gland, of the thyroid, and of the suprarenals, on the menstrual cycle; and the almost immediate activity aroused in them when an ovum is fertilized, as evidenced in the Aschheim-Zondek test. At the moment we can only speculate on the part they play in the growth of the uterus during pregnancy, in the activity of the uterus during labour, and in the involutionary processes of the puerperium. We have to admit, also, that application of our knowledge to practical problems has been disappointing. Hormonal therapy for functional disturbances of the reproductive system, apart from pregnancy, has not given the results hoped for. Possibly a little more encouraging are the results in certain dis-

Mr. C. E. Kindersley, F.R.C.S.: I should like to emphasize one or two points that Mr. Elcock has brought out in his designs, the first being that as a primary principle a hospital is a building for a scientific purpose and should be designed to that end. Secondly, that the motto of hospital design should be: "that which has to be done most often should be done most easily"—a motto often ignored by architects. To illustrate this I can quote a ward kitchen placed, according to an architect's designs, and even approved by the Ministry of Health, 35 yards from the ward door and 52 yards from the furthest bed, and it took a lot of trouble on the part of the medical staff to have this defect remedied. I have seen a sterilizing room attached to a ward beautifully fitted, but with no space for the sterilizer where it could be provided with the necessary drainage and supplies, and wards designed with nowhere to put the essential basin. These are major crimes. Thirdly, Mr. Elcock mentions an all-important factor in hospital design which he has called "traffics". The whole hospital design should be determined by traffics, and these traffics are not confined to the main corridors only; each department has its own traffics as well. Few appreciate that an operating theatre unit has at least five separate traffics in it—a point too seldom realized by those responsible for its design. A further suggestion he has made is that vertical traffics are better than horizontal traffics whenever possible.

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With regard to historical events, Mr. Elcock referred to Rahere as being the reputed founder of St. Bartholomew's Hospital. St. Bartholomew's Hospital has been going since the year 1123. Rahere was prior of the Order of St. Bartholomew. His tomb is near by in the great Priory Church; and probably he had to do with the founding of the hospital.

I think that the hospital wards of the future should be built with sliding windows and veranda-wards and parallel beds.

When the Heart Hospital in Westmoreland Street, was designed, plans were drawn by a colleague of mine, Sir Sidney Russell Wells, afterwards Vice-Chancellor of the University of London, and myself. Those plans went out with the name of an architect on them because we did not want to put on our own names; but they were drawn exactly to our scale plans. We put the hospital kitchen on the roof because we had transport in mind and we also wanted to avoid the smell of cooking.

Post-war planning should begin now with conferences of those who are likely to be actively employed and interested in hospital building and post-war planning in general.

It would also be a useful thing for hospitals to be inspected at regular intervals because they vary a great deal in efficiency.

Mr. Elcock (in reply) stressed the urgent importance of conferences between medical men, the nursing and administrative services and architects, so that certain standards might be arrived at for future hospital and health services planning. This should lead to simple and direct planning with recognized suitable equipment, and economy in capital outlay and in annual administrative costs.

operator when radium displaced hysterectomy, for the performance of a perfect Wertheim operation calls for very great technical skill.

Operative technique in both obstetrical and gynæcological surgery has made enormous strides in this century. When I was undergoing post-graduate training in Dublin, Berlin and elsewhere in the nineties—"the naughty nineties"—an aggressive *antiseptic* technique was employed. Many years passed before gloves and masks were introduced and an aseptic technique was perfected. The surgical technique of those days compared with that of to-day is as different as black from white. Consider the improvements in technique in such operations as the modern lower segment Cæsarian section, and the attention now paid to minute details in hysterectomy, myomectomy, salpingectomy, plastic operations for uterine and vaginal prolapse. Then again take the progress in the administration of anæsthetics which until recent years was considered by the family doctor to be his prerogative—to-day he does not desire to assume this responsibility. Specialists by perfecting anæsthetic technique in all its forms, have not only facilitated the surgeon's work, they have made possible operative procedures undreamt of early in the century. Very definitely, too, by careful choice of anæsthetic they have played an important part in bringing about the progressive lowering of the death-rate for all operations which this century has witnessed—here I would stress the great future for *block analgesia*.

I am convinced that the visits to clinics at home and abroad by both the Senior and the Junior "Visiting Gynæcological Society" have been of immense value to the members themselves, and to British obstetrics and gynæcology in general. The same can be said for our own special Journal. Under the direction of its Editor, Sir Comyns Berkeley, *The Journal of Obstetrics and Gynæcology of the British Empire* has been raised to a level equal to that of the best American and European journals in our speciality. The abstracts of current obstetrical and gynæcological literature, for which Frederick Roques is responsible, deserves special commendation. Furthermore, through our journal other countries have come to learn something of the great traditions and the cautious progress of British Obstetrics and Gynæcology.

But probably the greatest of all the corporate developments of the century, in our field of medicine, was the founding of the Royal College of Obstetricians and Gynæcologists. I wish to put it on record that this development of recent years was contemplated and discussed with me, when I was quite a youngster in obstetrics, by Sir Japp Sinclair, the founder of *The Journal of Obstetrics and Gynæcology*. But for all time the name of Blair Bell will be honoured as the founder of our College. His great benefactions and those of Lord Riddell to the College will ever be remembered with gratitude. Nor should be forgotten the services rendered by Fletcher Shaw, the indefatigable Secretary of the College during its years of protracted gestation and neonatal life, and now its President.

Another corporate development affecting our speciality is the Midwife Service which has been built up following the Midwives Acts of 1902, 1918 and 1926. I take this opportunity to extend to the Midwives Institute our congratulations on its new title—The College of Midwives.

To complete this summary of corporate advances I must mention once again the Public Health Service, whose serious invasion into our domain followed The Notification of Births (Extension) Act of 1915. As you are aware, by this and subsequent Acts, and especially by the Local Government Act of 1929, local authorities have been entrusted with the responsibility of ensuring that the maternity services of the country are satisfactory.

THE PRESENT

Putting aside such questions as the toxæmias of pregnancy, the treatment of functional diseases, and improvement in surgical technique already referred to, the problems which concern us more particularly at the moment are:

- (1) The maternal death-rate and disability resulting from pregnancy and childbirth.
- (2) The stillbirth and neonatal death-rate.
- (3) Abortion.
- (4) Birth control.
- (5) Falling birth-rate.

turbances of pregnancy; but even here it cannot be claimed that much has been achieved so far.

Another great service, which scientists have rendered, is the proof they have furnished of the value of vitamins. Here practical application has followed close on the heels of laboratory findings.

Long before vitamins were discovered there was much discussion on the influence of diet on the size of the foetus *in utero*—in these days the majority believed that the size of the foetus in many instances, but to a varying degree, could be influenced by the food taken by the mother during pregnancy.

As I was desirous to obtain expert opinion *re* the possible injurious effect of excess of vitamins on (a) the individual in ordinary life, (b) the pregnant woman (more particularly as it might affect the size of her child), I wrote to Sir Edward Mellanby who passed on my letter to Dr. T. Moore (Director of the Dunn Nutritional Laboratory, Cambridge). Dr. Moore in his reply said:

(a) "As far as the first question is concerned, I think the risks are very slight. Even in experimental animals only vitamins A and D are known to be toxic when given in great excess.

"There is ample proof that excess of cod-liver oil is sometimes injurious to animals, although it is improbable that the toxicity is due to the vitamin content. In common with other marine oils, cod-liver oil seems to reinforce the effect of vitamin E deficiency in causing either muscular dystrophy or the interruption of gestation. . . .

"I myself would be against giving massive doses of cod-liver oil in pregnancy, and particularly so if the diet was low in vitamins B and E.

(b) "Excess of vitamins will not raise the rate of growth above normal, although deficiency may often reduce growth below normal.

"If an abnormal pelvis makes it imperative in expert opinion to attempt to limit the size of the foetus by dietary methods, the reduction should be made only in the quantity of food consumed. Particular care should be taken to ensure that the vitamin content does not suffer as a result of the quantitative reduction."

This brings me to express my disappointment that more has not been accomplished to lessen the deaths from the "toxæmias of pregnancy". I cannot discover that we are much nearer a solution of the ætiology of *eclampsia* than when I attended Professor Leishman's lectures at Glasgow University over fifty years ago. In the latest edition (1941) of "William's Obstetrics", Stander, who has devoted great thought and much time to investigating the problem of *eclampsia*, is not prepared to do more than summarize the various theories which have been presented at different times. Possibly we see a little more clearly in respect to *hyperemesis gravidarum* which if caught early can almost always be arrested. Hypertension, persistent albuminuria, and the danger of chronic nephritis resulting therefrom, call for further investigation.

Radiography in obstetrics has made possible exactness of diagnosis, in respect of pelvic formation, position and attitude of foetus, size of foetus, plural pregnancy, malformations of foetus, condition of the ureters and so on. In respect to minor pelvic abnormalities which are very common, great caution is necessary in determining for or against Cæsarian section from a pelvic radiograph. In the days preceding labour, and especially early in labour, there take place relaxation of the pelvic joints, little adjustments of the foetal head to the pelvis and moulding of the foetal head, which cannot be estimated before labour. Then again the strength of the forces cannot be predicted. Thus in many instances the most rational and scientific procedure is to allow a "trial of labour". Only by long clinical experience can the obstetrician learn to determine in particular cases whether a "trial of labour" is indicated or not, and to what extent the trial should be permitted to continue.

Radium and deep X-ray therapy has revolutionized the treatment of carcinoma of cervix and that troublesome condition of chronic subinvolution (chronic metritis?) of the uterus. Mention of carcinoma of cervix recalls to mind the perfection of technique developed by a number of operators in this and other countries when the radical (Wertheim) operation was the vogue. I have never been convinced that this wonderful operation should be discarded and entirely replaced by radium. It was very fortunate for the indifferent

fœtus in pregnancy, vaginal manipulations in labour to bring the fœtal head into a more favourable position (which Smellie describes so graphically in his case records), gentleness in forceps delivery, breech extraction (associated with a very high fœtal mortality), and manual removal of placenta (frequently followed by shock and even death of the mother). Those and other little details of obstetric technique can only be learned by long apprenticeship and by constant practice.

(c) *Associated deaths*: In this group the death-rate has shown no improvement in recent years.

| Years | Death-rate per 1,000 live births |
|---------|----------------------------------|
| 1925-30 | 1.21 (average) |
| 1936 | 1.10 |
| 1937 | 1.24 |

It is unlikely that there will ever be pronounced reduction in the death-rate in this group. Undoubtedly careful supervision of pregnancy and institutional treatment under experienced obstetricians and physicians would bring about a reduction in the deaths due to influenza, pulmonary tuberculosis, heart disease, chronic nephritis, &c., included in this group.

Maternal disability: There is no means by which we can compute with any degree of exactness the injuries, chronic infections of uterus and tubes, functional disabilities and ailments due to pregnancy and childbirth. We gynaecologists know, however, from the patients admitted to our wards or seen as out-patients, and from odd cases seen in consultation in the wards of surgical and medical colleagues, that a very considerable number of women are temporarily or permanently disabled.

McIntyre in an analysis of 7,734 patients treated in the Royal Samaritan Hospital, Glasgow, found that the ætiologic factor was:

| | | | |
|---------------------|-----|-----|-------|
| Puerperal infection | ... | ... | 28.1% |
| Trauma childbirth | ... | ... | 35.3% |

This unfortunate legacy from childbirth can only be reduced by more assiduous antenatal and intranatal care—the aphorism that “good obstetrics is preventive gynaecology” contains more truth than most aphorisms.

(2) *STILLBIRTHS AND NEONATAL DEATHS*.—During the present century the infant death-rate for England and Wales has fallen from 140 to 35 per 1,000 live and stillbirths; but the number of stillbirths and neonatal deaths has diminished only very slightly. Here is the position as presented in the last “Text” Volume of The Statistical Review of the Registrar-General of England and Wales (1937, Table XIV, p. 34).

RATES PER 1,000 LIVE AND STILLBIRTHS.

| Year | Stillbirths | Neonatal death-rate (ages 0-4 weeks) | Combined rate |
|------|-------------|-----------------------------------------|---------------|
| 1928 | 40.1 | 29.8 | 69.9 |
| 1929 | 40.0 | 31.5 | 71.5 |
| 1936 | 39.7 | 29.0 | 68.7 |
| 1937 | 39.0 | 28.6 | 67.6 |

We must set ourselves to lower the stillbirth and neonatal death-rates. The 8-10% of deaths due to malformations possibly cannot be lowered; but the fœtal deaths from prematurity and injury during birth can be reduced materially by improved antenatal and intranatal care, to which I have already made reference. I recommend a perusal of a recent article (*Lancet*, 1941 (ii), 657) on the subject, by Dugald Baird and John F. B. Wyper of Aberdeen; and the Commentary on it (*Lancet*, 1941 (ii), 746) by R. H. Titmuss, who promises a statistical study of the subject at an early date.

Here let me stress the importance of *research of clinical and practical problems* which are peculiarly the province of obstetricians and gynaecologists. The more intricate problems which can only be investigated in biological, chemical and biochemical laboratories have grown beyond the scope of the majority of practising obstetricians and gynaecologists. By all means encourage them to gain a practical knowledge of laboratory methods and carry out simpler laboratory investigations, but few young specialists can give time to

(1) THE MATERNAL DEATH-RATE.—You are aware that the annual death-rate published by the Registrar-General includes deaths from: (a) Sepsis; (b) other diseases. It does not include (c) "Associated" deaths defined as "Deaths of women not classed to pregnancy and childbearing but returned as associated therewith". Let us consider the present position as regards each of these categories.

(a) *Sepsis*: It is cause for great satisfaction that there is an appreciable fall in the death-rate from sepsis in the last year or two. Here is the position for England and Wales.

| Years | Death-rate per 1,000 live births |
|---------|----------------------------------|
| 1925-30 | 1.7 (average) |
| 1936 | 1.39 |
| 1937 | 0.98 (lowest on record) |

How much further the death-rate from "sepsis" can be reduced is impossible to predict. The rate might in some years rise again should epidemics of infectious diseases develop, e.g. outbreaks of influenza; for epidemics of infectious diseases in the past have affected the death-rate from puerperal sepsis.

The pronounced lowering of the death-rate from sepsis is due to several factors, of which the following are the most important: (1) The drugs (sulphanilamides) now in our hands have furnished a weapon of inestimable value for combating infection should it develop. (2) A more thorough aseptic technique is being practised in the country generally. (3) Midwives who are responsible for so many deliveries in the country are a much better trained and more efficient body of women than ever before. Whether the fact that an increasing number of women are being treated in maternity hospitals may also be a factor cannot at the moment be determined. I, personally, am chary about making a statement on this point, because I am definitely in favour of institutional treatment as far as this is possible, and certainly for all primigravidae.

(b) *Other diseases*: In this group improvement has been disappointing.

| Years | Death-rate per 1,000 live births |
|---------|----------------------------------|
| 1925-30 | 2.5 (average) |
| 1936 | 2.41 |
| 1937 | 2.28 |

The toll of deaths from the toxæmias, hæmorrhages, and complications and accidents of labour (which include the bulk of the complications in this group) is much too high. All engaged or interested in obstetric practice are surprised and disappointed that in spite of the many antenatal clinics established by Local Authorities there is no marked improvement in the death-rate from eclampsia and other toxæmias in the country generally. I certainly had expected that by this time deaths from *eclampsia* might have been reduced by 20-30% at least. The reason is not far to seek. Antenatal care is still inadequate. The medical staffing of antenatal clinics under local authorities is in a great number of instances far from satisfactory. The individuals responsible for such clinics are out of touch with the practice of obstetrics as ultimately the patients are attended by midwives and/or other doctors—I shall refer later to remedial measures to correct this weakness in the antenatal services.

The deaths from hæmorrhages due to *placenta prævia*, and *accidental hæmorrhage* might well be lowered if the patient were immediately placed in an institution staffed by obstetric specialists on the first evidence of hæmorrhage or other untoward symptoms such as albuminuria, and heightened blood-pressure, which are so often the precursors of "accidental hæmorrhage". I need not remind you that in the interest of mother and child alike Cæsarian section is now accepted as the operation of choice for "placenta prævia", unless encountered in its simplest form.

Then take the deaths from *accidents and complications of childbirth*. Here again delivery in an institution by specialists would reduce enormously deaths from these causes. Some of the conditions responsible for these deaths can be recognized during pregnancy, e.g. pelvic disproportion; others, e.g. occipito-posterior positions of vertex may develop during labour. Many of the fetal deaths are definitely due to faulty technique on the part of a doctor inexperienced in obstetric practice. There is room for improving purely obstetrical procedures such as manual correction of faulty position of

fœtus in pregnancy, vaginal manipulations in labour to bring the fœtal head into a more favourable position (which Smellie describes so graphically in his case records), gentleness in forceps delivery, breech extraction (associated with a very high fœtal mortality), and manual removal of placenta (frequently followed by shock and even death of the mother). Those and other little details of obstetric technique can only be learned by long apprenticeship and by constant practice.

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the long and arduous training this type of research entails—they will always remain amateurs. Possibly in the future—it has been suggested—there may be established in the country one or two large maternity units complete with clinical material (beds), and laboratories for every type of investigation. I wonder if it is feasible, or necessary, or advisable to attempt so ambitious a scheme!

(3) ABORTION.—There are a number of problems connected with abortion which are interesting obstetricians and gynaecologists at the present time. Take as examples—the influence of hormonal irregularity and vitamin E deficiency, the great loss of foetal life, the progressive rise in criminal abortions and its influence on maternal mortality and morbidity, the legality of “therapeutic” abortion. Obviously I cannot do more than enumerate these problems, but I would like to express my convictions on three points: (a) That it is undesirable to press for “notification of abortion”; (b) that it is advisable to leave the Law governing abortion *in statu quo*. With the position as it is at present there is a freedom of action left us which we would never enjoy if the circumstances under which the performance of therapeutic abortion were defined by Act of Parliament; (c) Few if any of us are prepared to recommend extending the scope of the operation on economic or compassionate grounds as represented by the patient. There is still fresh in our minds the disastrous results following the free employment of induction of abortion in U.S.S.R.—results so disastrous that U.S.S.R. gave up the experiment.

(4) BIRTH CONTROL.—A dangerous development of the century is the extension of the practice of birth control. Not for a moment do I maintain that birth control is not justifiable by individuals in certain circumstances; but its free employment constitutes a danger to the country in which it is practised to anything but a moderate degree. That leads me to refer to the falling birth-rate.

(5) FALLING BIRTH-RATE.—Here is the position for England and Wales:

| Years | Birth-rate per 1,000 of population |
|---------|------------------------------------|
| 1881-90 | 32.4 |
| 1921-30 | 18.3 |
| 1939 | 14.9 |
| 1940 | 14.6 |

The danger for the nation is obvious. The Royal College of Obstetricians and Gynaecologists is alive to the danger. Eardley Holland represents the College on “The Population Investigation Committee”. In the brochure entitled “Population and Fertility” issued by the Committee in 1939 under the Editorship of D. V. Glass and C. P. Blacker the following statements are made:

“The more closely the problem is studied, the clearer does it become that, in fact, the causes of the decline in fertility are numerous, complex and deep-seated. In Western European countries, they form so intimate a part of what we may call the twentieth-century view of life, that we can prophesy with some confidence that really effective counter measures will not be easy to find.” (Page 6.)

“Surely what we should do to raise fertility is not to suppress birth control but to remove as many as possible of the obstacles which are impeding the free expression of the parental instinct. Conditions should be created in which people would want to have children and would have them deliberately and gladly. We should aim at minimizing, or if possible, removing the deterrents which now cause them, on balance, not to want children.” (Page 99.)

There is little prospect that the birth-rate can be increased unless the Government is prepared to tackle the problem very seriously and introduce radical measures to combat the danger. Unfortunately no political party looks very far ahead or has much vision. That is left to small minorities who are designated “alarmists”. Later, when the correctness of their views and warnings are confirmed, emergency measures are excitedly introduced and the minorities are then told that there must be no recriminations! I would warn the Government, as others have done, that the situation will become desperate in some years time—hurried legislation will then be too late!

Obstetricians can play a most important part in counteracting or lessening the grave national danger—they must do everything possible to save foetal life. The number of foetal deaths from prematurity, toxæmias, accidents and injuries in childbirth must (and can) be cut down; neonatal deaths must (and can) be cut down; the enormous wastage

of foetal life from abortion must (and can) be reduced. This can only be accomplished by means of a National Maternity Service in which obstetricians and the Public Health Service (with all its ancillary agencies) are in full and sympathetic co-partnership. The sands are running out—no longer may we procrastinate and set up “Committees of Enquiry”—impending disaster is on our doorstep!

THE FUTURE

It can hardly be expected that advances in obstetrics and gynæcology in the second half of the century will equal in impressiveness those of the last forty years. There is reason to hope, however, that some of the problems I have referred to will have been solved, or at least be nearer solution, when the century closes.

It is probable that the efforts of obstetricians and gynæcologists in the immediate future will be concerned very especially with organizing the maternity services of the country.

Suppose I were to put the question: What is your ideal of maternity service? Would you not answer: To ensure safe conduct during pregnancy and childbirth for the expectant mother and her child. Then suppose I asked: Who are the most experienced in obstetric practice to carry out this work? Would you not answer: Obstetric specialists and highly trained midwives. Then suppose I went further and asked the question: Where can expectant mothers be best treated during labour, and if the need arise, for any complication in pregnancy, would your answer not be: A well-appointed maternity institution.

I agree that at the moment it is impossible to provide institutional treatment for all; but it is feasible and is being approached in a number of cities at home and abroad. It might require something in the region of 25,000 to 30,000 beds for England and Wales—that is only a very approximate estimate. The figure can easily be worked out and the cost estimated. Certainly domiciliary midwifery gives fairly good results; but most doctors do not have their wives delivered in their own homes; and they also advise their patients to be confined in an institution. Why then should not the wife of the artisan living often in very unfavourable surroundings in an industrialized city, or the wife of the black-coated clerk living in a tiny bungalow, have equally good treatment by experts and in equally good surroundings.

It may be argued that this means taking away the obstetric practice from general practitioners. No one holds general, or as I prefer to call them, family practitioners in higher regard than I do. The speciality of general practice is of equal importance to that of any of the recognized specialities. The words of St. Paul “We are many members in one body, and all members have not the same office” apply very directly to our profession at the moment and for the future. For more than twenty years I have tried to persuade my colleagues in general practice that it is in the interests of pregnant and parturient women, and it is to their own advantage, to hand over obstetric practice to specialists. They usually have obstetric specialists to look after their wives—in one single year I attended the wives of 23 general practitioners. Let me quote from my Inaugural Lecture on February 10, 1927, when I was transferred from the Muirhead Chair to the Regius Chair of Midwifery in Glasgow University:

“The future of general medical practice lies more and more in the direction of *pure medicine*, and the general practitioner must be raised in pure medicine to a very high level. He, associated more intimately with the consulting physician, clinical pathologist, and officer of health, is the individual whose assistance will be of inestimable value in advancing medicine. For, as the late Sir James Mackenzie pointed out repeatedly, on the general practitioner must we depend for the early recognition of disease—the field of medicine which at the present moment offers the most promising harvest. Though this will ultimately be the vocation for the general practitioner, it does not mean that he will occupy a less important place in the community, nor that he will be less beloved than the old-time family doctor. The general practitioner, as I see him, will occupy a more important place, and incidentally he will have an infinitely more interesting professional life.” (*Brit. M. J.*, 1927 (i), 870).

Obstetricians are agreed that the ideal arrangement is that maternity hospitals should be units of the general hospital. There are now many examples of this arrangement

the long and arduous training this type of research entails—they will always remain amateurs. Possibly in the future—it has been suggested—there may be established in the country one or two large maternity units complete with clinical material (beds), and laboratories for every type of investigation. I wonder if it is feasible, or necessary, or advisable to attempt so ambitious a scheme!

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Obstetricians can play a most important part in counteracting or lessening the grave national danger—they must do everything possible to save foetal life. The number of foetal deaths from prematurity, toxæmias, accidents and injuries in childbirth must (and can) be cut down; neonatal deaths must (and can) be cut down; the enormous wastage

Now in conclusion permit me to digress for a moment or two from Maternity Hospitals and a "National Maternity Service" to the wider question of General Hospitals and a "National Medical Service". Here one of the outstanding questions is: Should Voluntary Hospitals be preserved as definite entities in the hospital services of the country; or should there be unification of voluntary and municipal hospitals and one type of hospital established?

I am well aware that there is a large and influential body of opinion in favour of preserving voluntary hospitals; but there is also a large body of opinion in favour of *unification of hospitals* at the earliest possible date—obviously impossible at the moment! We, for I include myself amongst the latter, consider that it is an anomaly to have two types of hospital doing the same work. We think it is inevitable that comparisons will be made regarding their respective services in general and in particular regions. We believe that the standard of efficiency in modern municipal hospitals is no whit below that in voluntary hospitals—take as an example the results in the maternity units of the L.C.C. hospitals. Speaking for myself I served all my medical life in voluntary hospitals, except for one year when I was one of the R.M.O.s in the large Fever Hospital (Belvedere) on the outskirts of Glasgow. The standard of medical work and nursing has always been of an extremely high order in this hospital, and it has had some very distinguished medical superintendents.

To witness the passing of Voluntary Hospitals will be particularly sad for us who have served or are serving in them. We can understand also the regrets of Chairmen, Governors, subscribers, and many others who have ungrudgingly given of their time and substance to maintain these wonderful institutions.

I am under no illusion that any words of mine, or of others of a like opinion, will have any influence in preventing an attempt being made to maintain voluntary hospitals as distinct units of a National Hospital Service. Already a *most* complicated machinery of Divisional and Regional Councils for furthering this object is being set up. Each will have its subcommittees, special committees, *ad hoc* committees and so on. It is, I admit, quite impossible to carry on any public service without committees and subcommittees; but let us try to cut down and simplify as far as possible this Committee work, which clogs the wheels of progress and wastes the time of men of ability and action.

I have permission from Sir Frederick Menzies, K.B.E., F.R.C.P., to quote from a letter received a few days ago.

"My dear Professor,

"In reply to yours of the 6th, I should unhesitatingly agree that the *ideal* arrangement is *one* Hospital Service for the whole country, and provision in such a service for all classes of the community; and payments for the same in accordance with 'capacity to pay.'

"But, and unfortunately it is a great big 'But'—the attainment of such an ideal is, at all events at the moment unattainable. All history goes to show that *progress in this* country is by a process of *evolution* and not revolution. *War conditions tend to expedite the process of evolution in some respects at all events.*

"It seems to me, therefore, that these two conditions: (a) the inevitable changes in the voluntary hospitals and (b) the changes for the better in municipal hospitals will combine ultimately in the merging of the two types of hospital service into one—that is to say—Your ideal will come about by a natural process."

One group of hospitals colloquially designated "Teaching Hospitals" should be given special consideration in any scheme for a National Hospital Service. Obviously that is too controversial a question to discuss in this Address. Conceivably they might be retained as definite units and subsidized by grants made by "The University Grants Committee".

While we are slowly progressing to a "National Medical Service" for insured persons with incomes up to £420 per annum (possibly up to £500 per annum in the near future), and their dependants, might it not be advisable that members of the medical profession give consideration not only to the "regional organization" but also to the "central body" at the Ministry of Health, which will direct the service?

I have quoted from St. Paul, now let me quote the words of St. Matthew: "Agree with thine adversary quickly while he is in the way with you." I do not mean to suggest

in England, Scotland and Northern Ireland—one of the latest to be erected is the Simpson Memorial Unit of the Royal Infirmary, Edinburgh.

The staffing of maternity hospitals or maternity units of general hospitals, and the antenatal clinics associated with them, and the antenatal clinics of local authorities by specialists who will be responsible for both antenatal and intranatal care is simple, for large cities. Very different is the position in respect to small towns and sparsely populated rural areas. For them (and it has been done already in certain areas) it is suggested that the local practitioners should select some of their number for maternity work. These family practitioners selected would be expected to undergo special post-graduate training and take a diploma in obstetrics.

Obviously the administrative bodies of the "region"—presumably the country will be divided into "regions"—will be the local authorities of the region with the Ministry of Health as the "Central Directing Body". Agreement in respect to the constitution and personnel of the "Regional Bodies" will probably be reached comparatively easily—indeed there has been a good deal of spade work done already by The Medical Planning Commission and other bodies. Time permits me to deal only with the constitution and personnel of the "Central Directing Body".

The arrangement which I personally advocate is an *ad hoc* body at the Ministry of Health and at the Department of Health for Scotland designated "The Central Obstetric Board of the Ministry of Health for England and Wales", and "The Central Obstetric Board of the Department of Health for Scotland". We should not be put off with an "Advisory Committee"—such committees have no authority and in my experience, their views are received with the greatest courtesy, then filed and forgotten in most instances. The "Central Obstetric Board" for England and Wales should be representative of all concerned—Ministry of Health, Regional and Local Authorities, Obstetricians (nominated by the Royal College of Obstetricians and Gynecologists), General Practitioners (nominated by the British Medical Association), Midwives (nominated by the College of Midwives), and Insurance Commissioners. It might be advisable to include representatives from other bodies. The Board for Scotland would be constituted on similar lines. Personally I favour the view that the Chairman should be the Permanent Secretary of the Ministry or the Chief Medical Officer of the Ministry of Health, and in the case of Scotland the Permanent Secretary or the Chief Medical Officer of the Department of Health. Some might prefer an independent Chairman; but there are obvious objections to that arrangement. My scheme for a National Maternity Service was published in the *Journal of the Royal Sanitary Institute*, 1931, 52, No. 4, 156.¹

The views expressed when I presented my scheme are in the main the same as I hold to-day. Every year that passes some come nearer fulfilment. Of one detail I have seen no promise of fulfilment so far, viz. the creation of a "Central Obstetric Board" at the Ministry of Health for England and Wales, and at the Department of Health for Scotland. *That I consider of vital importance* if we are to have a National Maternity Service with all the agencies concerned working together harmoniously.

This gives me the opportunity of referring to concessions which each and all must make to establish the co-operation so freely advocated by individuals from the highest to the humblest in the Public Health Service. I would point out that obstetric specialists, family practitioners, midwives, and voluntary hospitals would make considerable sacrifices should a National Maternity Service be established. They have such an abhorrence of bureaucratic control that one and all hesitate to give it their support. If, however, the Ministry of Health for England and Wales and the Department of Health for Scotland would indicate their approval and support of a Scheme, such as I have suggested, in which the "Directing Body" was representative of all the agencies working the service, objectors to a National Maternity Service might be persuaded to co-operate, and to do so with a certain degree of enthusiasm.

¹ Also as Chapter XVII (page 332) in "Maternal Mortality and Morbidity—A Study of Their Problems" (by J. M. Munro Kerr, E. & S. Livingstone, Edinburgh, 1933). Blair Bell presented a scheme as did also Ministry of Health Interim Report of Departmental Committee on "Maternal Mortality and Morbidity" (1930), and the British Medical Association in its Memorandum on a "National Maternity Service" for England and Wales, published in the *Brit. M. J.*, 1929 (i), Suppl. 258. A very exhaustive review of these four Schemes was published in the *Lancet*, 1931 (ii), 367.

Section of Dermatology

President—H. C. SEMON, M.D.

[January 15, 1942]

Erythroplasia.—CLARA M. WARREN, M.R.C.S., L.R.C.P.

Examples of extra-mammary Paget's disease are rare, and this one in a man of 66 shows many typical features. The initial lesion was said to be an infected fissure on the dorsum of the penis, first noticed about four years ago. There was difficulty in getting the area to heal, and it has relapsed at intervals, the erythema gradually spreading until half of the circumference of the organ is now involved. It is well margined; the centre shows some atrophy, and subcutaneous thickening is palpable, particularly at the periphery. The Wassermann reaction is negative. The diagnosis was confirmed by microscopical examination.

Discussion.—Dr. W. FREUDENTHAL: A biopsy shows a moderately irregular acanthosis with a few clumping cells of the Bowen type. The histological findings in cases of erythroplasia vary greatly.

The PRESIDENT: Has anyone seen a case in which the glans escaped? And what is the prognosis here?

Dr. JOHN FRANKLIN: For treatment the best chance of getting a good result short of surgery would be contact X-rays.

Dermatitis Atrophicans (Atypical).—J. E. M. WIGLEY, F.R.C.P., and W. FREUDENTHAL, M.D.

An apparently healthy man, aged 20, with no history of previous skin disease. The present lesion on the left thigh has been noticed about five years, whilst those on the right arm and lower legs have been present about four years. He does not know the duration of the small lesion on the right thigh. The lesions are symptomless, though he admits there may have been occasional very slight itching. He says he feels perfectly fit, and was only discharged from the Army this year as the skin lesions were diagnosed as lupus. He has never had X-ray treatment. W.R. is negative.

The two principal lesions are those on the inner side of the right upper arm and the outer side of the left thigh. Each is about the size of the palm of the hand and of irregular oval shape.

The lesion on the right upper arm is an ill-defined area, showing a wrinkling of the skin suggestive of superficial atrophy, some sparse scaling, telangiectasis and irregular pigmentation. There are a few red papules scattered over the areas and the whole

that The Ministry of Health is an adversary of the medical profession in the literal sense of the word; but I do state that it is an autocratic body—I wish it to be *more democratically constituted for dealing with matters that directly concern medical practitioners (family practitioners and specialists alike), who after all do the great bulk of medical work.* The “Central or Controlling Body”, I have suggested for a “National Maternity Service” might be created in triplicate, so dear to officialdom. Thus we might have a *Medical (including the Medical Specialities) Board* a *Surgical (including the Surgical Specialities) Board* and an *Obstetric Board*—each constituted on lines such as I have indicated for obstetrics—within the Ministry of Health under the Chairmanship of the Permanent Secretary and the Vice-Chairmanship of the Chief Medical Officer of the Ministry, and with one representative from each of the three Boards and from the body of general practitioners in the Ministry additional to the Chief Medical Officer of the Ministry of Health. There is an “Advisory Medical Committee” associated with the Ministry and there is the “Board of Control” loosely associated, but independent of the Ministry. Thus there exists already bodies containing the foundations of “a directing body” such as I have suggested.

But while I am convinced that such an arrangement is ideal for Obstetrics I am not prepared to stress it for Medicine and Surgery. I leave Physicians, General Practitioners and Surgeons to indicate their wishes.

It is of vital importance that the State has whole-hearted co-operation from the medical profession in its National Health Service.

Cases were reported and specimens shown at the meeting as follows:

Miss Gladys Hill: An Unusual Uterine Tumour.

Miss M. Moore White: Two Cases of Sarcoma of Uterus. Report of case of spontaneous delivery following course of radium therapy for recurrence of epithelioma of vulva when patient two months' pregnant.

Photographs and sections of epidermoid carcinoma of cervix showing extension to body of uterus. Slides of ovarian cyst containing granulosa-cell tissue in wall. Slides of solid ovarian tumour. ? Anaplastic carcinoma. ? Spindle-cell sarcoma.

Mr. J. V. O'Sullivan: Case of irreducible prolapse, complicated by stones in the bladder.

Biopsy (Two sections were shown under the microscope, prepared and examined by Dr. Freudenthal).—A nodule looking like lupus was removed from the right forearm, and one suggestive of lichen planus from the left forearm. Microscopically the former showed several sharply demarcated foci of tubercloid tissue with necrotic centres, and at the periphery were smaller foci of epithelioid cells. These were separated from the epidermis by a narrow strip of normal connective tissue. This histological picture is characteristic of lupus vulgaris and is never met with in lichen planus.

The other specimen (from left forearm) presented the typical histology of lichen planus without a trace of tubercloid tissue. It showed hyperkeratosis, granulosis, and acanthosis. A small-celled infiltration, sharply limited below, runs along the upper part of the cutis. There is also a group of colloid blocks of the same shape and arrangement as depicted in Kyrle's illustration of lichen planus ("*Histo-Biologie der Menschlichen Haut und Ihrer Erkrankungen*," Wien: Springer, 1, 186, fig. 108). Such colloid blocks are seldom seen in any other disease.

Comment.—At my first clinical examination I was doubtful whether the diagnosis was tuberculosis verrucosa and lupus vulgaris, or lichen planus et verrucosus. It is not easy clinically to pick out which lesion is which, but the histology leaves no doubt that both diseases are present. It is remarkable that they should both be confined to the same regions.

The case, as pointed out by Dr. Freudenthal, is of some significance in connexion with the nature of lichen nitidus. It has been suggested that this might be lichen planus which provokes a tuberculous type of tissue reaction when it occurs in a tuberculous subject. But here we have lichen planus maintaining its own peculiar histology in close juxtaposition with tuberculosis of the skin.

Discussion.—Dr. H. W. BARBER: Is the condition of the palms tuberculous, or is it lichen planus?

Dr. GOLDSMITH: I should expect it to be tuberculous. There is a history of the removal of a multiple ganglion. The condition of the overlying skin has developed from that operation wound for twenty years, quite gradually spreading—so gradually that the man took no notice of it.

Though no tubercle bacilli were found a skiagram showed tuberculous changes in the lungs.

Dr. GRAY: I think that it is extremely difficult to distinguish between the lesions on the right forearm and those on the left. I should have said that whatever was the histology, the lesions were the same on both. I suggest more pieces be examined. I think that where lesions are clinically the same but differ histologically one should not be satisfied with one histological examination.

Cellular Nævus Resembling Lupus Vulgaris.—GEOFFREY DUCKWORTH, M.R.C.P.

This case is interesting because it has been mistaken for and treated as lupus vulgaris for many years. The patient is a healthy girl aged 19. At 8 years one or two papules appeared, followed by others. The present lesion gradually developed on the chin-strap area of the right side of the neck, extending from the angle of the jaw to the mid-line, in front, where it stops abruptly. It is an oval patch, made up of discrete reddish-brown papules and nodules, that show a brownish-yellow staining on glass pressure. Wrinkling of the epidermis is present and slight atrophy is to be seen, but nowhere is this localized—it is spread diffusely over the patch. Presumably this is due to the treatment which has consisted of a few applications of chemical caustics and many of ultra-violet light, given locally. Histology: Sections show a cellular nævus, pigmented in places.

Discussion.—Dr. J. E. M. WIGLEY: Whether the condition is lupus or a nævus could not excision with subsequent grafting be undertaken? Then one could obtain many sections.

The PRESIDENT: I remember a recurrence in the scar after excision for which the patient was X-rayed several times. He then came under my care and I sent him to Professor Rollier who failed, however, to do any good whatever with solar therapy, and explained that the X-ray treatment had spoiled his chance of getting a good result. I have always been averse to excision of lupus vulgaris on the face.

appearance is strongly reminiscent of a chronic X-ray dermatitis, being also suggestive of *poikiloderma atrophicans vasculare* (Jacobi).

The lesion on the outer side of the left thigh is more sharply defined and the atrophy is more marked. There is considerable loss of substance, producing a saucer-like depression, about 4 by 3 in. in area and up to $\frac{1}{3}$ in. deep, and the larger vessels are easily seen through the wrinkled skin. The pigmentation is well marked and there are one or two scabbed lesions which look like the result of trauma (e.g. scratching).

The lesion on the right thigh is an ill-defined slightly atrophic area, in which a definite "gap" can be felt in the substance of the skin on palpation. The other lesions are similar in appearance (though smaller) and give the impression of being intermediate between those already described. The total number of lesions is 5.

Biopsy A. (from the lesion on the right thigh): The special features are regressive connective tissue changes in parts of the deep cutis and in the strands between the fat lobules. The normal, thick collagen bundles are mostly replaced by very fine connective tissue fibrils which form a close, irregular network. Most of the fibrils stain distinctly red with van Gieson, others only very faintly. The elastic fibres in these parts are greatly diminished. Regressive changes are also seen in adjacent areas of the subcutis where the meshes of the fat tissue are torn and stain faintly.

Throughout the cutis there is a considerable, mainly perivascular round-cell infiltrate. The walls of some of the vessels are greatly thickened. In the areas where regressive changes are seen, however, the infiltrate is almost entirely absent.

Biopsy B.: A small, flat, red, shiny papule, 1 in. from the saucer-like depression on the left thigh. There is a fair amount of round-cell infiltration, diffusely arranged in the papillary body and perivascularly in the middle and deeper cutis. Within the infiltrate the elastic fibres are diminished. No regressive changes in the connective tissue.

Comment.—The case is one of primary or essential atrophy of the skin. Although it does not appear to be identical with those already described either as *poikiloderma atrophicans vasculare* or *acrodermatitis atrophicans* it has some clinical characteristics of each of these. It is therefore recorded as *dermatitis atrophicans* (atypical) as we think it is better than to add to the already numerous names of the subdivisions of primary atrophy.

Dr. ST. R. BRÜNAUER: This case shows clinical signs of different types of skin atrophy. On the right upper arm there is an area of scaly, cigarette-paper-like atrophy, the surroundings of which show subacute redness. Changes of such a kind mostly occur in cases of diffuse atrophy of the skin. On the outer part of the left thigh there is a larger area of deeper atrophy with the inner parts slightly depressed. Here and there near the atrophic areas small, flat papules of bright red colour are to be seen as they were described by Holoussi Behdjat of Ankara as primary lesions of *poikiloderma vasculare atrophicans*. Telangiectasis and pigmentation which are signs of *poikiloderma atrophicans vasculare* as well, are to be found in nearly every one of the atrophic areas.

It might well be that this remarkable case is a transitional kind of atrophy to be placed between the diffuse idiopathic atrophies and the *poikiloderma atrophicans vasculare*.

Tuberculosis Verrucosa Cutis and Lichen Planus.—W. N. GOLDSMITH, M.D.

L. P. W., man aged 51. Chauffeur.

Present condition (25.11.41).—The right palm is contracted and covered by a hard warty plaque with mauve border. On both forearms are small irregular purple scaly infiltrated patches. Some look like lupus vulgaris, and others having a whitish flat surface, rather more like lichen planus. Those tested with a matchstick did not give the usual sign of lupus vulgaris.

History.—The eruption on his forearms is of two and a half years' duration. Twenty years ago he had a compound ganglion (probably tuberculous) removed from his right palm. The present appearance of the skin of the palm developed gradually. His general health is good and there is no tuberculosis in the family.

Investigations.—W.R. negative. Skiagram of the lungs shows extensive active tuberculous changes.

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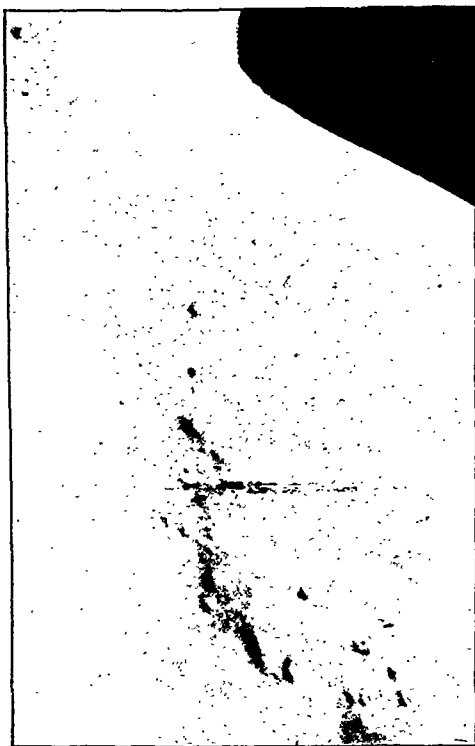
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Investigations.—W.R. negative. Skiagram of the lungs shows extensive active tuberculous changes.

Internal examination revealed an adenoma of the right lobe of the thyroid gland, with deviation and compression of the trachea from which the patient at present suffers no discomfort. Mr. K. Eden suggested the removal of the tumour.



Numerous dome-shaped papules on the back. A few, in the right lower corner, show some dimpling. (For another photograph of this case showing annular lesions, see report of this meeting in *Brit. Journ. Dermat.*)

Comment.—Three groups of cases of myxœdema might be distinguished: (1) The classical generalized myxœdema of cretins (Gull and Ord). (2) The pretibial myxœdema, especially in Graves' disease. Cases (after thyroidectomy) were shown at previous meetings (Dowling, G. B., *Proc. Roy. Soc. Med.*, 1934, 27, 1361; Bamber, G. W., *ibid.*, 1937, 31, 350). (3) The "atypical" myxœdema of Jadassohn-Doessekker (Doessekker, W. *Arch. f. Dermat. u. Syph.*, 1916, 123, 76), with a great variety of skin manifestations: circumscribed or, like our case, disseminated or eruptive, tuberos, plaque-like, lichenoid, papular, fibromatoid, &c. With increasing knowledge we should soon be able to classify these "atypical" cases. A connexion with thyroid dysfunction is usually less obvious in this group than in the two others and is sometimes only shown by the involution of the skin lesions after administration of thyroid or, as in Jadassohn-Doessekker's case, after implantation of thyroid tissue.

Discussion.—Dr. ST. R. BRÜNAUER: This case is almost unique. Similar cases in the literature do not show such numerous lesions all over the skin, nor the presence of papulo-lenticular, dimpled or umbilicated, and annular lesions in the same case.

Mr. KENNETH EDEN: This patient had a small nodular goitre, but no evidence of thyrotoxicosis or myxœdema, the basal metabolic rate being normal. I have never seen a case of this type before.

Dr. H. W. BARBER: Dr. Whitfield was in favour of excising patches of lupus vulgaris when practicable, but emphasized the necessity of removing the underlying subcutaneous tissue. I believe that Mr. Arthur Edmunds obtained excellent results in several cases referred to him by Dr. Whitfield.

Dr. A. M. H. GRAY: That is true where there are patches of lupus in such positions that free excision is possible. In such cases, after taking away the fat underneath, a recurrence is rare. The story is very different in the case of lupus on the face.

? Epidermoid Tumour of Nævoid Origin.—GEOFFREY DUCKWORTH, M.R.C.P.

Male, aged 63, A.R.P. worker. Has complained of an intensely itchy patch over his right shoulder-blade for two years. Clinically, the lesion is benign; pathologically, from the limited evidence available, possibly malignant.

On examination.—There is a circular patch, about an inch in diameter, of lichenification, situated over the upper part of the right interscapular region. There is a marked infiltration of the skin. It is circumscribed, and freely movable over deeper parts.

The patient does not remember having had a mole or other blemish on the affected part of the skin. The W.R. is negative.

From examination of the small piece of tissue removed for biopsy a positive diagnosis seems scarcely possible. The section shows, in the corium, well-defined epithelial cell masses, made up of prickle cells externally and horny pearls centrally. No basement membrane is present. There is a varying degree of cellular infiltration in the corium between the clusters of epithelial cells. The epidermis is acanthotic, shows a well-marked granular layer, and is moderately hyperkeratotic, especially at the mouths of the pilosebaceous follicles.

The possibilities seem to be: A prickle-celled epithelioma, perhaps taking origin from a small sebaceous cyst, or wall of a hair follicle; or an epidermoid tumour of nævoid origin.

POSTSCRIPT.—Microscopical examination of the nodule after its excision showed epidermal changes consistent with the clinical findings of lichenification. In the dermis there were many small and a few larger cysts with epidermoid walls, resembling sebaceous cysts of nævoid origin. Malignant proliferation appeared probable in one part. There was some perivascular cellular infiltration.

Myxœdema Papulosum et Annulare.—W. FREUDENTHAL, M.D., and ST. R. BRAÜNAUER, M.D.

E. A. R., clerk, aged 39. Has always been in good health, except for a "fit of giddiness" in March 1933 when a high blood-pressure was found. Later on he had two or three similar but slighter attacks. In July 1934 lesions appeared on the backs of the hands and wrists. In the following years they spread very slowly to the arms, trunk and legs. In recent months a great number of fresh lesions have appeared as we have seen in the last ten weeks while he has been under our observation. These lesions are symptomless except for slight itching and reddening on exposure to sunlight.

Present state: Hundreds of lesions are present on all parts of the body except the face, palms and soles. They are particularly numerous on the back and arms. The initial lesion is a small dome-shaped papule which slowly increases to 6-8 mm. in diameter. Papules larger than this may become dimpled in the centre and, with increasing size (12-15 mm.) form small rings with apparently normal centres. In all stages of development—papule, dimpled papule and ring—the lesions are very soft; their colour is that of the surrounding skin or slightly paler, looking somewhat opaque. In a few places, e.g. the wrists, some lesions have regressed and left ring-like vestiges and a very superficial scarring.

A biopsy of a papule showed a large amount of mucin especially in the uppermost part which was covered by an epidermis with rete pegs flattened by pressure. Mucin was also present below the papule, the amount gradually decreasing towards the middle and still more, towards the deep part of the cutis. A biopsy of an annular lesion showed a fair amount of mucin in the area of the ring or a little below, in the central part there was only a little mucin left, but there was some round-cell infiltration.

Tuberculosis Nodularis Cutis with Glandular Involvement.—H. C. SEMON, M.D.

The patient has complained of cough, palpitation, and feeling tired for a number of years; and Dr. Lisle Punch reported "arteriosclerosis with myocardial degeneration and cardiac failure". The Wassermann reaction was negative, and an X-ray of the chest revealed cardiac enlargement and pulmonary congestion and hilar shadows "reported as being glands". Several soft elastic glands were palpated at that time in the neck and in the right axilla, but the spleen and liver were not palpable and a blood-count was normal—Hb. 98%, W.B.C. 7,000. In November 1941 there was "great enlargement of all accessible lymphatic glands, liver slightly palpable, spleen not palpable, enlarged heart, congested pulmonary bases with signs suggestive of obstruction by hilum glands, also purple subcutaneous nodules on both arms and on the trunk. These nodules are apparently increasing in number, and they are said to vary in size and to become less noticeable on holiday. Some of them show reduced sensitivity to pin-prick and cotton-wool. They have been observed by the patient for the last eighteen months".

No history of tuberculosis.

The patient feels languid, cannot concentrate, sleeps badly, coughs a lot. Sputum scanty, never hæmoptysis. He is gaining weight. Marked breathlessness on exertion.

All accessible glands enlarged, but not tender or painful. The upper extremities symmetrically involved on extensor aspects. The back shows similar though less marked lesions of one type only: nodules of a bluish purple colour of various sizes from a pea to a large plum. They all involve both the epidermis and the hypoderm, from which they appear to take their origin. Their distribution on the extensor surfaces strongly suggests a lymphatic spread. On the back they are situated mainly on the flanks in the lines of cleavage. There is no tendency to ulceration, while on the upper arms they are undergoing a definite shrinkage and tendency to involution.

Histology of skin lesion displays a tubercular structure with sparse giant cells and a rather degenerative tendency of both nuclei and cells. A second slide reveals acid-fast granules and bacilli in moderate number.

Blood examination (Dr. K. M. MacLaren): R.B.C. 4,600,000 per c.mm.; Hb. 94%; C.I. 1.0; W.B.C. 7,000; polys. 42%, 2,940 per c.mm.; lymphos. 41%, 2,870 per c.mm.; monos. 8%, 560 per c.mm.; eosinos. 9%, 630 per c.mm.; basos. nil.

The case had been diagnosed as leprosy, but with the exception of a single cruise as far as Italy the patient has never been out of the country. My own feeling was rather against that diagnosis. On biopsy of one of the nodules on the forearm we were surprised to find acid-fast granules and also quite a number of acid-fast bacilli. The generalized adenitis which this patient has would, I think, be rather against a diagnosis of leprosy and more in favour of a tubercular manifestation. One member suggested sarcoid, another Hodgkin's disease with skin manifestations; both these diagnoses were rather upset by the finding of the acid-fast bacilli. Dr. Gray has suggested that we remove a gland and inject into a guinea-pig to settle the diagnosis as between leprosy and tubercle.

Discussion.—Dr. W. FREUDENTHAL: A skin biopsy shows a large amount of sarcoid and tuberculoid tissue with a very small number of acid-free bacilli. There are also acid-fast granules present. These alone, however, should always be interpreted with caution in skin sections, as normally sweat glands contain acid-fast granules. This fact was not sufficiently known, so these granules had been mistaken for the granular form of tubercle or leprosy bacilli.

Dr. A. M. H. GRAY: This case appears to be one of a very definite group of cases which apparently are tuberculous. It is very similar to two others I have seen. At first glance this case looked like a sarcoid, but the glands are enormous for sarcoid, and the histology and the finding of bacilli seems to be against it. There are two things to be noted: the very large glands and the fact that they were present for a long time before skin lesions appeared. That suggests a Hodgkin's disease on the history. A woman came to me at University College Hospital with a huge mass on one side of her neck; she had had it for eleven years. She then developed a patch of lupus on the right side of her face of very much the same type as in this case, and this has gone on spreading in spite of treatment over a considerable number of years—eight or nine. We clear up one patch by one method or another, and then fresh nodules of the same type come up at the periphery. She has lost practically the whole of her ear on the right side, as well as considerable facial tissue. The second case was seen at

Dr. Trotter and I have, however, been interested for some time in localized myxœdema associated with thyrotoxicosis. At a thyroid clinic during the last three years, we have observed three cases of circumscribed pretibial myxœdema with toxic diffuse goitre in a series of 134 cases of toxic diffuse goitre and 254 cases of goitre, so that the incidence of this skin disease is not apparently insignificant in toxic diffuse goitre.

The first of these cases was the most obvious, developed before the thyrotoxicosis was treated, and persisted after treatment. The second case was less obvious, and the skin plaques first appeared eight months after thyroidectomy when the disease was quiescent. The third case had only a slight degree of the condition and was noticed before operation, only because we were looking for it. In the last two cases the plaques have tended to disappear spontaneously over a period of many months.

The association of this pretibial type of localized myxœdema with thyrotoxicosis is a feature of all the recorded cases, and we have been chiefly interested in this apparently paradoxical association. Unfortunately so far we have not found the explanation, though we have assured ourselves that the skin condition does not respond to treatment with thyroid extract, and apparently pursues an independent course. In addition, biopsies of the pretibial skin in other cases of Graves' disease have shown no excess of mucin.

Dr. Freudenthal's case is different from the pretibial form, but it is interesting to find that although thyrotoxicosis is absent there is some evidence of thyroid disease. I think the adenoma should be removed in view of the tracheal obstruction and this will provide histological evidence to complete the case.

Dr. GORDON: Is the distribution of mucin in this patient the same as in the pretibial myxœdema of the skin?

Dr. W. FREUDENTHAL: In pretibial myxœdema mucin is found mostly in the middle and deeper parts of the cutis and subcutis, although sometimes, e.g. in warty lesions, it may also be seen in the upper part of the cutis.

Mr. KENNETH EDEN: In the article on this subject (Pillsbury, D. M., and Stokes, J. K., *Arch. Dermat. & Syph.*, 1931, 24, 255), two forms of localized myxœdema are distinguished. One is the pretibial plaque-like form, and is always associated with thyrotoxicosis; the second is nodular, and is unassociated with obvious thyroid disease. It is possible that Dr. Freudenthal's case might fall into the second group.

Lichen Simplex Controlled by Stilbœstrol.—H. C. SEMON, M.D.

A woman, aged 47, has had lichenification of the right thenar eminence with pronounced itching on and off for twenty years. There are no lesions elsewhere. After hysterectomy three years ago there were menopausal flushings until recently. She was first seen by me on October 27, 1941, and was treated with 1 mg. tablet of stilbœstrol daily for ten consecutive days in each month. The cutaneous symptoms (and the flushing) appear to be controlled without local applications of any kind.

Scleroderma Circumscriptum.—H. C. SEMON, M.D.

This patient gave a history of two years' pruritus vulvæ, with a "feeling of dryness". She also complained of frequency of micturition, without stress. Menopause nine years previously, at the age of 39. She had previously been treated for an ulcer at the junction of middle and lower third of front of left leg, which had been labelled provisionally "? tuberculous". The Wassermann reaction was negative. Cervix and uterus: nothing abnormal. There were redness and induration around the vulva and on adjoining skin of both thighs, with atrophy of the labia and contraction of vaginal inlet. This is tight posteriorly and cracks on examination.

The most active and troublesome of the lesions in this case are on both sides of the vulva, which is itself involved in the cicatrizing process. The patches here are circumscribed and show marked atrophy. There is a rough symmetry. Additionally there is a small brooch-like lilac ring type of lesion on the front of the right side of the chest, while the skin above and behind the right shoulder displays the type known as "white spot" disease. The white patch on the front of the left leg has undergone ulceration, as not infrequently happens over bony prominences as a result of trauma. Some relief of the pruritus appears to have followed the administration of stilbœstrol *per os*.



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Great Ormond Street. A child came up with huge glands all over the body. A diagnosis of lymphadenoma had been made. It proved to be lupus of the hypertrophic type, associated with the pre-existing general adenitis. The case now shown is, I think, of exactly the same type—apparently a case, not of Hodgkin's disease but of true tuberculosis.

Bullous Livedo from Heat.—E. W. PROSSER THOMAS, M.D.

Mrs. E. H., aged 42, complaining of blisters on legs on and off for five years. Eruption comes in attacks lasting about six weeks. Blisters leave behind pigmentation. Present attack has lasted four weeks. She had three attacks in past year. Has had similar blisters on the back of the right lower forearm, otherwise no other part of the body has been affected and there has been no previous skin trouble. She ascribed the original outbreak to her occupation, raking out fires, the areas involved being those most exposed to heat.

On examination.—Numerous thin blisters containing clear serum are present over the inner aspect of the left leg and the outer aspect of the right leg. A few have erythematous bases but the majority seem to arise in and to follow the outlines of a livedo reticularis-like background. There is also a good deal of light brown pigmentation in the affected areas apparently from healed blisters. There is a similar faint reticular pigmentation over the lower extensor surface of the right forearm. In view of the distribution of the lesions over the right sides of the legs and the back of the right forearm, her explanation that heat is responsible seems reasonable. Adamson (*Brit. J. Dermat.*, 1916, 28, 281) in a paper on livedo reticularis mentions a chronic vesicular form due to heat and shows an illustration of a large blister of the leg on a livedo background.

In the present case, however, some of the attacks do not seem to have been associated with fire-raking and have occurred in the summer, which is difficult to explain.

Section for the Study of Disease in Children

President—A. G. MATTLAND-JONES, O.B.E., M.D.

[January 23, 1942]

DISCUSSION ON INTRACRANIAL INJURIES IN CHILDHOOD

Mr. D. W. C. Northfield: My experience of cases of head injury in children, has for the most part been acquired since the outbreak of war. Although the air-raid casualties are included, the majority of the injuries have resulted from ordinary accidents and have an immediate application to everyday practice.

The series includes 43 cases, the ages ranging from 3 months to 14 years. In 35, the injury was closed, and of these 11 were air-raid casualties. 9 of these closed injuries were cases of scalp wound—without a break in the underlying bone. The remaining 8 cases had sustained a compound fracture of the vault—that is to say the damage to the brain was of an open variety, with a real risk of intracranial infection. This is the main reason for separating head injuries into “closed” and “open” groups. Cases of scalp wound could be classified as “open”, but I regard them as virtually closed—though needing proper surgical attention. Of these 8 cases of compound fracture, 6 were air-raid casualties, a higher proportion than in the case of closed injuries—which is only to be expected.

Analysis of the 35 closed injuries.—There were nine scalp wounds. In 10 cases there was a large hæmatoma of the scalp, a frequent condition in children. This presents as an extensive fluid swelling under the scalp, usually tender to the touch; within a day or so its edges become indurated so as to simulate, in textbook fashion, a depressed fracture. This difficulty can often only be settled unequivocally by an X-ray. The condition in my opinion is rarely found in adults, the reason being the greater fragility of the child's skull. A fracture of the vault of the skull was revealed by X-rays in 15 cases, nearly one-half of all the cases of closed injury, and a much higher proportion than in corresponding adult cases. Of the 10 cases of cephalhæmatoma, a fissured fracture (not depressed) of the bone in the region of the effusion of blood was proven in 8. It was possible to demonstrate radiologically a fracture of the base of the skull in only 1 case, in which there was also clinical proof—a discharge of blood and of cerebrospinal fluid from the ear.

There was an acute brain injury in 26 cases; the criteria employed in making such a diagnosis fall into three groups—unconsciousness, abnormal neurological signs, and symptoms of disturbance of cerebral function. Although these groups will first be considered separately, a given case might have satisfied any or all of the criteria.

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watched for five years will be referred to when epilepsy is considered. Of 15 cases of closed injury, 4 still have occasional slight headache at diminishing intervals of weeks; the others are all quite normal except that in 3, the mother complained spontaneously that the child was very restless at night. 1 of 3 cases of open injury has occasional headache.

Epilepsy has occurred in 3 cases, in 2 of which it was focal and motor in character, and developed soon after the injury. One, a baby of 9 months, tumbled from the back seat on to the floor of a car when the brakes were applied suddenly. There was no loss of consciousness or any other apparent disturbance, but a few hours later the left arm was noticed to be hanging limp; about an hour afterwards clonic spasms of the left arm and leg developed and the baby became very drowsy. The arm and leg appeared paralysed. The epilepsy continued on and off for some twenty-four hours, thereafter it ceased, and the arm and leg gradually recovered strength within the next few days. The head appeared normal; a lumbar puncture was attempted unsuccessfully several times. This child (referred to earlier) has been watched for five years and is physically normal, except for occasional attacks of twitching of the left leg. She has taken phenobarbitone throughout this period.

The second case, a boy of 11 years, after six hours' unconsciousness as a result of a bicycle accident, progressed satisfactorily, apart from headaches, for six days. He then developed epileptiform attacks confined to the left side of the body associated with adverse turning of the head to the left. He became very drowsy with virtually complete paralysis of the left side of the body; sensory tests were not possible. The brain was explored through burr holes, on the supposition that there was a subdural hematoma. No intracranial hemorrhage was found but the pia-arachnoid seemed unduly tough and edematous. Unfortunately I have been unable to keep in touch with this patient.

Both these cases illustrate a well-recognized group, in which epilepsy develops very early after a brain injury, apparently related to an acute disturbance of neural function. Clinical experience supports the belief that this type of traumatic epilepsy may not necessarily persist, and has a better prognosis than the variety which comes on later, with its incidence roughly highest in the second year after the accident, a view favoured by the work of Dennis Williams upon electro-encephalography.

The third case of epilepsy illustrates a different aspect of head injury. A child of 2, injured in an air raid and with a scalp wound, was taken to a hospital. Next morning the child was sent out, the wound having received no surgical attention. The following day, on the advice of the private doctor, the mother took the baby to another hospital where it was admitted, as they discovered and confirmed by X-ray a fissured fracture of the right parietal region beneath the linear wound some 3 in. long. The child when transferred to my Unit four days after the injury, showed no evidence of any brain injury. The scalp wound was exuding pus, due to *Staphylococcus aureus*, and in its depths could be seen the fissure of the fracture. The next day there was a convulsion, not repeated, involving the left arm and face. The wound gradually healed and the child made a complete recovery. Here the epilepsy was due to cortical irritation from a localized patch of pachymeningitis. I quote this case in order to emphasize again that scalp wounds must be regarded as important. A fracture must always be suspected, and surgical attention must always be paid to it. The child with a compound fracture of the occiput, who died of streptococcal septicaemia, received no surgical attention to the wound within four days prior to transfer to my care. The reason offered was that the child was deemed too ill. That was an error of judgment, but too often scalp wounds go septic, and underlying fractures escape detection owing to negligence.

This review points to some conclusions peculiar to head injury in children. The incidence of fissure fractures of the vault in closed injuries is much higher than in adults; and often there is no loss of consciousness, probably owing to the greater fragility of the child's skull, which although probably more elastic, is yet much thinner than the adult's. With the frequency of fracture may be coupled the frequency of cephalhematoma simulating depressed fracture. On the other hand, the child tolerates a cerebral injury much better than an adult, and recovers from it much more rapidly and completely. The popular lay notion that "concussion" is a grave condition in a child is not borne

In 14 cases there was definite proof of loss of consciousness, about which certainty is even more difficult in children than in adults. The child may be playing alone, or with friends too young to give correct information; where the patient is transferred from another hospital, the information is lacking, usually due to carelessness in recording what may be the most obvious physical sign, or in obtaining the history. Of transient unconsciousness it may be impossible to obtain the desired evidence in the rush of rescuing and evacuating air-raid casualties. Failing a reliable witness of loss of consciousness, traumatic amnesia of the incident has been accepted as equivalent evidence, and probably more than 14 of the patients lost consciousness. 7 of these cases sustained a fracture of the skull. A point to be referred to later is that among the 15 cases of fractured vault, there were 10 in which consciousness was apparently not lost. Loss of consciousness varied in degree and duration; in some cases it was only for a minute or so, but in the most severe the patient was unresponsive for hours, delirious and uncooperative for several days.

Abnormal neurological signs were found in 9 patients. These signs always disappeared, and usually rapidly.

The commonest feature of acute brain injury was the group of symptoms which in the adult are associated with a major cerebral contusion, namely *headaches, irritability and resentfulness, drowsiness and vomiting*. These occurred in 16 patients, for periods varying up to a maximum of fifteen days.

When one or more of these clinical criteria of acute brain injury was present in the same case, the common combinations were unconsciousness and symptoms, 6 cases; signs and symptoms, 6 cases; and symptoms only, 6 cases. In 3 cases unconsciousness was followed by neither signs nor symptoms, and in 2 by signs and no subsequent symptoms. There were 3 cases in which the complete triad of unconsciousness, signs and symptoms was present, and 2 of these were very ill children. There was one other severe cerebral injury making only three in all for the total 35 cases of closed injury, a very small proportion when compared with adult cases of injury.

The immediate prognosis was excellent. None died, and all recovered speedily. Some stayed in hospital for only one to two weeks, the most severely injured stayed for three months, but that was due more to the difficulty of finding him appropriate accommodation than to unusually slow recovery, which was interrupted by an acute appendicitis.

Analysis of eight open injuries.—There have been 8 cases of compound fracture of the skull, and as already stated, the scalp wounds with no underlying fracture are included in the series of closed injury, as is a case of fractured skull-base although technically compound. Air-raid incidents have been responsible for the injury in 6 cases. The fracture has occurred over any region of the vault and due to a blunt object or to a missile, the dura mater being penetrated in 4 cases, and there has been a varying degree of damage to the underlying brain. In 3 of the cases there was loss of consciousness, and in 3 there were signs of brain damage. In only 2 were there the symptoms of a major cerebral contusion. These cases, though too few to analyse, are in keeping with one's experiences in all head injuries, namely, that in a case of open injury there is commonly no disturbance of the nature of a general cerebral injury, and that if there is loss of consciousness or other evidence of a general disturbance of cerebral functions then the injury is indeed a very grave one, out of all proportion to the degree of damage as judged by the external wound. Abnormal neurological signs are more likely to be found, and are more likely to be permanent, because of the focal nature of the damage to brain beneath the fracture. 2 of these cases of open injury died; 1 from intracranial hæmorrhage in spite of operation—a metal fragment passing through both frontal lobes. The other child died from streptococcal septicæmia, after a very long illness; the organism gained entrance through a compound depressed fracture in the occipital region.

SEQUELÆ.—The follow-up of these cases of children has not been so thorough as in the case of adults, possibly owing to problems of evacuation, and to death of parents. 18 cases have reported for examination for periods up to a year after the injury, and 1 case

a rise of intracranial pressure, sudden anaemia from collapse of cerebral vessels and loss of function of nerve cells, *unconsciousness*, of short duration. The recent important work of Denny-Brown and Ritchie Russell (1941) on experimental cerebral concussion in animals strongly supports the theory of concussion as the direct effect of physical stress on neurones; they define it as a traumatic paralysis of neural function in the absence of lesions. In the common type of injury with violent movement of the head, *acceleration concussion* is dependent on the changes from primary inertia of the brain to secondary momentum and finally the sudden arrest of momentum of the brain as the skull strikes a hard surface. Acceleration concussion was not found experimentally to be associated with any significant change in the intracranial pressure. In *compression concussion* when the head is fixed and force brought against it locally by striking, concussion was more difficult to produce. *Contusion* these authors outline as the result of physical stress upon the supporting vascular structures with varying degrees of hæmorrhagic effect.

As consciousness is regained in concussion immediate symptoms acceptable as after-effects are a general increased excitability of tendon-jerks, nausea or vomiting, giddiness and headache. Denny-Brown and Ritchie Russell consider that a prolonged state of stupor followed by intellectual impairment can occur as the result of the same mechanism which causes the momentary unconsciousness of concussion and without visible lesions in the brain.

In O'Connell's (1941) 250 head injuries, mostly war casualties (age-groups not given, but mainly adults), of 102 of the *Closed* type, 102 had concussion. In the *Closed* group it was the concussion which brought them to the neurological unit. In Northfields' 34 closed cases in children, 13 had had concussion. This disparity is due to the manner of selection in the first group and the fewer cases of concussion in children compared to adults owing to the greater elasticity of the skull, though other damage such as fissure fracture may be present.

Vascular and other lesions.—When unconsciousness is prolonged or a lucid interval is followed by unconsciousness or irritation or stupor, the possible causes, difficult to differentiate, include subarachnoid hæmorrhage, contusion (cerebral grazing), cerebral œdema, subdural and extradural hæmorrhages and sepsis. Each has distinguishing features, but in practice the picture is often blurred.

(1) *Subarachnoid hæmorrhage.*—Concussion is not always present, and the onset of headache, drowsiness, stiffness of the neck, pyrexia, unconsciousness may not appear until after an interval of half an hour or so. Slight paralysis is common on the same side of the body as that of the head struck, as the vessels are usually damaged by *contre-coup*. The cerebrospinal fluid is bright red with blood.

(2) *Contusion.*—The lesion is a localized meningo-cortical hæmorrhage either direct or by *contre-coup*. Its presence is suggested by a rapid onset of local paralysis, seldom a complete hemiplegia, provided the injury is not to a silent area of the brain. Jacksonian fits, indicative of the site of the lesion, are likely to develop early in the first one or two days. Only in the more severe grades of contusion is some blood or yellow-staining to be expected in the spinal fluid.

A boy of 1½ years fell from a table striking his head, was not unconscious but apathetic, did not talk and vomited twice. On examination the following morning slight weakness of the right side was found with hypotonicity of the limbs and right extensor plantar response. The cerebrospinal fluid was normal. He made a good recovery.

(3) *Extradural hæmorrhage* by itself would be expected to produce a fairly slow onset of hemiplegia over several days and Jacksonian epilepsy of later onset would favour the diagnosis. In uncomplicated cases the fluid is normal, or yellowish and with a raised protein content.

(4) *Subdural hæmatoma* is usually an encapsulated collection of blood of very slow accumulation. An unlikely result of head injury in children, it occurs at the extremes of life in babies and old people following apparently trivial head injuries.

out by experience. It is interesting to speculate on the relationship between these findings and the experimental work of Denny-Brown on acceleration-concussion (Denny-Brown, D., and Russell, W. R. (1941), *Brain*, 64, 93). Both the elasticity of the child's skull and its fragility will tend to block or delay the transmission of the acceleration imparted by the impact, through the skull, to the brain.

There was evidence of mental or psychological disturbance resulting from head injury in only 2 of the cases. One was the case of severe closed injury, in which acute appendicitis occurred. For about three weeks, this boy was petulant, irritable, self-centred and thoroughly difficult; thereafter he rapidly improved and now over a year later he is a normal happy schoolboy. The other patient lost both parents in an air raid, and for a week or so was very quiet and aloof, but gradually thawed and I think his troubles were not directly related to his head injury. The restless nights mentioned by three mothers may be some reflection of a mental stress, not otherwise evident, a small point worth consideration in similar cases.

Dr. W. G. Wyllie: For the most part similar problems present themselves in head injuries in children as in adults. Yet, there are some differences between the young and the mature in their reactions to head injury both in the immediate and in the after-effects. These dissimilarities, and the features which commonly give rise to difficulties in the diagnosis of the different forms of lesion are best illustrated by actual case-histories.

From whatever cause, head injuries at all ages are to be divided into *Open* and *Closed* types. The *Open* injuries include anything from a minor scalp wound to a compound or a depressed fracture or to cerebral penetration. The grosser forms of *Open* injury, requiring immediate surgical aid, commonly present, from the outset, signs of focal cerebral damage, provided the injury is not confined to a silent part of the brain.

My remarks are chiefly concerned with cases of the *Closed* type and cases of the *Open* type with minor scalp wounds, with skulls intact radiographically or showing fissure fracture, and with intracranial complications of onset either immediate or delayed for several days. In this group the commonest mode of damage is by violent movement of the head against a solid substance rather than by the sudden impact of a missile against the stationary head. This occurs in civilian practice when babies and toddlers fall out of cots, prams, off beds or chairs, and when older children, chiefly boys lorry-hiking, are knocked down in the street. In this commoner type of head injury these points stand out: the high proportion of cases without scalp wounds, the frequent occurrence of fissure fracture, and the high incidence of concussion.

Fissure fracture.—The first peculiarity of head-injuries in children is the relative ease with which fissuring can occur, frequently without intracranial complications. The cause is a massive diffuse violence as when the head comes forcibly in contact with the ground. When the momentary deformity, chiefly of the vault, reaches the limits of elasticity of the skull, bursting or fissuring occurs. Children's skulls, more elastic owing to their sutural flexibility, might be expected to need more deformity to produce a fracture, with consequently a greater likelihood of cerebral damage. Actually, the relative thinness of their cranial bones allows of fissuring under lesser degrees of stress than is the case with the adult. In infants cephalhæmatoma overlying the fracture is common.

A baby girl of 3 weeks had her head knocked against some railings and was then dropped on the pavement. She was stunned, had a mild fit, but showed no abnormal signs except a swelling on the side of the head; she made a good recovery. X-rays showed a long horizontal fracture of the right parietal bone.

A baby girl of 4 months fell out of her pram, was "knocked out", and developed a large fluctuant left-sided cephalhæmatoma; X-rays showed a fissure fracture of the left parietal bone. There were no other symptoms.

CLINICAL FEATURES FOLLOWING HEAD INJURY

Concussion.—Up till recently the rival views on the production of concussion were the *mechanical* or "jarring effect" (Russell) and the *vascular*. The latter, the more popular, did not postulate hæmorrhage but presupposed a sequence of deformity of the skull.

ntriculography.—In the later stages of recovery in cases of the *closed* type, if Jacksonian fits develop or local paralysis persists ventriculography can be of great value in determining the need or otherwise of operation.

What are the indications for operative exploration when signs of cerebral damage are present? The view that vascular lesions commonly cause intracranial damage in head injury has been largely altered. Often, when physician and surgeon agree that exploration is needed, no collection of encapsulated blood is found. Local oedema and confusion, more often than bleeding, cause symptoms. Operation should only be considered when signs of deterioration in the patient's condition appear and persist, such as slowing of pulse and respiration, persistence or increase of local paralysis and Jacksonian fits; following evidence of ventricular shift revealed by ventriculography.

SEQUELÆ OF HEAD INJURIES IN CHILDREN

The child escapes the late effects of head injury more than the adult. Headache, so prominent a feature of the post-concussional state in adults, passes off rapidly in the child. A period of reduced mental activity, initiative, or concentration is rare. Most cases return to school after a sufficient rest do not show scholastic deterioration, possibly because the intellectual processes in the child's brain are less advanced. In cases of more severe injury mental alteration does occur.

A boy of 5 years had a fractured skull from a fall in the road, was unconscious, then delirious and vomited. He appeared to recover after at first dragging his right foot in walking. Two years later he was brought to Great Ormond Street because of screaming fits, nightmares, violent outbursts of temper, being "right out of control", and occasionally going stiff, when he did not recognize his parents. A second X-ray two years after the injury still shows a widely ununited fracture of the right frontal bone. Extradural clot may be present. Here ventriculography is indicated.

The rarity of residual paralyses and epilepsy at all ages, except in penetrating wounds of the skull, is striking considering the degree of change found in the brain in cases of head injury with apparently complete recovery, dying from other causes some time after the injury. Greenfield (1939) has described cortical defects at the summits of convulsions where severe oedema has occurred, and demyelination in the underlying white matter.

Two unusual instances of late sequelæ in children are as follows. Two boys aged 5 and 7 years respectively gave histories of head injury with fissure fractures extending to the base of the skull. Both had initial unconsciousness but made good immediate recoveries. After a period of roughly three months, obesity with bulimia developed in one and diabetes insipidus in the other. The lesions presumably were basal hæmorrhage with slow organization of blood-clot and arachnoiditis in the region of the tuber cinereum and infundibulum.

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Mr. J. E. A. O'Connell: Dr. Wyllie has pointed out that the incidence of cerebral concussion is considerably lower in Mr. Northfield's group of children with closed head injuries than in a series of patients of all ages referred to by me in a recent paper (*Lancet*, 1941, ii, 719). The real explanation of this difference in the incidence of concussion in the two groups lies in the differing classifications adopted by Mr. Northfield and myself. I have excluded scalp wounds as well as all deeper wounds from the group of closed injuries and consequently all the patients in this group of mine were brought to hospital either because of unconsciousness following a blow on the head or some complication arising subsequently. Obviously patients who had neither a scalp laceration nor a period of unconsciousness after a head injury would be very unlikely to come to a hospital or even to see a doctor.

A case of delayed onset of hæmorrhage, subarachnoid. A girl of 11 months, having fallen from her pram six months and three weeks prior to admission, had attacks of going limp and unconscious with increasing frequency. On admission she was unconscious, temperature 101° F., bulging fontanelle, numerous hæmorrhages, some subhyaloid, in both fundi, but no neurological signs. The cerebrospinal fluid was uniformly and heavily blood stained with an initial pressure of 300 mm. of water. She improved and when seen one month later was in perfect health.

(5) *Cedema of the brain*, probably the commonest complication of all forms of cerebral injury, occurs locally around a traumatic vascular lesion, or at the various poles, but seldom becomes generalized. Symptoms of the cerebral swelling—stupor, irritation, paralyses, unconsciousness—usually appear on or after the fourth day following the injury.

(a) ? *Cedema*. A boy of 6, an air-raid casualty, was concussed for a short period and had an abrasion of the right forehead without visible fracture. Ten days later he again became drowsy, vomited, then unconscious, the pulse-rate being 60-80 for seven days. The cerebrospinal fluid was normal (pressure not estimated). He made a good recovery.

(b) ? *Cedema and hæmorrhage*. A boy of 4 years fell on the back of his head from a roundabout on to concrete. He cried bitterly, was not unconscious, but had severe pains in the back of his head, neck and spine. A transverse fracture of the occipital bone was found. Two days later he walked badly on his left leg, held his head to the left and began to "fall down". The headache was worse and he vomited and was drowsy. After a further two days he had weakness of the left arm as well as the leg. He had six more fits always commencing in the left leg. Recovery has been apparently complete.

(6) *Sepsis*. A case of compound fracture with hæmatomata of the right orbital region and of the scalp in a boy of 10 years. A fracture was not seen but presumably one existed and involved one of the bony sinuses. The boy was not unconscious but restless and irritable, with vomiting, severe headache and pain in the neck. On the day after the accident—a fall from a bicycle—the temperature rose to 104° F. That day on lumbar puncture the initial fluid pressure in millimetres of water was 400; 180 four days later. The cell count in spite of sulphonamides was between 2,090 and 340 for eighteen days, chiefly polymorphonuclear leucocytes; culture: pneumococci. He made a complete recovery.

Pupillary changes in head injuries.—Pupillary inequalities and some degree of nystagmus are often present following a head injury. Apart from bilateral dilatation due to high intracranial pressures these changes need not be interpreted as evidence of pressure or damage to the hemispheres. They depend upon concussion possibly with petechial hæmorrhages in the brain-stem. In violently arrested movement of the head a forceful downward movement of the cerebrospinal fluid can produce petechiæ in the region of the aqueduct, or a contusion of the quadrigeminal plate against the tentorium cerebelli will interfere with normal pupillary functions.

Diagnostic aids. Lumbar puncture.—In all cases at least one diagnostic puncture should be made. In a large proportion of head injuries some blood or staining of the fluid is found and is of little significance unless the quantity is excessive. Then a subarachnoid hæmorrhage is suggested and a lumbar puncture on every second day is advisable.

Much attention lately has been given to pressure estimations of the fluid and to measures of reducing it when high by means of dehydration. My impression is that low readings are more common than high ones but that usually they are within normal limits. High pressures in head injuries are at about a level of 200 mm. of water, and rarely reach 300 mm., not being comparable with the high levels reached in cases of cerebral tumour and meningitis. Ritchie Russell (1932) found no evidence that increased intracranial pressure itself caused death in head injuries. McKissock and Brownscombe (1941) consider that lumbar puncture reduces headache and does not facilitate renewed bleeding, but agree that there is no constant relationship between abnormal clinical signs and the state and pressure of the cerebrospinal fluid.

Ventriculography.—In the later stages of recovery in cases of the *closed* type, if Jacksonian fits develop or local paralysis persists ventriculography can be of great value in determining the need or otherwise of operation.

What are the indications for operative exploration when signs of cerebral damage are present? The view that vascular lesions commonly cause intracranial damage in head injury has been largely altered. Often, when physician and surgeon agree that exploration is needed, no collection of encapsulated blood is found. Local œdema and contusion, more often than bleeding, cause symptoms. Operation should only be considered when signs of deterioration in the patient's condition appear and persist, such as slowing of the pulse and respiration, persistence or increase of local paralysis and Jacksonian fits; or following evidence of ventricular shift revealed by ventriculography.

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I believe that pupillary changes after head injuries are of great significance. Unilateral dilatation and fixation of the pupil on the same side as an expanding intracranial lesion has been clearly shown by Reid and Cone to be due to the herniation of the hippocampal gyrus through the tentorial opening with stretching of the oculomotor nerve beneath it. After injury such pupillary changes are of the greatest value in localizing an intracranial hæmorrhage which will be on the side of the dilated and fixed pupil.

Section of Laryngology

President—E. D. D. DAVIS, F.R.C.S.

[February 6, 1942]

DISCUSSION ON THROAT AND NOSE MANIFESTATIONS OF BLOOD DISEASES

W. M. Mollison: The diseases chosen for consideration are: (1) Bleeding of the newborn. (2) Essential thrombocytopenia or purpura hemorrhagica. (3) Polycythæmia vera. (4) Hæmophilia. (5) Hereditary telangiectasia or Osler's disease. (6) Agranulocytosis. (7) Leukæmia. (8) Achlorhydic anæmia. (9) Avitaminosis. (10) Glandular fever. Though the first three of these diseases exhibit bleeding from the nose and gums, in the absence of any local cause the pædiatrician takes over the case.

(4) *Hæmophilia* has generally declared itself in the boy before the laryngologist is consulted. All operations are to be avoided. In suspected cases full examination of the blood must be carried out before any operation. Even a normal clotting and bleeding time and normal platelets do not always save the surgeon from disaster.

(5) *Osler's disease* is rare but embarrassing; bleeding occurs spontaneously from the skin and mucous membranes; several members of the family are similarly afflicted. The following case gives a typical picture: A man aged 40 gave a long history of severe epistaxis. This occurred daily for three weeks, resisting all accepted treatment, but the resulting anæmia may have caused cessation of the bleeding. On recovery from anæmia bleeding again started; this cycle had recurred over a period of years. Since cauterization had failed it was decided to use surgical diathermy under an anæsthetic; bleeding was extremely difficult to stop and there was no lasting effect. His father, an uncle, brother and a sister suffered from epistaxis.

(6) *Agranulocytosis*, as its name indicates, is a blood disease in which there is absence or great diminution of the granular cells in the blood. It occurs after the ingestion or injection of certain drugs—pyramidon, sulphanilamides (though not till 30 g. have been taken), sanocrysín, arsenical compounds and benzol. It is seen also after exposure to deep X-rays. Owing to the inflammation and ulceration of the tonsils, palate and gums the disease is called agranulocytotic angina. The disease has a high mortality. The patient is very ill with a high temperature often out of proportion to the condition of the pharyngeal lesions; death may take place in a few days. There is too, a chronic form in which treatment leads to temporary improvement in the blood picture; cases have lived for two years or more. Even slight operations are apt to produce serious relapses with extensive ulceration in the mouth: in one case extensive ulceration of the palate, gums and tonsil followed extraction of a tooth. Alcohol increases the symptoms; in some cases symptoms are connected with menstruation; a case has been seen as a complication of enteric fever. Rigors produced in the course of the disease lead to an enormous leucocytosis with cure. In a case recently published by Cross a leucocytosis of 40,000 followed a third blood transfusion for agranulocytosis and cure was immediate and permanent. One case of pemphigus has been seen in which prontosil caused disappearance of the skin lesions but was followed by agranulocytosis and on withdrawal of the prontosil the pemphigus recurred with fatal results.

(7) *Leukæmia* produces severe ulceration of the tonsil and is only distinguished from agranulocytosis and other tonsil ulcerations by a blood examination. Prognosis is fatal.

(8) *Achlorhydic anæmia*.—Professor Wiatts wrote an admirable paper on this subject analysing 50 cases; the symptom of dysphagia was one of the least frequent exhibited. The disease is a fruitful cause of chronic invalidism in women; its age-incidence is 30 to 50 (compared with 45 to 60 in Addison's anæmia). Most of the cases occur in the asthenic type of woman, her hair prematurely grey contrasting with dark eyebrows; the skin is pigmented but not icteric; anæmia is not obvious till a blood-count is done; the patients are often nervous and difficult; some have never felt well since girlhood, others date the onset from influenza or pregnancy. In 44 of the 50 cases achlorhydria was complete while in the remaining 6 faint traces only of free acid were present. Treatment consists in giving large doses of iron (iron and ammonium citrate) up to 2·40 grains a day, and is successful but return of hydrochloric acid in the gastric juice is never seen. The tongue may be bald towards the tip and dorsum. The dysphagia was only slight and transient in 4 cases, severe and the dominant symptom in 2. In my experience complete removal of the teeth has been carried out in almost all cases.

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of the blood, or, if necessary, of the bone-marrow, will usually provide a certain differentiation.

The atrophic lesions include the glossitis, stomatitis, and pharyngitis that are associated with the deficiency anæmias, in which there is typically an atrophy of the surface epithelium; sometimes of underlying muscle accompanied by chronic inflammation and occasionally ulceration. There are many points of similarity between pellagra, sprue, steatorrhœa, pernicious anæmia, the tropical macrocytic anæmias, and the anæmia associated with gastro-intestinal operations and diseases. In all these there is a tendency to develop glossitis, stomatitis, and disorders of the gastro-intestinal tract, changes in the nervous system, pigmentation and lesions of the skin, anæmia, usually macrocytic, leucopenia and thrombocytopenia. Moreover all these conditions have been reported to respond favourably to treatment with crude liver, and their manifestations may be produced experimentally by withholding the factors contained in autolysed yeast known collectively as vitamin B. While pernicious anæmia will respond to a highly purified liver extract, the other conditions often fail to do so, though responding to crude liver or autolysed yeast.

It may be supposed, therefore, that these diseases are the result of a deficiency of the factors contained in crude liver, and derived from autolysed yeast, of which at least one, the anti-pernicious anæmia factor, requires the collaboration of Castle's intrinsic factor for its synthesis.

In pernicious anæmia the deficiency is conditioned by the absence of the intrinsic factor, and may be limited to the anti-pernicious anæmia factor as represented for the moment by anahæmin. The failure of other conditions, to respond to anahæmin shows that these manifestations result from the lack of some other, or more than one factor.

The group of factors required probably includes nicotinic acid, riboflavine, the so-called extrinsic factor and others as yet unidentified. The lesions produced by their deficiency are almost certainly the result of the lack of these substances in the enzyme systems on which the living cell depends for its metabolism. The oxidation reduction processes, from which the energy for vital activity and growth is derived, are the result of such enzyme chain reactions. The first link of this chain is dehydrogenase, specific for each particular substrate, followed by co-enzymes 1 and 2, flavine enzyme and cytochrome. The co-enzymes contain nicotinic acid, flavine enzyme contains riboflavine, and cytochrome contains iron. The absence of any one of the links of this chain would bring the whole vital process to a standstill, and the partial deficiency of any one is likely to impair functional activity and growth.

If such a deficiency exists, evidence of it might be expected in those parts of the body where there is normally great activity, particularly in the production of new cells. The most obvious example of such tissue is the bone-marrow. Here there is a continual production of relatively enormous numbers of cells, roughly 50 g. of red cells and leucocytes, and an unknown quantity of platelets, every day. Deficiency of one or more of the factors required for growth and maturation results in a partial or complete breakdown in the process of production with consequent anæmia or leucopenia. Bone-marrow studies in the nutritional macrocytic anæmias reveal a very characteristic picture, that of arrested maturation. The marrow is full of primitive cells, most of which seem unable to develop beyond a certain point. The process of maturation when it does occur is distorted, resulting in the production of abnormal red cells and leucocytes. The disorder affects the red cell, white cell, and platelet precursors equally and in the peripheral blood the leucopenia and thrombocytopenia are usually as pronounced as the anæmia. If cells so dissimilar are all affected by lack of one or more of the maturation factors it is probable that other types of cell undergoing rapid production and maturation will be affected also. Witts (1932) has already compared the development of the cells of the bone-marrow to the development of the cells of the skin. Undoubtedly the skin and mucous membranes must provide a continual supply of new cells to replace those lost from the surface. Atrophic skin changes occur in the diseases we are considering, being most pronounced in pellagra, and least in pernicious anæmia, but the skin, being protected by its horny layer, is less susceptible to injury than the mucous membranes.

The epithelium of the mouth has probably to contend with more injury, chronic irritation, gross infection, violent changes in temperature and the application of noxious substances than any other part of the body. It would not be surprising therefore that any retardation of the continual production and maturation of new cells were followed by soreness, atrophy, or even ulceration. The changes found in the deficiency anæmias may be reasonably explained on this basis of general arrest of maturation. That each of the manifestations may occur separately, and will respond to treatment with crude liver suggests that each is the direct result of deficiency and that they are not inter-dependent. Whether it is the skin, gastro-intestinal tract, bone-marrow or nervous

In 1919 Brown-Kelly described a symptom-complex in women complaining of dysphagia: spasm at the entrance to the œsophagus; smooth tongue; fissures at the corners of the mouth and pale pharyngeal mucous membrane. Œsophagoscopy showed a small circular lumen which could be opened easily with much relief. Similarly Paterson described a clinical type of dysphagia with spasmodic dysphagia and/or superficial glossitis; he noted also changes in the mucous membrane which tended to make it crack on the passage of a tube. The narrow lumen just below the cricoid appeared to be due to spasm of the crico-pharyngeus muscle and this led to the suggestion that this muscle was unable to relax.

There seems to be some tendency towards the development of malignant disease at the site of the narrowed lumen or perhaps on the posterior wall of the cricoid. Authorities are by no means in agreement about the frequency of this complication. Some speak of it as frequent but Vinson and Lamb consider it rare, and I incline to this view.

(9) *Avitaminosis* supplies two diseases with nose and throat manifestations—scurvy and rickets. Scurvy occurs usually between the ages of 6 months and 3 years; it causes hæmorrhages under the periosteum. When the swelling occurred over the frontal bone a mistaken diagnosis was made of acute osteomyelitis; incision revealed no pus and some lemon juice cured the patient. Laryngismus stridulus is a manifestation of rickets and may be confused with other causes of stridor.

(10) *Glandular fever* is characterized by sore throat and enlarged glands, at first cervical but later epitrochlear, axillary, inguinal and mediastinal; it is the last group that produces cough and perhaps stridor. The blood shows a leucocytosis up to 60,000 of which 80% are lymphocytes.

R. G. Macfarlane: I have subdivided the buccal manifestations of the blood disorders morphologically into hæmorrhagic, infiltrative, atrophic and ulcerative.

The hæmorrhagic lesions are usually manifestations of one or other of the hæmorrhagic states, associated with a vascular defect, or with faulty blood coagulation. In the hæmorrhagic purpuras there is usually a demonstrable vascular defect, revealed by decreased capillary resistance or a prolonged bleeding time which is often, but not always, accompanied by thrombocytopenia. These purpuras may be idiopathic, or secondary to toxæmia, aplastic anæmia, or leukæmia, and the type of bleeding they produce is characteristic. There is often persistent oozing from apparently intact mucous membranes, particularly of the nose and gums, which may in fact be the only symptom of the underlying disease. Bleeding into the tissues is in the form of petechial hæmorrhages or superficial ecchymoses, and deep hæmorrhages are rare. Hæmorrhagic telangiectasia is an example of a localized vascular defect. Lesions in the nose may produce such persistent epistaxis that a severe anæmia develops, but in the mouth they do not often bleed unless injured.

The other type of hæmorrhagic diathesis is due to defective blood coagulation, and includes hæmophilia and hypoprothrombinæmia. In these states uncontrollable bleeding as a rule follows injury and there is a liability to form extensive hæmatomata. I have seen a hæmophilic boy die of asphyxia following hæmorrhage into the base of the tongue before anything could be done to save him, and another who on several occasions developed hæmorrhage into the tissues of the throat, and required intubation, to prevent the impending asphyxia.

Infiltrative lesions are the result of deposition or proliferation of abnormal cells. In the leucoses, the cell type affected may infiltrate the mucous membranes of any part of the nose, throat, mouth or larynx. This infiltration may be diffuse or localized. Ulceration of the surface follows such invasion of the mucous membranes, usually in the acute leucoses, and particularly in the monocytic type in which angina and gingivitis may be the presenting signs. The lesions are complicated by the hæmorrhagic purpura that is frequently the terminal event in these diseases. There is no clearly defined boundary between the leucoses and frankly malignant states with a leukæmic blood picture. Leukæmic plasmocytoma may arise from any of the bones of the skull; leucosarcoma, involving the tonsils may have a leukæmic blood picture; and chloroma, with the blood picture of myeloblastic leukæmia, may invade any part of the skull and upper respiratory tract.

The origin of the mouth and throat lesions of glandular fever is more obscure, as autopsy material is rare. They are probably due to infiltration by the abnormal cells present in the blood, and Glanzmann (1930) considered that lymphocytic infiltration of the mucous membranes in some of his cases are responsible for them. Throat lesions occur in about 80% of cases and angina is not uncommon. The throat lesions of acute leukæmia, agranulocytosis, glandular fever and acute infection may be very similar. Examination

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The President said that hæmophilia was probably the commonest blood disease encountered in routine work. Hæmophilia seemed to vary; at one period there might be very little bleeding, and at another time the patient was very susceptible. He had a child patient whom he knew to be a hæmophiliac and who had otorrhœa following measles and all the classical signs of an acute mastoid. He resolved, with a little misgiving, to operate. About three-quarters of an hour before operation a hypodermic injection of atropine was given. When the child was ready for operation he was still bleeding from the needle puncture, and therefore the operation was not done. The mastoid cleared up and the child recovered, but died later from hæmophilia.

Another type of case was acute leukaemia. During his long experience at the Royal Dental Hospital he had seen perhaps three of these cases. Attention was drawn to them by reason of a septic condition of the mouth and gums. Teeth were extracted, but bleeding continued, and the patient was admitted to hospital very ill, with a temperature of 104° or 105°, and ultimately died in about five days of septicæmia. Blood examination proved these to be cases of acute leukaemia.

Another condition which was seen frequently was an ulceration in the mouth or on the tonsil of which the diagnosis was uncertain. The Wassermann reaction was negative. It might be an agranulocytosis or some other obscure blood condition. Some cases of acute tuberculosis which were doing badly, with a painful superficial ulceration of the pharynx, epiglottis and larynx, might be mistaken for agranulocytic angina. It would be very easy to confuse a case of acute tuberculous ulceration of the pharynx with one of agranulocytosis. The white cell count in some cases of pulmonary tuberculosis was very low.

R. L. Flett said that caution was necessary when removing tonsils and adenoids in children with lymphatic leukaemia. In one case for removal of enlarged tonsils and glands in the neck in which he himself was not actually concerned the child died on the table and was found post mortem to have lymphatic leukaemia. In certain cases of leukaemia he had seen numerous ulcers down the pharynx and on the epiglottis, and there was some blood staining around each ulcer. He had also seen a Hodgkin's deposit on a vocal cord.

With regard to secondary anæmias where it was necessary to dilate the upper end of the œsophagus, he thought they did the cases some good, but a large number of those with atrophic mucous membrane began to form carcinomas afterwards. Recently he had dilated one of these strictures, secured a very good dilatation, and got the œsophagoscope to go past the stenosed area. A fortnight later he was called to see this patient because she was unable to swallow and was very ill. He thought he must have produced mediastinitis from his over-extensive dilatation, but when the patient died about a fortnight afterwards, a carcinoma was found about 3 in. from the cardia. The stenosed area was shown to be a healing area with a diamond-shaped space where it had been broken.

R. G. Macbeth, in expressing his appreciation of the papers, said that it was interesting to learn that avitaminosis perhaps took such an important part in blood diseases. Had Dr. Macfarlane noticed any increased tendency to bleeding from minor operations, such as tonsils and adenoids or removal of teeth, also had they noticed more cases of epistaxis associated with lowered vitamin intake? He thought he had been having more trouble during the last twelve months from reactionary hæmorrhage, and the dental surgeons said that they were encountering the same difficulty.

They were all accustomed to meet cases of, what he called, "normal abnormalities" such as the child admitted for tonsils and adenoids whose mother would say that, "of course, he always bled excessively if he cut his finger"; but in the majority of these cases there was an entirely normal bleeding time and clotting time. Occasionally these cases seemed to lose rather more blood at operation, but it was a clinical matter and could not be ascertained in absolute terms.

The question of female bleeders, otherwise healthy, also suggested itself. They were told that hæmophilia was transmitted on the female side but not inherited by the female. One occasionally found women who bled a good deal, and the nursing staff at his hospital were convinced that red-haired women always bled, and the anaesthetists sometimes gave their authority to the same story. A final question was: Should tonsil operations be avoided during the menstrual period?

system that is mainly affected depends probably on variations in diet, absorptive and digestive efficiency, and the requirements of the tissues concerned.

Iron deficiency may be accompanied by more specific changes resulting in a reduction of the hæmoglobin content of the red cell, and disordered development of the nails, both of which can be corrected by treatment with iron. Iron is also a factor in general metabolism, as it has already been shown that it is a constituent of cytochrome. The glossitis, atrophy of the lingual papillæ and pharyngitis may all be manifestations of this particular deficiency. The dysphagia that completes the Paterson-Kelly syndrome occurs in about 15% of iron deficiency anæmias. It has been reported in a few cases of pernicious anæmia, and is of importance because the atrophic changes and chronic irritation may predispose to carcinoma, particularly of the post-cricoid area.

Ulcerative lesions of the mouth and throat may be merely an exaggeration of the atrophic changes just described. I have recently been able to observe a case of nutritional anæmia with acute ulceration of the mouth, leading to perforation of the lower lip. The patient was a girl aged 14 years, with a previous history of coeliac disease. On admission she was desperately ill, with fever, and severe ulceration of the mouth. There was profound anæmia, the red cells numbering 1.6 million per c.mm., hæmoglobin 26% and only 600 neutrophils per c.mm. The platelets were 50,000 per c.mm. Bone-marrow examination showed a cellular marrow, with almost complete arrest of erythrogenic and leucogenetic elements. The primitive red cells were definitely megaloblastic, and the whole picture resembled that found in pernicious anæmia. She was transfused and given liver and marmite, and in seven days the neutrophils rose to over 5,000 per c.mm., the platelets to 300,000 per c.mm., and the ulcers, one of which had perforated, were beginning to heal. She left hospital perfectly well in a month. A second case very similar to this was seen shortly afterwards, this time in a woman of 32 who had suffered from rickets and chronic diarrhoea. The ulceration was not so severe, but again there was an extreme degree of anæmia and leucopenia, and a megaloblastic hypercellular marrow; a rapid improvement followed blood transfusion and a course of liver and marmite. A similar condition has been described by Bowen-Davies (1934).

These cases introduce the problem of agranulocytosis, and the question whether the mouth lesions were due primarily to the maturation factor deficiency, or to the absence of granulocytes. Undoubtedly the granulocytes play an important part in limiting the spread of infection, once a break in the surface has occurred. Agranulocytosis superimposed on the atrophic stomatitis of the deficiency anæmias might be expected to result in severe angina. The granulocytes may be greatly reduced, however, without the occurrence of angina. I have seen cases in which neutrophils numbered 100 per c.mm. or less as a result of aplastic anæmia, X-ray irradiation and Felty's syndrome, without mouth lesions. Yet in toxic agranulocytosis there may be acute angina with 2,000 or more granulocytes per c.mm. (Plum, 1937).

Many cases of toxic agranulocytosis have followed the use of amidopyrine and related compounds, including the sulphanilamide derivatives. These drugs may produce in sensitive persons arrest of maturation or even disappearance of the granulocyte precursors in the bone-marrow. The red cells are usually stated to be unaffected, but in my experience, fatal cases following the use of sulphanilamide developed a profound anæmia and thrombocytopenia before death.

The possible mechanism of the toxic action of sulphanilamide in this respect might be considered more closely, because the effects of the drug on living cells have been carefully studied. Sulphanilamide not only inhibits the growth of leucocytes in certain people, it inhibits the growth of certain bacteria. Investigation of the latter action has shown that sulphanilamide blocks the metabolic process of the organism by interfering with a growth factor identified as para-aminobenzoic acid. This latter substance has a very similar structure to the sulphanilamide, and it has been suggested that the inactive sulphanilamide is incorporated, in mistake, so to speak, for the similar, but active para-aminobenzoic acid in an essential enzyme chain reaction that is thus brought to a standstill. The action can be inhibited by excess of para-aminobenzoic acid.

Dr. R. H. Mole has pointed out that sulphanilamide may have a similar action on developing leucocytes, and has suggested the use of para-aminobenzoic acid in the treatment of agranulocytosis. There is some evidence to favour the view that some at least of the toxic effects of sulphanilamide may be due to interference with the enzyme systems in which the vitamin B factors are concerned. Thus abnormal excretion of porphyrins occurs both in pellagra and following the use of sulphanilamide. Sulphanilamide produces a neuritis in animals that may be prevented by giving vitamin B, and it also produces skin lesions that may resemble those of pellagra. Vitamin B deficiency has already been shown to produce leucopenia and stomatitis, and it is possible that toxic agranulocytosis following sulphanilamide may be an extreme form of the same process. If this be so, therapy should be directed towards the supply of the deficient factor.

Section of Therapeutics and Pharmacology

President—R. D. LAWRENCE, M.D.

[February 10, 1942]

DISCUSSION ON VITAMINS AND HÆMORRHAGIC STATES

Dr. Harold Scarborough: The literature dealing with the relation of the various vitamins to the different parts of the hæmostasis mechanism of the body is confusing, not to say contradictory. This unsatisfactory state of affairs is due to the injudicious selection of experimental material and to infrequent examination of the material selected. In order that future results shall be acceptable the following point should constantly be in the minds of those investigating these difficult and important problems: that "chronic" rather than "acute" forms of disorder be selected for investigation; and that "idiopathic" and not "symptomatic" cases be studied; that cases should be under close observation for at least fourteen days (preferably longer) before the effect of any therapeutic procedure is examined; and that all cases should give a history of excessive or abnormal bleeding extending over at least eight weeks, preferably longer; that female cases be excluded; and that observations should be made frequently since alterations of the various functions investigated sometimes occur with great rapidity and may be temporary.

The number of cases in any one centre suitable for investigation by these criteria is not large and my suggestion, therefore, is that comparatively little is likely to be achieved except by concentrated and, especially, co-ordinated attack.

Vitamin D.—The first known report of fatal bleeding in association with jaundice was apparently made by Wedels in 1683, since when it has been increasingly recognized that a proportion of jaundiced patients exhibit a latent hæmorrhagic tendency, which is liable to become manifest especially after operation. The observations on the relation of vitamin D to this condition which were originally made by Ivy and his associates (1935), and which have been confirmed by Boys (1937) and also by Johnston (1937) stand in danger of being swept away in the spate of literature on vitamin K.

Ivy developed a new technique for the determination of the bleeding time, the essential point of which is that a venous occlusion of 40 mm.Hg is applied through the cuff of a sphygmomanometer before the test is made. This manoeuvre has certain consequences, viz. it may cause the bleeding time to be prolonged when the more usual technique (Duke) gives normal values, and, as a result, it becomes possible to demonstrate that vitamin D can reduce the bleeding time in patients with jaundice and certain other conditions. It is plain that a number of things may happen following the venous occlusion. One can imagine, for example, the increased intracapillary pressure exerting a greater tension on any clots that may be formed in the ruptured capillaries, a tension sufficient, if the clots be abnormally fragile, to force them from the capillaries and so prolong the bleeding time. On this basis vitamin D might be supposed to influence directly, or indirectly, the mechanism of clot formation. However, this explanation, which is evidently favoured by Ivy himself, raises the whole problem of the relative importance of the various factors taking part in the arrest of hæmorrhage from a small injury—a problem which itself awaits a complete answer.

One may recall in this connexion that Macfarlane (1941) has described structural and functional abnormalities in the capillaries in certain of the bleeding diseases, and I have frequently observed that venous occlusion may exert a profound effect on capillary resistance. There thus arises the attractive possibility that vitamin D may be concerned with the functional activity of the capillary walls.

Apart altogether from such considerations vitamin D is capable, in certain circumstances, of increasing the serum Ca, although the mechanism by which this increase is brought about and the fraction or fractions of the serum Ca which are so increased, are still undecided. A part of the serum Ca—and it is not at present clear which part—fulfils an indispensable function in blood coagulation and it may be on this basis that the phenomenon mentioned is to be explained. It is generally held that the non-diffusible Ca fraction consists of Ca bound to protein but it has been suggested (Watchorn and McCance, 1932) that it is to phospholipoid rather than to protein that the Ca is bound and there is experimental support for this view. Since there is evidence that vitamin D can alter the proportion of non-diffusible Ca in serum, then vitamin D might be supposed to influence the amount of Ca bound to phospholipoid. These points may then be considered in the light of the observations of Ferguson (1936, 1937) who believes that

J. F. Simpson said that it was quite true that one of his colleagues at a Royal Air Force hospital was disturbed during a short period by an unusual number of tonsillar hæmorrhages. He carried out a series of investigations on the vitamin content of the blood and put a number of the patients on to ascorbic acid treatment. The investigations did not show anything conclusive.

I. G. Robin said that recently a man aged 60, who had been treated at many hospitals for epistaxis from telangiectases, came in very ill with hæmoglobin under 25%. He had gradually improved on big doses of iron, vitamin K and nasal ionization and liquid paraffin drops. The mucous membrane of the nose was very dry and he bled from many points in the nose. The speaker was not sure how long he should go on ionizing; he was sceptical of the true value of the vitamin therapy.

Recently he diagnosed the Paterson-Kelly syndrome in a woman aged 50 who had had attacks of dysphagia several times and had been admitted to another hospital on a previous occasion. Before doing œsophagoscopy he got a physician to see her and he diagnosed pellagra; the condition cleared up completely on vitamin B. She had a low hydrochloric acid stomach content but no skin changes. The only changes were in the mouth, a very atrophic tongue and marked dysphagia.

J. C. Hogg said that there was a condition known as reticulosis which was characterized by ulceration in the upper air passages. It was apparently a disease of the reticulo-endothelial system, but perhaps Dr. Macfarlane could tell them whether it was, strictly speaking, a blood disease or not. The first case of the kind in his experience had Vincent's organism and looked like a typical Vincent's infection of the tonsils, though every investigation proved negative. His blood was examined by different pathologists, biopsies were taken of glands in the neck, but all were negative, and only when the patient died six months afterwards was the diagnosis established. He had ulceration of his larynx, pharynx, œsophagus, and intestines, and he believed of his stomach as well. Such cases were rare, but he had lately seen a similar case.

The President asked whether it was true that in cases of hæmophilia the clotting time was normal. The family history was very important.

Mr. Robin's case resembled multiple telangiectases of the septum. In one or two cases he had found a 5% solution of quinine and urethane injected between the cartilage and the mucoperiosteum prove successful.

This treatment was recorded by G. H. O'Kane (*J.A.M.A.*, 1938, **III**, 242).

R. G. Macfarlane, in reply, said that in glandular fever sometimes the hæmatologist could not help very much in the diagnosis. The Paul-Bunnell reaction might be negative in 30 to 40% of cases and in these diagnosis could only be made on the abnormal type of lymphocyte occurring in the blood, which might not be present in very large numbers.

Several speakers had raised the question of the diagnosis of hæmophilia. In true hæmophilia he would have said the diagnosis was comparatively easy, because in almost all cases (about 98%) over the age of 5 or 6 years hæmorrhage into the joints occurred and was a characteristic feature of the condition. There was considerable variation in severity from time to time, there might be a normal coagulation time by the ordinary technique, but it was most important that the investigation be done with extreme care. If venipuncture were bungled there might be a normal coagulation time in hæmophilia, whereas if it had been done properly, the time would be four or five times the normal.

True hæmophilia, he believed, never occurred in females, but there was undoubtedly a hæmorrhagic tendency in some of the females through whom hæmophilia was inherited. He had seen one case of pseudo-hæmophilia in a woman in the sense that he could make no distinction between the condition in her case and that of true hæmophilia, but this was very rare indeed.

Unfortunately the tendency to bleed was not limited to the typical hæmophilic. There were undoubtedly people who bled idiopathically, people in whom it was not possible to demonstrate any change whatever in the hæmostatic mechanism, but who were liable to bleed after operation or injury. In one woman who bled for six weeks after a small cut of the finger it was not possible to demonstrate any change in the hæmostatic mechanism.

Menstruation might be regarded as a hæmorrhagic diathesis. At the menstrual period there was a depression in the number of platelets and sometimes a prolongation of bleeding time, and although it was of no practical significance in the general run of cases it might be advisable to avoid the menstrual period for operation if possible.

Reticulosis was a collective term covering a large number of conditions, including Hodgkin's disease, so-called "atypical Hodgkin's disease", the leukæmias, the chloro-leukæmias, and the leuko-sarcomas, all of which might have a leukæmic blood picture. Reticulosis might not always have a typical blood picture. Hæmatologists could not as a rule diagnose reticulosis unless there were characteristic cells in the blood-stream.

especially in the arms. There had been no previous or family history of hæmorrhagic disease and the dietary history was not significant. On examination the patient was a fairly well-nourished, healthy woman. Nothing abnormal was detected on examination of the cardiovascular system. The blood-pressure was 140/80. Renal function was satisfactory. Hæmatological examination revealed no cause for the hæmorrhagic tendency. The capillary resistance was found to be low.

A diagnosis of vascular purpura (purpura senilis) was made.

Fig. 1 (Case I) is a record of the capillary resistance of the patient determined in three separate areas of skin by a method I have already outlined (Scarborough, 1941b). The development of petechial bleeding is indicated at the bottom of the chart. The capillary resistance remained low during the preliminary period of twenty-six days, during which time a hæmorrhagic tendency was manifest. Administration of a preparation

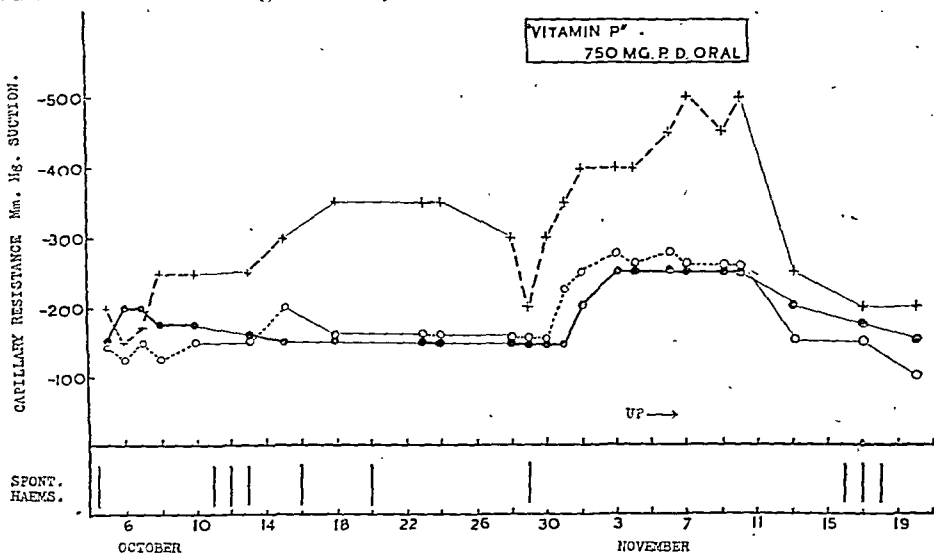


FIG. 1.—Chart of capillary resistance determined in terms of the least negative pressure (mm.Hg) required to produce a single petechia in each of three areas of skin. The development of spontaneous petechial bleeding is indicated below the chart by vertical lines. The effect of administration of a vitamin P preparation on capillary resistance and petechial bleeding is shown.

containing vitamin P coincided with an elevation of the capillary resistance and an absence of the manifest bleeding tendency in spite of the fact that the patient was allowed to get up during this period. With the cessation of vitamin P therapy the capillary resistance fell to its original level and the hæmorrhagic tendency returned.

CASE II.—A female, aged 65, for about ten years had complained of a marked tendency to bruising but of no other symptom. There were invariably one or more bruises on each arm and usually several on the legs. Ecchymoses could be readily induced by the "pinch" test. She had never complained of purpura but, on close examination on several occasions before the investigation began, petechiae were noted in both upper and lower limbs. There was no previous or family history of bleeding and the dietary history appeared to be satisfactory. Nothing abnormal was detected during examination of the cardiovascular system. The blood-pressure was 135/64. Renal function was not impaired. Hæmatological examination revealed no cause for the bleeding tendency. The capillary resistance was low. A diagnosis of vascular purpura (purpura senilis) was made.

TABLE I.—CAPILLARY RESISTANCE. MM.HG. NEGATIVE PRESSURE.

| | May | June | July | August | October | December |
|----------|-----|------|------|--------|---------|----------|
| Area I | 150 | 400 | 300 | 400 | 150 | 150 |
| Area III | 150 | 400 | 300 | 350 | 125 | 150 |
| Area V | 200 | 450 | 350 | 400 | 200 | 225 |

Bruising

Bruising

PERMIDIN
3 × 0.25 g.p.d. by
mouth for 40 days.

PERMIDIN
3 × 0.25
g.p.d. by
mouth for
29 days.

thrombin is a "definite calcium-phospholipoid-prothrombin complex". On the experimental side it has been shown by a number of workers that irradiated ergosterol can shorten the clotting time in rats.

As in certain cases of jaundice, so in thrombocytopenic purpura, the bleeding time is usually prolonged. It is stimulating, therefore, in this connexion to recall the two cases of thrombocytopenic purpura reported by Lowenburg and Ginsburg (1936) to have been cured by means of a parathormone hypercalcaemia. A single similar case has also been reported by Levine and Michelson (1940).

Possibly these observations may be related to the fact reported on at least two occasions that the symptoms of parathyroid tetany are relieved by massive hæmorrhage, following which the serum Ca increases (Cruickshank, 1923, Swingle and Wenner, 1925). It is not at present clear how these various points are related.

Vitamin C.—Ascorbic acid immediately controls the hæmorrhagic manifestations of scurvy which, it may be noted, have been related to certain pathological changes in and around the smaller blood-vessels originally described by Wolbach and Howe (1926). Thrombocytopenic purpura is also characterized by a tendency to bleed and in this condition there is an associated capillary abnormality—a low capillary resistance. It seems not entirely unreasonable, therefore, by analogy to suppose that ascorbic acid might benefit this condition also. Vaughan, in 1937, came to the conclusion that the chances of favourably influencing the course of thrombocytopenic purpura by means of ascorbic acid was about 33%. Excluding the 21 cases referred to by Vaughan, we have been able to find 10 reports in the literature, 6 being unfavourable and 4 favourable. Further investigation would, therefore, seem to be justified. I have recently had the opportunity of treating 7 cases of thrombocytopenic purpura with massive doses of ascorbic acid without any demonstrable effect on the bleeding time, the thrombocyte count, or the capillary resistance.

It is widely held that a low capillary resistance is found in scurvy and that this is controlled by ascorbic acid. Determinations of the capillary resistance have been, and indeed are, used as a test for so-called sub-clinical scurvy. There is, however, an increasing body of evidence to suggest that the practice is unjustifiable. I have never found ascorbic acid to be capable of increasing capillary resistance except in very special circumstances. It is true that a low capillary resistance is frequently found in scurvy but it does not, therefore, follow that the lowness of capillary resistance is necessarily due to ascorbic acid lack. Scurvy, as met with to-day, is a complex deficiency state. It is also true, on the other hand, that the capillary resistance in scurvy may be found to be high. This is due to the fact that following the extravascular suffusion of blood into the tissues the capillary resistance becomes temporarily markedly increased (Scarborough, 1941a). Thus, if the test be performed shortly after bleeding into the tissues has occurred, a high capillary resistance will be found; if the capillary resistance be determined either before, or at some time after a hæmorrhage has developed, then a low result may be found.

Vitamin P.—The presence of this vitamin, whose very existence is still regarded by many as resting on the most tenuous evidence, was first announced by Szent-Györgyi and his associates (1936) as the result of observations on certain cases of purpura which were not benefited by administration of ascorbic acid. Although my observations require further independent support, I personally regard it as established that there is a substance or substances present in fruits, their juices and in certain extracts made therefrom which is capable in man of increasing the resistance of capillary walls to the application of pressure (Scarborough, 1939). The substance is not ascorbic acid but, so far as I know, no satisfactory proof exists that it is a flavanone although statements have been made to this effect. Animal experiments have as yet produced no collateral evidence though there is reason to suppose that this may be forthcoming shortly. In two subjects, by means of experimental feeding, evidence has been obtained that a clinical syndrome may be produced as a result of a specific deficiency of vitamin P in man. The major feature of this syndrome is petechial bleeding and an exhaustive hæmatological investigation of both cases at the time bleeding occurred revealed a remarkably low capillary resistance and a slightly prolonged bleeding time (Scarborough, 1940). These findings also require independent confirmation. It is well known, and I have repeatedly observed, that certain of the bleeding diseases are associated with a low capillary resistance. If it could be shown, therefore, that the development of purpura were dependent in whole or in part on an excessive fragility of the capillary walls, then the administration of a substance capable of controlling this function, viz. vitamin P, might be expected to be of value in their treatment. It has been found difficult to obtain convincing evidence on these points, but the following two cases are suggestive:

CASE I.—A female, aged 76, had for some years noted a tendency to bruise very easily and had for two years prior to her admission to hospital, been troubled with intermittent epistaxis. During the last nine months petechiae had frequently appeared,

especially in the arms. There had been no previous or family history of hæmorrhagic disease and the dietary history was not significant. On examination the patient was a fairly well-nourished, healthy woman. Nothing abnormal was detected on examination of the cardiovascular system. The blood-pressure was 140/80. Renal function was satisfactory. Hæmatological examination revealed no cause for the hæmorrhagic tendency. The capillary resistance was found to be low.

A diagnosis of vascular purpura (purpura senilis) was made.

Fig. 1 (Case I) is a record of the capillary resistance of the patient determined in three separate areas of skin by a method I have already outlined (Scarborough, 1941b). The development of petechial bleeding is indicated at the bottom of the chart. The capillary resistance remained low during the preliminary period of twenty-six days, during which time a hæmorrhagic tendency was manifest. Administration of a preparation

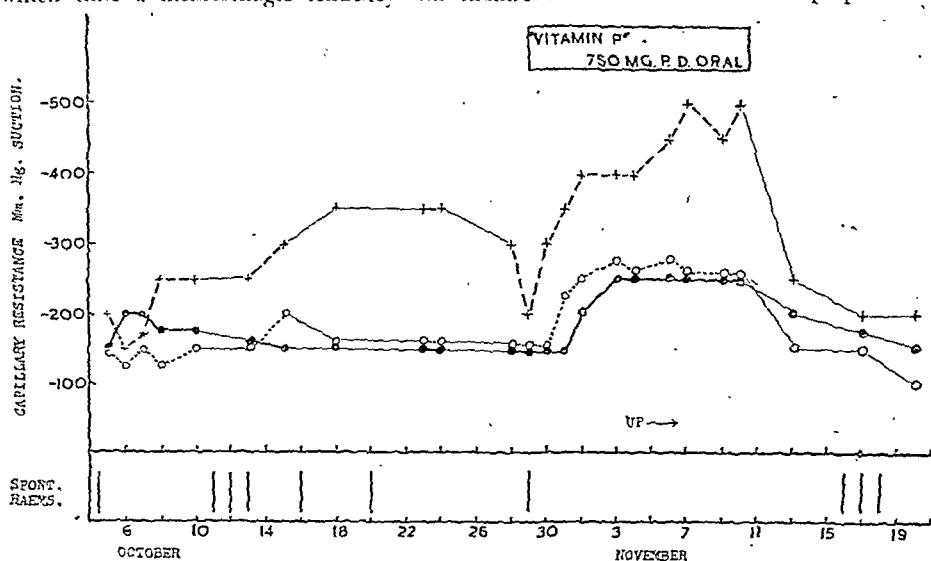


FIG. 1.—Chart of capillary resistance determined in terms of the least negative pressure (mm.Hg) required to produce a single petechia in each of three areas of skin. The development of spontaneous petechial bleeding is indicated below the chart by vertical lines. The effect of administration of a vitamin P preparation on capillary resistance and petechial bleeding is shown.

containing vitamin P coincided with an elevation of the capillary resistance and an absence of the manifest bleeding tendency in spite of the fact that the patient was allowed to get up during this period. With the cessation of vitamin P therapy the capillary resistance fell to its original level and the hæmorrhagic tendency returned.

CASE II.—A female, aged 65, for about ten years had complained of a marked tendency to bruising but of no other symptom. There were invariably one or more bruises on each arm and usually several on the legs. Ecchymoses could be readily induced by the "pinch" test. She had never complained of purpura but, on close examination on several occasions before the investigation began, petechiae were noted in both upper and lower limbs. There was no previous or family history of bleeding and the dietary history appeared to be satisfactory. Nothing abnormal was detected during examination of the cardiovascular system. The blood-pressure was 135/64. Renal function was not impaired. Hæmatological examination revealed no cause for the bleeding tendency. The capillary resistance was low. A diagnosis of vascular purpura (purpura senilis) was made.

| | TABLE I.—CAPILLARY RESISTANCE. MM.HG. | | | NEGATIVE PRESSURE. | | |
|----------|---------------------------------------|------|------|--------------------|---------|----------|
| | May | June | July | August | October | December |
| Area I | 150 | 400 | 300 | 400 | 150 | 150 |
| Area III | 150 | 400 | 300 | 350 | 125 | 150 |
| Area V | 200 | 450 | 350 | 400 | 200 | 225 |

Bruising

Bruising

PERMIDIN
3 × 0.25 g.p.d. by
mouth for 46 days.

PERMIDIN
3 × 0.25
g.p.d. by
mouth for
29 days.

Table I (Case II) summarizes the progress of the case. Capillary resistance was initially low and associated with a tendency to bruise. A similar condition was again present at the end of the investigation which covered a period of seven months. During the periods indicated, a vitamin P preparation (Permidin) was administered. This apparently had the effect of increasing the capillary resistance and of controlling completely the bleeding tendency.

These two cases are merely suggestive and even when taken together certainly do not constitute proof of the value of vitamin P in the treatment of purpura. The present position is, I think, that vitamin P does exist but that it cannot at present be regarded as a potent therapeutic agent in any one of the bleeding diseases; indeed, I would go so far as to say that preparations of vitamin P should not be used for this purpose outside experimental centres. The clinical worker must await the labours of the chemist for the isolation or identification of the materials in fruit juices which possess capillary-resistance-increasing activity. It is, after all, illogical to make statements about the ineffectiveness, or otherwise, of vitamin P in the treatment of various forms of purpura until: (a) We know what vitamin P is; (b) we have preparations of it suitable for administration; and (c) we test their effect on selected cases.

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VITAMIN K, AND THE ESTIMATION OF PROTHROMBIN

Dr. R. G. Macfarlane: The discovery of vitamin K, and the recognition and treatment of conditions in which it is deficient, mark one of the greatest advances in the field of the hæmorrhagic states.

Several naturally occurring derivatives of 1:4-naphthoquinone have vitamin K activity. Vitamin K₁ occurs in many plants and vegetables, particularly alfalfa, spinach and cabbage. A slightly less active analogue, vitamin K₂, occurs in putrefying fish meal presumably as a result of bacterial synthesis. The substance now known as vitamin K₃ was isolated in 1933 by Anderson and Newman from tubercle bacilli, though at that time they were unaware of its activity as a vitamin, and named it phthiocol.

Apart from these, some thirty synthetic compounds with similar activity have been reported. The majority are also derivatives of 1:4-naphthoquinone, of which the 2-methyl-derivative known as vitamin K₁ is the most familiar. The activity of the various synthetic derivatives is greatly influenced by apparently minor alterations in structure.

Vitamin K deficiency in man may be due to deficient diet, absorption, or utilization. Simple dietary deficiency is rare, although a few probable instances have been reported (Kark and Lozner, 1939; Scarborough, 1940). Vitamin K is found in large amounts in the faeces, where it is probably derived from bacterial synthesis, and this intrinsic source may well reduce the incidence of clinical manifestations that might otherwise result from deficient intake. Hæmorrhagic disease of the newborn has been shown by many workers to respond to treatment with vitamin K, and it has been suggested that there is normally a deficiency of the vitamin during the first few days of life, conditioned by deficient intake and the absence of bacteria from the intestinal tract (Quick and Grossman, 1939). Such

a deficiency accentuated by other factors is probably responsible for the disease. Deficient absorption of the vitamin is a more frequent abnormality and may be due to absence of the bile salts following obstructive jaundice, or to intestinal defects resulting in general malabsorption, such as sprue, steatorrhœa, gastro-intestinal fistulæ, and short-circuiting operations. Deficient utilization is said to follow liver damage and to be unresponsive to treatment. It has even been suggested that the response to vitamin K is an index of liver function as reliable as the excretion of hippuric acid (Wilson, 1939). Recent work, however, suggests that there is actually little correlation between liver damage, evidence of vitamin K deficiency and response to treatment (Lucia and Anggeler, 1941).

The result of extreme vitamin K deficiency in man and in animals is the development of a hæmorrhagic diathesis, which responds, except in some cases of liver damage, to adequate treatment with the vitamin. The type of bleeding that occurs is illustrated by hæmorrhagic jaundice, and hæmorrhagic disease of the newborn. There is a liability to bleed persistently from injuries, from the gastro-intestinal tract, or into the tissues. Coagulation of the blood is delayed, the bleeding time is sometimes prolonged in jaundice and almost invariably prolonged in hæmorrhagic disease of the newborn, the platelets and capillary resistance are apparently normal. It is now almost universally accepted that these manifestations are due to a reduction in the amount of plasma prothrombin, in turn dependent on the deficiency of vitamin K. While there can be no doubt that the coagulation of the blood is impaired in these conditions, it is difficult to explain all the manifestations on this basis alone. The prolonged bleeding time is an example. It has been repeatedly shown that the bleeding time is not influenced by coagulation of the blood and I maintain that it is an index of capillary contractility (Macfarlane, 1941). The same remarks apply to the "needle puncture hæmatomata" regarded by Kark and Souter (1941) as characteristic of hypoprothrombinæmia, and which do not occur in other conditions with defective coagulation, such as hæmophilia. Macpherson (1941) has shown that the prolonged bleeding time in hæmorrhagic disease of the newborn becomes normal following treatment with vitamin K, suggesting that a capillary defect may accompany the vitamin deficiency.

If the existence of a coagulation defect is established beyond doubt in these conditions, the nature of this defect is not quite so obvious, and it is necessary to examine the methods which claim to estimate prothrombin before the significance of their results can be assessed. These methods fall into two main groups, those using the single stage as introduced by Quick (1935) and modified by a large number of subsequent workers, and the two-stage methods. The latter as devised by Warner, Brinkhous and Smith (1934), and particularly the modification by Herbert (1940) avoid many of the fallacies of the single-stage group, but are so elaborate and introduce so many pitfalls of their own that they are not yet available for the ordinary laboratory practice.

Quick's test, or some modification, has been widely used in the detection of hypoprothrombinæmia, and the assay of human responses to vitamin therapy. The procedure depends upon the dogma that "in the presence of an excess of thromboplastin and a sufficient amount of calcium, the coagulation time of blood or plasma is proportional to the amount of prothrombin it contains". Details of the remaining part of the procedure do not seem to matter so much. The test can be done on whole blood (Ziffren *et al.*, 1939), oxalated blood (Innes and Davidson, 1941), oxalated plasma (Quick, 1935). If recalcification is carried out it can be done with a fixed amount of calcium (Quick, 1935) or a variable optimum amount (Pohle and Stewart, 1939). The term thromboplastin has included rabbit brain, fresh, heated, dried or extracted with acetone; human brain (Owen and Toohey, 1941); dog, horse or ox lung, Russell's viper venom (Fullerton, 1940) or Russell's viper venom and lecithin (Hobson and Wits, 1941). The normal coagulation time by these different methods varies from about 7-35 secs. and it would be proportional to varying prothrombin concentration only if certain assumptions are correct. Some of these are as follows, beginning with the fibrin end of the clotting process:

(1) It is assumed that a given amount of thrombin will necessarily clot different samples of blood or plasma in the same time. This may not be the case. Thrombin is rapidly destroyed in normal blood and variations in this antithrombic activity will alter the coagulation time. Moreover, quite large differences in the reactivity of fibrinogen to thrombin have been observed (personal observation, Herbert, 1941) and variations in fibrinogen concentration may be large and will alter the speed of the reaction.

(2) It is assumed that the rate of thrombin production and hence the coagulation time depends only upon the concentration of prothrombin in the presence of excess of thromboplastin. This is not the case. The rate of production of thrombin depends also on the reactivity of the prothrombin to the particular thromboplastin used, on calcium and electrolyte concentration, pH, and many other factors often not controlled. The time relations between activation and coagulation are also involved. If we accept Warner,

Table I (Case II) summarizes the progress of the case. Capillary resistance was initially low and associated with a tendency to bruise. A similar condition was again present at the end of the investigation which covered a period of seven months. During the periods indicated, a vitamin P preparation (Permudin) was administered. This apparently had the effect of increasing the capillary resistance and of controlling completely the bleeding tendency.

These two cases are merely suggestive and even when taken together certainly do not constitute proof of the value of vitamin P in the treatment of purpura. The present position is, I think, that vitamin P does exist but that it cannot at present be regarded as a potent therapeutic agent in any one of the bleeding diseases; indeed, I would go so far as to say that preparations of vitamin P should not be used for this purpose outside experimental centres. The clinical worker must await the labours of the chemist for the isolation or identification of the materials in fruit juices which possess capillary-resistance-increasing activity. It is, after all, illogical to make statements about the ineffectiveness, or otherwise, of vitamin P in the treatment of various forms of purpura until: (a) We know what vitamin P is; (b) we have preparations of it suitable for administration; and (c) we test their effect on selected cases.

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VITAMIN K, AND THE ESTIMATION OF PROTHROMBIN

Dr. R. G. Macfarlane: The discovery of vitamin K, and the recognition and treatment of conditions in which it is deficient, mark one of the greatest advances in the field of the hemorrhagic states.

Several naturally occurring derivatives of 1: 4-naphthoquinone have vitamin K activity. Vitamin K₁ occurs in many plants and vegetables, particularly alfalfa, spinach and cabbage. A slightly less active analogue, vitamin K₂, occurs in putrefying fish meal presumably as a result of bacterial synthesis. The substance now known as vitamin K₃ was isolated in 1933 by Anderson and Newman from tubercle bacilli, though at that time they were unaware of its activity as a vitamin, and named it phthiocol.

Apart from these, some thirty synthetic compounds with similar activity have been reported. The majority are also derivatives of 1: 4-naphthoquinone, of which the 2 methyl-derivative known as vitamin K₄ is the most familiar. The activity of the various synthetic derivatives is greatly influenced by apparently minor alterations in structure.

Vitamin K deficiency in man may be due to deficient diet, absorption, or utilization. Simple dietary deficiency is rare, although a few probable instances have been reported (Kark and Lozner, 1939; Scarborough, 1940). Vitamin K is found in large amounts in the faeces, where it is probably derived from bacterial synthesis, and this intrinsic source may well reduce the incidence of clinical manifestations that might otherwise result from deficient intake. Hemorrhagic disease of the newborn has been shown by many workers to respond to treatment with vitamin K, and it has been suggested that there is normally a deficiency of the vitamin during the first few days of life, conditioned by deficient intake and the absence of bacteria from the intestinal tract (Quick and Grossman, 1939). Such

hous, 1940). By the two-stage method, dogs and men have approximately identical prothrombin values, but by the single-stage method man has only one-fifth of the normal dog value (Quick *et al.*, 1935; Warner *et al.*, 1939). In newborn babies, the two-stage method indicates that prothrombin is reduced to 14-40% of normal, but the single-stage method gives normal values (Brinkhous *et al.*, 1937; Quick and Grossman, 1939).

The most striking anomaly has been provided by the use of the substances responsible for hæmorrhagic sweet clover disease in animals, considered to be due to a prothrombin deficiency (Roderick, 1931; Quick, 1937). Link and his associates have discovered that the disease is the result of a toxic agent identified as 3; 3'-methylenebis (4-hydroxycoumarin). The effect of this substance on dogs was studied by Bingham and his co-workers (1941), who found that it produced in addition to hæmorrhagic manifestations, liver destruction and changes in the capillaries. The protocols of their observations on the blood reveal the extraordinary fact that whereas the ordinary blood coagulation time was only a few minutes, the so-called prothrombin times on corresponding samples by Quick's method was many hours (Bingham *et al.*, 1941, *see* Table I, p. 568). In other words, the thromboplastin added inhibited coagulation. Not the least remarkable point is that the authors themselves make no comment on this anomaly and apparently accept the delayed clotting times as an estimate of prothrombin.

Similar results have been obtained by Witts (1942) in the case of a man given the coumarin compound. Again there was inhibition of coagulation by 'dried rabbit brain' since the "prothrombin time" was more than half an hour, as against four minutes if calcium alone were added. Fresh human brain slightly accelerates the clotting time, and if Russell's viper venom were added to the dried brain coagulation occurred in 13½ seconds. We were able to confirm these results.

It is quite clear that Quick's test has broken down completely where the coumarin compound is concerned. Any attempt to explain the results on the facts so far available would be pure speculation but I cannot resist the temptation. It may be that the inhibition by rabbit brain is an exaggeration of the species specificity, already mentioned. A more attractive but less probable explanation is that coumarin reduces the enzyme component of thromboplastin normally present in the plasma. While there remains enough to produce clotting in four minutes on the addition of calcium the addition of a great excess of lipoid in the form of dried brain results in inhibition, a phenomenon frequently seen in precipitin and agglutinin reactions if one of the reactants is in great excess. Fresh brain, because it contains some enzyme, does not inhibit the reaction so markedly. Venom and brain produce rapid coagulation, because the venom supplies the required enzyme.

I have tried to show that Quick's test as usually performed may measure prothrombin, but certainly measures many other variables as well. Russell's viper venom and lecithin seem to be a more reliable source of thromboplastin than the various animal tissues, variously treated, that are so widely used. If Quick's test is to be used at all, and it is a simple and in many cases a valuable procedure, it should no longer be called the "prothrombin time" but rather the "accelerated clotting test" as suggested by Professor Witts.

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Brinkhous and Smith's definition of a unit of thrombin as that amount which will clot 1 c.c. of standard fibrinogen solution in fifteen seconds, then normal plasma will produce about 300 units per c.c. Coagulation may occur within a few seconds of the production of a small fraction of the available thrombin, and further thrombin production goes on for several hours after coagulation is complete (Warner *et al.*, 1936). In other words, when Quick's test is all over, anything up to 95% of the prothrombin that was to be estimated remains unconverted. The practical importance of this relationship is that the test is sensitive to slight changes in reactivity of the prothrombin but relatively insensitive to changes in prothrombin concentration. This is illustrated by Quick's own curve (1938) which demonstrates little change in coagulation time between 100% and 40% prothrombin concentration.

(3) It is assumed that equal amounts of any one of the thromboplastic agents already mentioned will clot different samples of blood or plasma in the same time if the prothrombin concentrations are equal. This assumption leads us into very deep water, because so little is known about thromboplastin. The generally accepted view is that thromboplastin is a lipoid substance present in platelets and body tissues, and the older view that thromboplastin was an enzyme—the thrombokinasase of Morawitz—has been discounted (Mertz *et al.*, 1939). It has seemed to me for some time, however, that both views might be correct, and that what is known as thromboplastin is in fact two separate substances, one being a water-soluble enzyme, and the other a lipoid substance that is either catalyst or substrate.

In support of this view there is the analogy of the action of Russell's viper venom. The venom is a water-soluble enzyme-like substance, which behaves as an extremely powerful thrombokinasase. Trevan and I found that its action could be greatly accelerated by the addition of tissue suspensions, fat fractions, or by crude lecithin (1936). Moreover, we have been able to show that if plasma is defatted by spinning at 25,000 revolutions per minute or by extraction with solvents, Russell's viper venom is incapable of clotting it unless lecithin or tissue fat is added (Macfarlane, Trevan and Atwood, 1941). We have not been able to show as yet that substances analogous to the venom and lecithin are actually required for normal coagulation, but there is some indirect evidence in support of this view. For instance, saliva contains a thrombokinasase-like coagulant, and since the venom is a modified saliva, it is not unreasonable to suppose that the action of one may be merely a greatly exaggerated form of the other. Defatted saliva, like venom will not clot defatted plasma until the fat fraction is supplied. A major difference is in the effect of lecithin which will actively potentiate venom but has no effect on ordinary coagulation. Lecithin has, however, occasionally brought about the coagulation of defatted plasma, suggesting that it can activate the normal enzyme in the absence of the normal lipoid.

The provisional hypothesis, therefore, is as follows. Fresh tissues contain a venom-like enzyme, and a lipoid factor. The two factors together constitute what is known as thromboplastin and each is inactive separately. Plasma also contains both factors, the enzyme being in relative excess. Ordinary coagulation is the result of the reaction between these two factors and prothrombin in the presence of calcium. The addition of tissue extracts accelerates the normal reaction, because it increases the concentration of available lipoid. Russell's viper venom and lecithin represent the addition of both factors and result in the most rapid coagulation yet observed.

If this view of thromboplastin is correct, it will be seen that, in the estimation of prothrombin, no attempt to keep the enzyme factor constant is made, except in the methods using venom. Venom alone is liable to give variable results as it is greatly affected by small variations in available lipoid as shown by Crosbie and Scarborough (1941). The addition of a relatively large constant amount of lecithin as used by Hobson and Witts should control this variable.

Whether this view of the composition of thromboplastin is correct or not the procedures in use in the single-stage method are open to many objections. No account is taken of varying antithromboplastic activity either in the blood under test or in the thromboplastin itself. What is probably more important is that no allowance can be made for variations in the individual reactivity between prothrombin and thromboplastin, nor of the species specificity which has been shown by Trevan (unpublished) to be an important factor in the speed of the reaction, and which may vary from one sample of plasma to another.

There are many other theoretical objections to the single-stage method, but what is more significant is that there are serious discrepancies between the results obtained by the probably more reliable two-stage method and those obtained by the single-stage method. For instance, the incidence of hypoprothrombinæmia in jaundice as determined by the two-stage method is nearly twice that as determined by Quick's method (Brink-

Section of Odontology

President—HAROLD ROUND, M.D.S.Birm., L.D.S.Eng.

[January 26, 1942]

Experiences with Various Methods of Skeletal Fixation in Fractures of the Jaws

By RAINSFORD MOWLEM, F.R.C.S.

IN order to find fifty instances of the use of some method of fixation which was not solely related to the teeth, it was necessary to review the records of only 120 consecutive cases of fractures of the mandible or maxillæ, which had been treated in the Plastic and Jaw Centre at an E.M.S. Hospital. This, then, is one excuse for the incursion of a surgeon into what might be regarded as a dental field. Another is the fact that as a member of the Repair Team, the surgeon is often dependent upon immediate primary replacement of facial fractures before he can undertake the repair of soft tissues. He is therefore concerned to use methods which are immediately applicable and which do not impose limitations on his activities. A further excuse is that the method of fixation of mandibular fragments which are to be the site of a bone graft, may affect the technique of the operation.

The multiplicity of the fractures in any given case may have some bearing on the method of treatment of each part, and it may determine the order in which this treatment should be applied. The nearest intact bone may well be the vault of the skull; the plane of the teeth must be rigidly anchored to this and once this is done a basis for support of, say, the orbital floor or the malar zygomatic compound is obtained. In such circumstances it seems obvious that efficiency can be obtained only by a Team consisting of a surgeon, a dental surgeon, and a mechanic. To these must be added an anæsthetist, upon whose skill the possibility of early treatment is often dependent. Each man must have a working knowledge of the technical problems confronting the other members of the Team. Under such a regime it is unlikely that any part of the repair will fail to receive its due share of attention. Soft tissues will not be sutured without adequate primary bone fixation. Dental occlusion will not be obtained whilst orbital disorganization is overlooked. Bone control will not necessarily be thought of only in terms of teeth.

The cases illustrated have been chosen partly because they are unorthodox, but mainly because they show how combinations of simple methods may often solve difficult problems. It is not to be assumed that any illustration is regarded as the correct method of treatment for any given fracture. Some of the methods we may never use again, and others may well be inapplicable to a case which is apparently identical with that illustrated.

WIRE

Wire has been used in dentistry for many years and there can be very little that is new in its application. One of its great advantages is that it is readily available and by a little ingenuity can be made to solve awkward problems, particularly when the services of a mechanic and his laboratory are not available. There appear to be occasions upon which its very simplicity may determine its use even in the best equipped unit. At the beginning of the war, soft brass ligature wire, 22-24 gauge, was chiefly used. Later we were able to obtain annealed stainless steel of the same gauge, and this is now preferred because it has less tendency to stretch.

MANDIBULAR APPLICATIONS

Circumferential wires have been used for many years. For the edentulous jaw they are often the only method of treatment. There appears to be some diversity of opinion about the difficulties associated with their use. The first difficulty is that of insertion. This is simplified by the use of a fine curved cannula, the bore of which will just accommodate the wire. There has also been some difference of opinion as to whether it is safe to insert the wire from within the mouth. We have always used this route because it is easier to

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Mr. A. L. Bacharach said that recent work had confirmed the claims of Zacho and of Ruszynák and Benko to have produced a lowered capillary resistance in guinea-pigs receiving a scorbutogenic diet together with large daily doses of pure ascorbic acid. This capillary fragility was prevented or reversed by the administration of certain concentrates from citrus fruits and other sources, including the black-currant and rose-hips. Highly purified hesperidin, one of Szent-Györgyi's original sources of vitamin P activity, was also active, though less so than a more soluble product made from citrus peel by a modification of Szent-Györgyi's published procedure for preparing "citrics". The results so far obtained had been made the basis of a quantitative biological assay method, and it was hoped that this would help towards elucidating the distribution and chemical nature of vitamin P, and so indirectly its physiological role and therapeutic use.

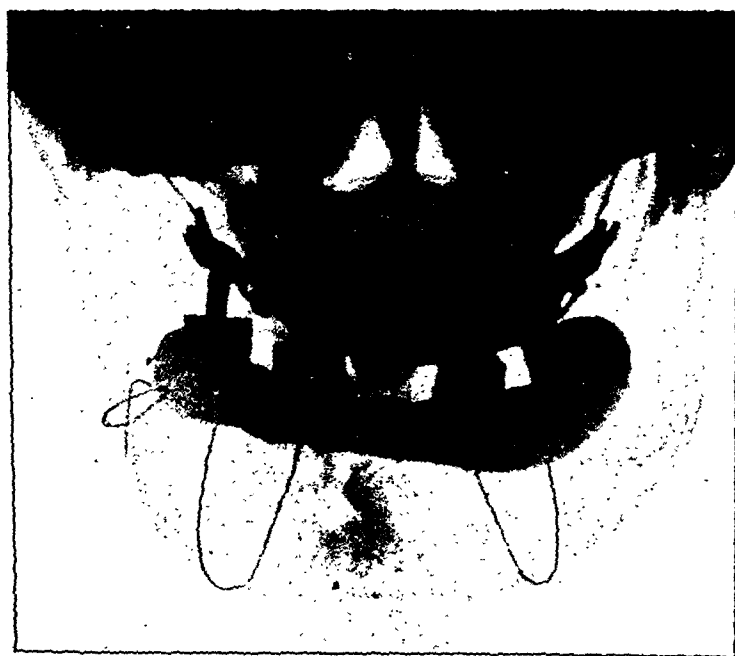
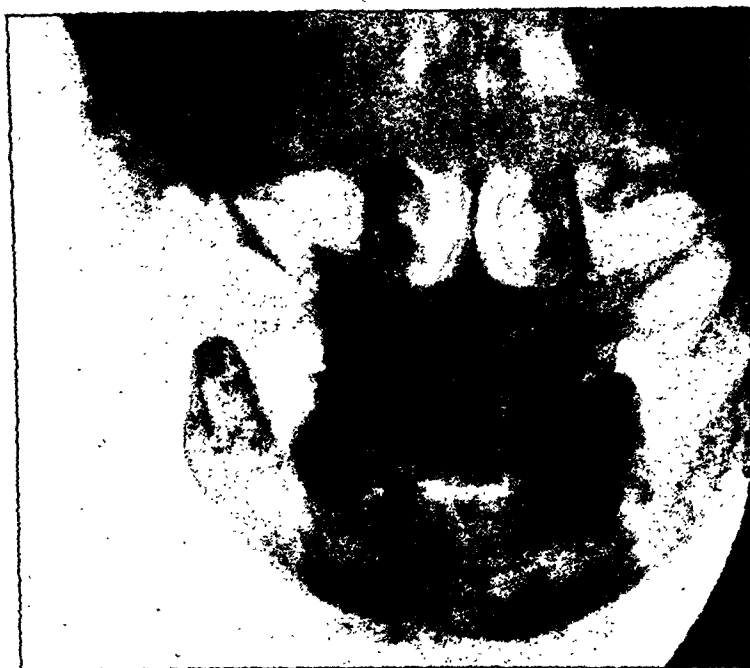


FIG. 1.—The loss of right ascending ramus. Mouth edentulous. Fixation obtained by double metal trough splint, the upper half being suspended to a skull-cap and the lower half wired on to the mandible.

keep close to the bone, and thus avoid the inclusion of soft tissues in the bite of the wire. At the lower margin of the mandible there is a small triangle of tissue which cannot be avoided. This is always severed by sawing the wire backwards and forwards three or four times before twisting it into position over the splint. This obviates two difficulties; first, pain due to the inclusion of this tissue, and second, slackening of the wire which occurs when the included tissue gradually gives way under pressure. With regard to the question of infection when the oral route is used, we have experienced only one case in which it was necessary to drain a small abscess, and even this was at the site of a previous infection secondary to osteomyelitis. The X-rays shown in fig. 1 illustrate the ordinary type of metal trough splint tied into position over the mandible. In this case it was used to fix the mandible prior to the bone graft shown in the lower illustration. A useful elaboration is shown in fig. 2. In this case there was a fracture in the body of the mandible together with a fracture of the right condyle, and both the condyle and the coronoid process on the left. The posterior circumferential wires carried additional wire loops which emerged through the skin. These were then fixed to vertical rods and distraction of the condylar fractures so obtained.

Angle wires.—These have been almost completely discarded because of their failure to immobilize a posterior fragment. With traction to a headcap, the ordinary angle wire does control flexion at the temporo-mandibular joint, but does not correct adduction without increasing rotation. In addition the fragment still moves with swallowing.

MAXILLARY APPLICATIONS

Cheek wires.—When the maxilla is completely floating and perhaps especially when there is an antero-posterior palatal fracture with differences in level of the two sides, a simple method of suspension by wire appears to work well. A plaster skull-cap with horns over the outer halves of the supra-orbital ridges is applied.

(It may be justifiable to diverge here to say a word about these caps. Our own preference is for the completely unpadded type from which the hair is protected by a single layer of stockinette. There are two essential parts to the cap. The first is the horizontal band which carries the appliance. This must be carefully moulded well down below the occipital protuberance, and must not press on the supra-orbital ridges. The second part consists of a vertical band from the preauricular area on one side across the vertex to the opposite side. This should carry the weight, and no pressure beyond that necessary to obtain close apposition to the occiput should be exerted by the horizontal band. The posterior quadrant of the cap is left open, both for the sake of lightness and to allow the hair to escape. This will be seen in figs. 3, 4, 5 and 6.)

Cap splints are constructed for the upper and lower jaws. On the maxillary splint an inverted hook is provided in the region of the first premolars on each side. The jaws are closed together in correct occlusion, and it is obvious that for this purpose the mandible must be used as a guide. Elevation of both the maxilla and the mandible is obtained by passing a wire from the horns of the plaster skull-cap to the hook on the cap splint through the soft tissues of the cheek. Spring loading can be applied if necessary and by this means correct reposition obtained. Fig. 4 shows a wire on the left side, with elastic loading to elevate the maxilla. The right half of the bone was controlled by a tube threaded over the wire. This will be mentioned later.

The lower X-ray in fig. 1 shows the attachment of such wires to the cap splint.

It might be thought possible to raise the maxilla too high by such a method, but in our experience this is not the case.

A modification for use in the absence of facilities for the construction of cap splints has been tried in an extremely extensive fracture of the maxilla. Diplopia and cerebro-spinal rhinorrhœa were present. Arch wires were fixed to upper and lower teeth and correct occlusion obtained. The cheek wire was then passed downward, not to the maxillary but to the mandibular arch. This was to avoid any tendency to laceration of the gums. Immediate complete control was obtained and there have been no difficulties in spite of the fact that the patient was nursed by reason of other injuries under an ordinary surgical regime. The presence of the wire in the cheek occasions the patient no discomfort and subsequent scarring is rarely visible, if care is taken to insert the wire in a direct line from the cap to the splint.

Maxillary alveolar wiring was introduced in this country by our colleagues, Messrs. Kelsey Fry, McLeod and Walker, at East Grinstead. Briefly, it consists of actually tying a splint on to the superior maxilla by two or three wires passed through the alveolus. Care must be taken to avoid the antrum. There are one or two technical problems concerned with the insertion of the wire at a level which makes it possible to keep the splint tight, but these are not insuperable. Fig. 2 shows a case in which the upper splint was tied into position and the wires effecting this can be seen in the X-ray views.

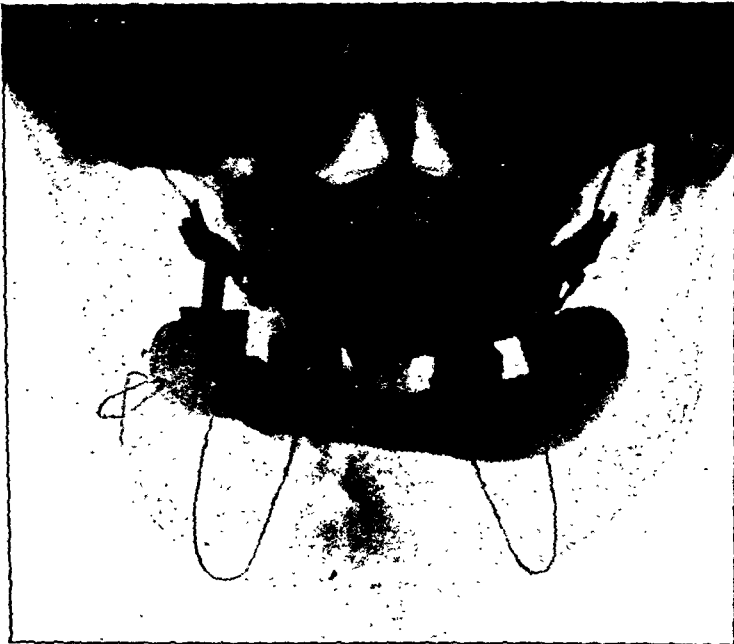


FIG. 1.—The loss of right ascending ramus. Mouth edentulous. Fixation obtained by double metal trough splint, the upper half being suspended to a skull-cap and the lower half wired on to the mandible.

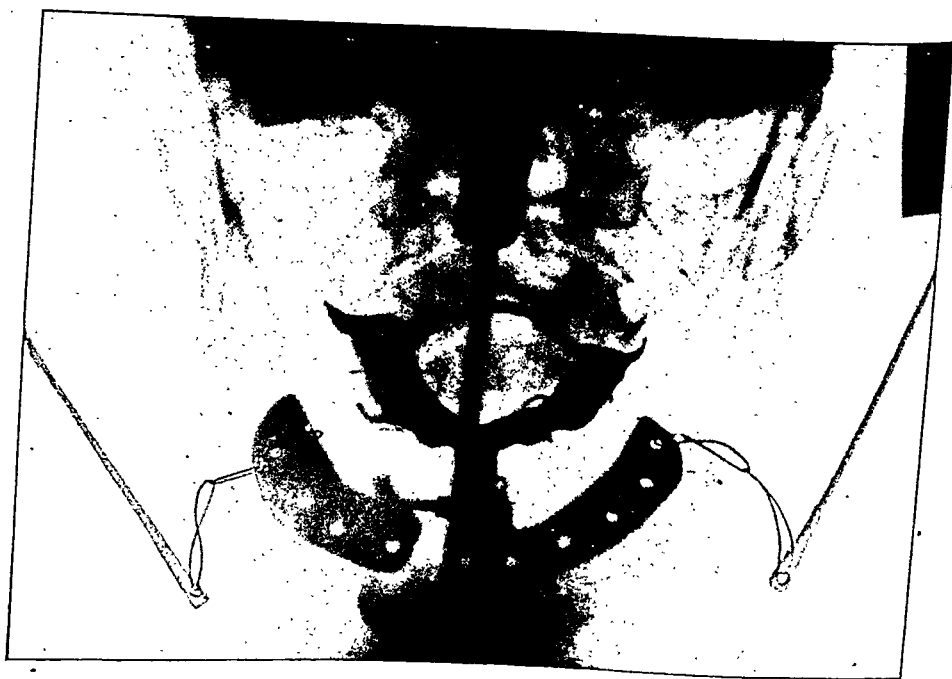


FIG. 2.—Fracture of right condyle, body of mandible, left condyle and left coronoid. Patient edentulous. Mandibular splint wired on, downward distraction at the angles, maxillary splint secured by alveolar wiring. The same patient is illustrated in fig. 3.



FIG. 3.—Views of pre-operative, operative and post-operative treatment of patient whose X-rays are shown in fig. 2.

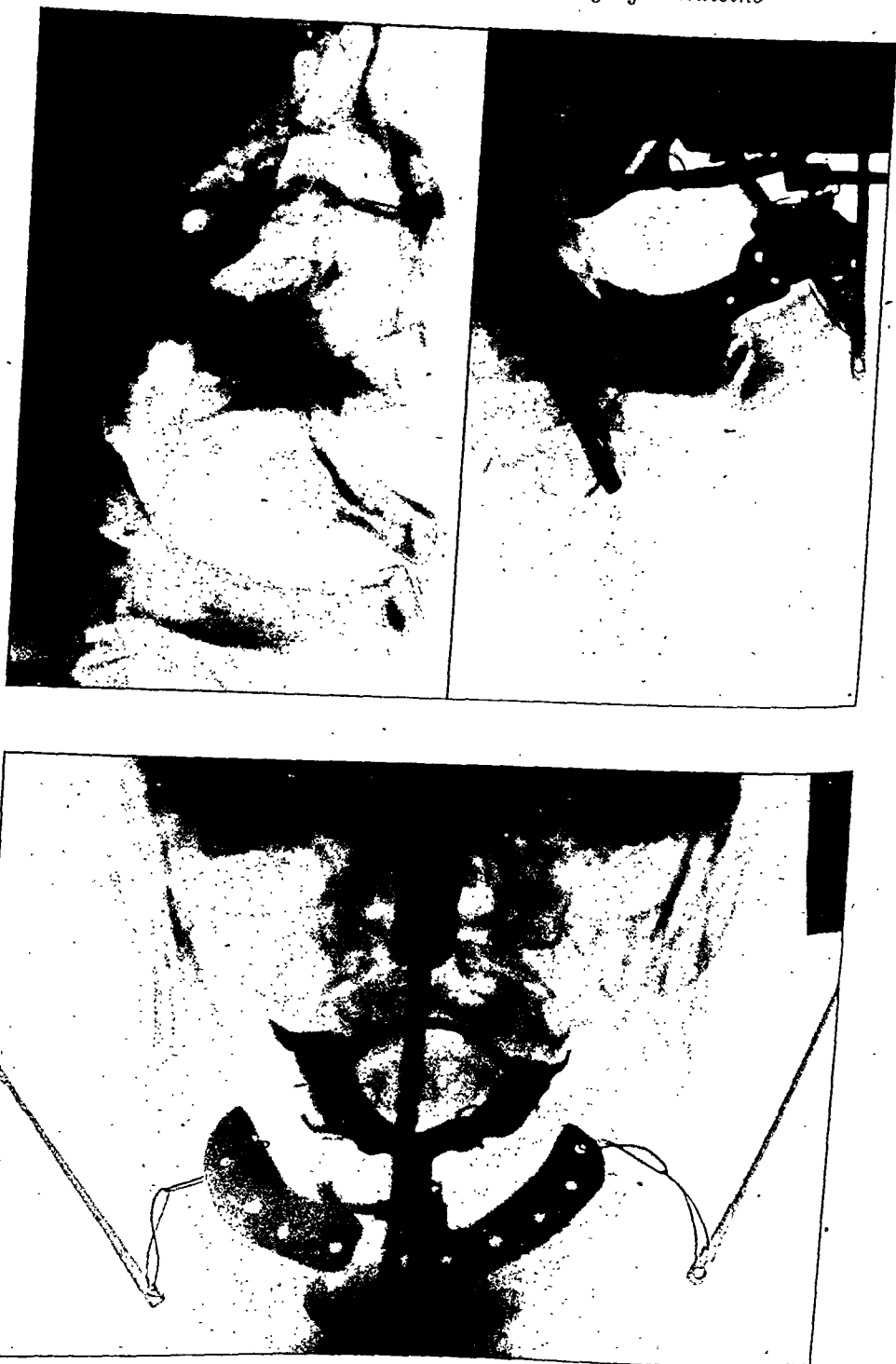


FIG. 2.—Fracture of right condyle, body of mandible, left condyle and left coronoid. Patient edentulous. Mandibular splint wired on, downward distraction at the angles, maxillary splint secured by alveolar wiring. The same patient is illustrated in fig. 3.

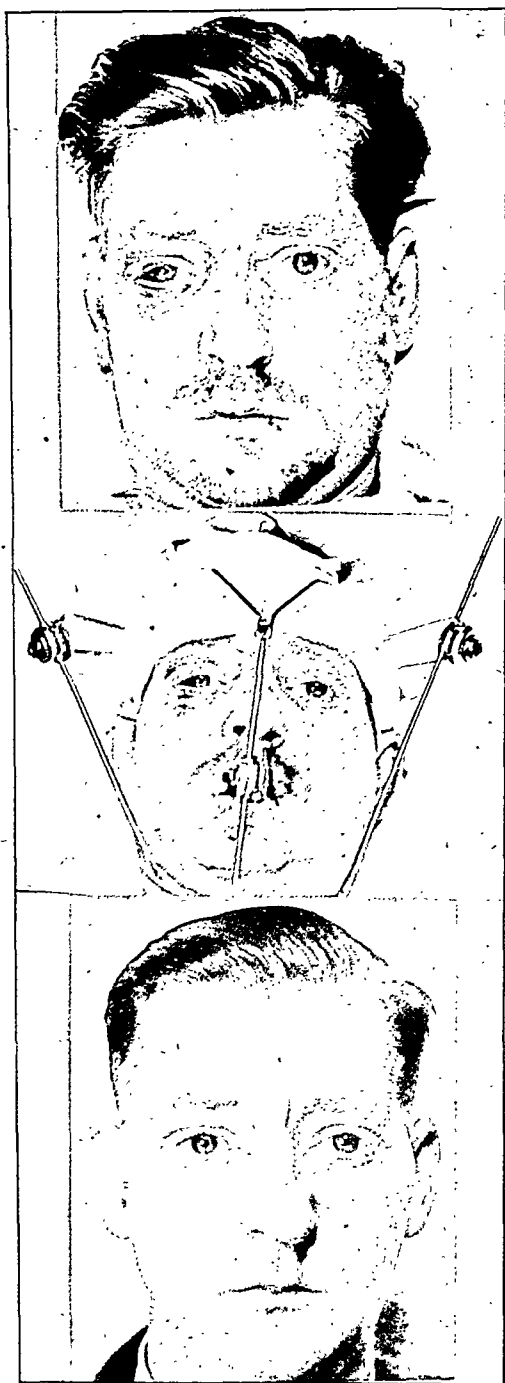


FIG. 3.—Views of pre-operative, operative and post-operative treatment of patient whose X-rays are shown in fig. 2.

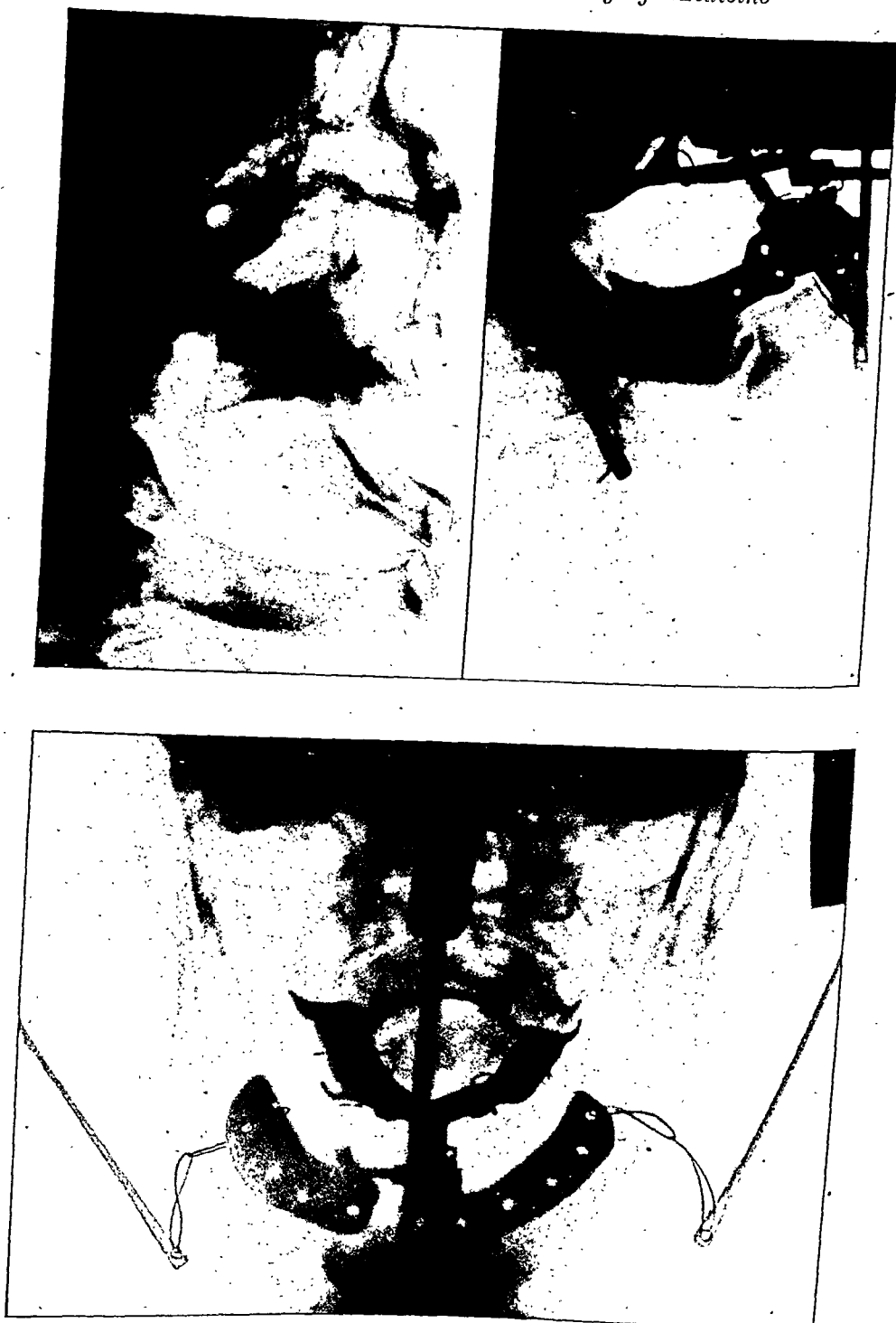


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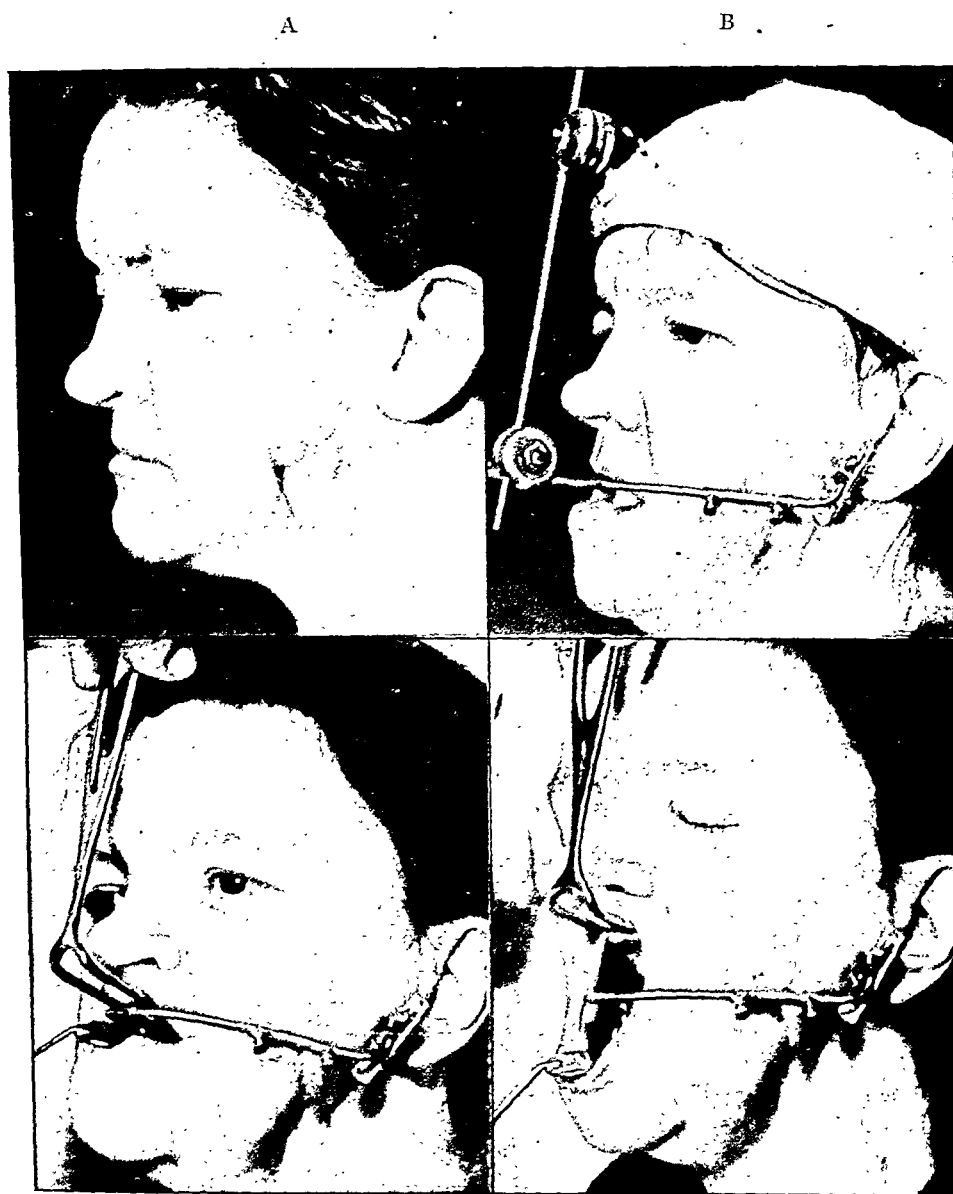


FIG. 5.—Bomb injury with bone loss. Marked flexion of posterior fragment shown at A. Pin fixation to sliding bar from lower trough splint, together with fixation to skull-cap shown at B. Degree of movement of mandible as a whole shown at C and D. X-rays of this patient shown in fig. 9.



FIG. 4.—Transverse fracture of superior maxilla with displacement backward and to the right, of three months' duration. Surgical mobilization followed by spring loaded traction on the left side, counter-pressure on the right side and forward traction by weight and pulley.

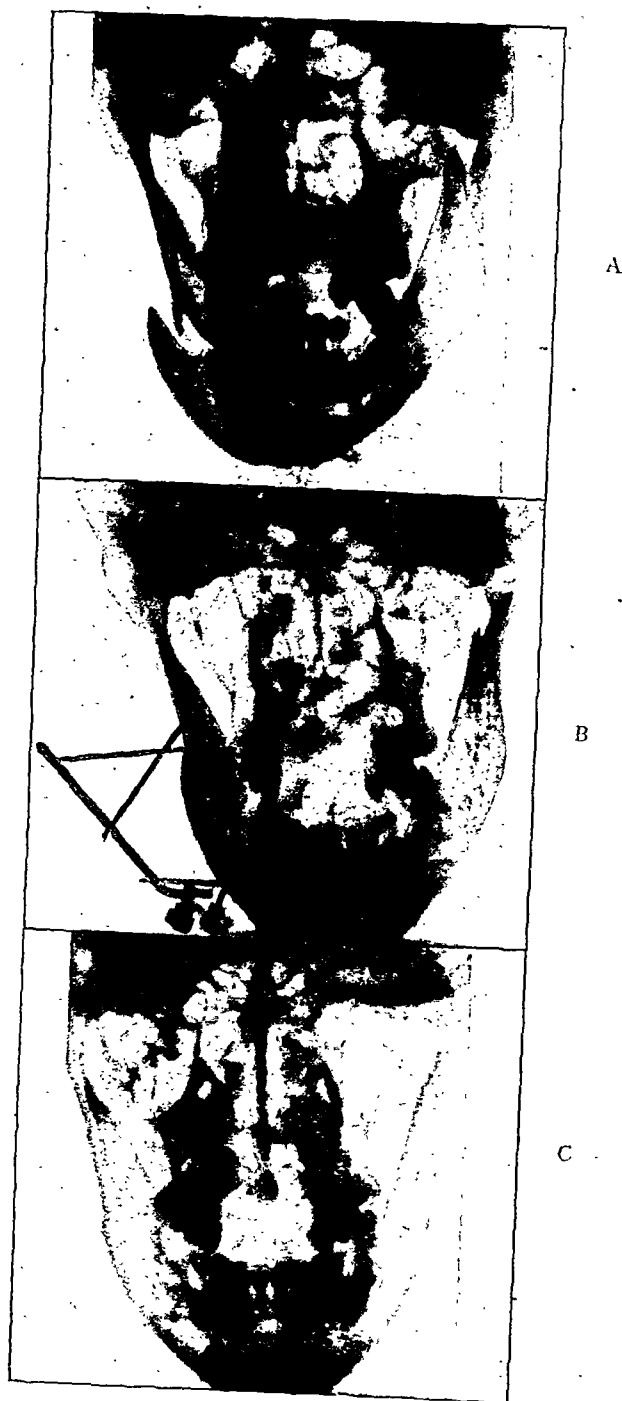


FIG. 8.—Fracture at the angle with medial displacement, rotation and flexion of the posterior fragment, shown at A, appliance in position shown at B and result at C.

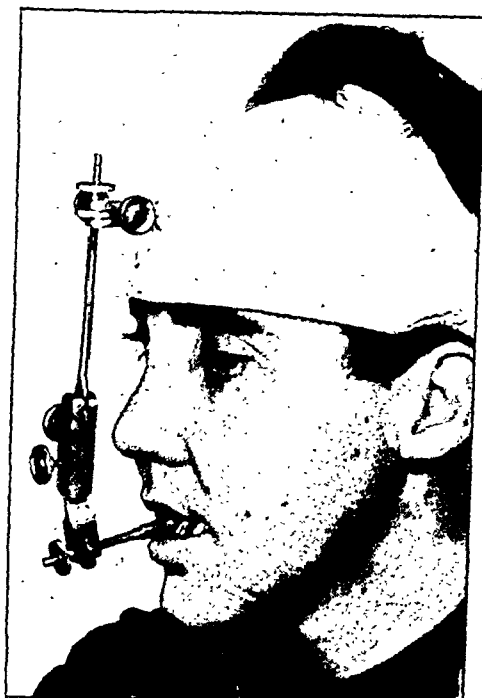


FIG. 6.—Apparatus devised by Squadron Leader A. B. MacGregor, R.A.F.V.R., giving threaded screw control for the maxilla.



FIG. 7.—Typical application of the cross-pin method of fixation for fractures at or near the angle.

ROD AND BAR FIXATION

This is a well-tried method, though some of its applications may be relatively new. We have abandoned the Kingsley type of splint because of its inaccuracy and instability. Its place has largely been taken by direct rod fixation from the plaster cap; the use of small universal joints has simplified this technique enormously and fig. 5 illustrates a typical vertical bar with two universal joints. One-eighth inch silver steel or German silver rod is most commonly employed.

The horizontal part of the appliance in fig. 5 is described later.

MANDIBULAR APPLICATIONS

Control of the edentulous posterior fragment.—Though many of the methods previously used have now been superseded, it may be interesting to review some of them. It has already been stated that we have been dissatisfied with angle traction wires. In an endeavour to obtain more rigid control, we first applied a U-shaped loop of flattened German silver to the angle by means of two wires; one limb of the loop was therefore rigidly attached to the bone, while the other, emerging through the skin, pointed forward. This was attached by a sliding tube to a rod somewhat like a Kingsley bar from the mandibular cap splint. The method was reasonably satisfactory except for the length of rod involved. Later, therefore, we carried the bar through the soft tissues of the cheek directly from the splint to the angle. This meant that we were dependent upon the use of a malleable bar for accurate repositioning, and though on the model this is easy to manipulate, in practice it is prone to be inaccurate.

MAXILLARY APPLICATIONS

A simple type of fixation, as illustrated in fig. 5, is frequently used. It may, however, be found that one side of the maxilla is raised too high in relation to the other. Fig. 4 illustrates a method of exerting downward pressure by means of a tube threaded over a cheek wire. The left side is spring loaded to raise it. In this particular instance, retro-position of the maxilla as a whole was corrected by traction with a 4 lb. weight over the foot of the bed. Naturally, such extensive procedures are necessary only when the initial treatment has been inadequate. In this case the maxillary fracture had been ignored for three and a half months and the models show an open and an oblique bite.

A modification of the rigid bar has been introduced by Squadron Leader MacGregor (fig. 6). This gives threaded screw control in all planes. The extremely heavy pressures necessary to replace an old fracture may not be tolerated by this appliance, though the difficulty could be overcome by adding cheek wires to obtain lift at the point of maximum resistance, i.e. in the pterygoid region. For recent fractures it gives a precision of control which is unique.

None of these methods is new in principle, though some of the modifications are relatively recent. They have been brought forward only to point out that combinations of the various standard appliances will cover most eventualities.

PINS

There is, however, one principle which has not previously been applied to the mandible. This is the crossed pin method of external fixation. In previous publications it has been pointed out that this principle does not supersede any of the accepted and well-tried methods. It is, however, most applicable to that fracture which has hitherto been least easily controllable, namely, the fracture at or near the angle with a short posterior edentulous fragment. The actual method of fixation varies in different Units. One type is illustrated in fig. 7. This dispenses with any type of universal joint, whilst still providing universality of movement. The technique of application is simplified by having an assistant. Primary reduction is effected by him from within the mouth. The surgeon marks out on the skin the position of the lower margin of the mandible and the fracture line; he then inserts a pair of pins into each fragment, crossing them as shown. Care is taken to keep close to the lower margins of the bone to avoid tooth roots and the inferior dental canal, and not to penetrate completely through the bone. He then couples each pair of pins to its plate, and now has control of the bone fragments from outside the mouth. He maintains the reduction whilst his assistant checks the bite, and then tightens the screws controlling the intervening fixation bar. Minor corrections can easily be made without subsequent anaesthesia. Fig. 8 shows the three phases in the treatment of a typical fracture.



FIG. 9.—X-ray of patient illustrated in fig. 5. Lower metal trough splint wired on to mandible, bar emerging from mouth to connect with pins in the posterior fragment. Bone graft is shown in position. The appliance shown in view A corresponds with 5B, view B with 5D.

Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

[January 23, 1942]

Man-Power—Medical Aspects in a World Army To-day

[Abridged]

By Colonel D. GORDON CHEYNE, O.B.E., M.C., M.D., D.P.H., D.T.M.&H.,
late R.A.M.C.

IN considering the subject of man-power to-day we must reflect on the social conditions in this country during the past twenty-five years, the period towards the end of the Great War and immediately following. The post-war conditions of unemployment, poor housing and limited recreational facilities affected the youth of the country both physically and mentally.

To improve the men physically and alter the outlook of the population as a whole—to make them war-minded instead of pacifist—a tremendous drive was necessary. Amenities in barracks were much improved; in addition to good food and accommodation, mental relaxation was supplied by the provision of lectures, recreations, reading and concerts. The physical standard of recruits was carefully considered and disabilities were more closely assessed so that the flat-footed man did not find himself drafted to an infantry regiment or the skilled draughtsman to a Pioneer Corps.

Shortage of recruits always has had its effects on physical standards. Height and weight may be lowered and more men below recruiting standards are then accepted as special enlistments approved by hygiene officers. So short were we of recruits in 1938 that battalions at home were only at half-strength. The situation was so critical that we were forced to lower our standards. We dispensed with dental standards and provided dentures, we lowered the visual standards for motor transport and Line of Communication Units and took men whose natural vision was insufficient but who could be brought up to standard by glasses which we provided. We accepted men with severe foot defects and employed them in the mechanized M.T. and L. of C. Units.

We took undeveloped and undernourished recruits and sent them to special physical development centres where 80% of them were brought up to recruiting standard. In fact recruiting was the most important part of the work of the hygiene officers in areas. As a result these officers adopted a standard of their own. They knew the common disabilities of that section of the population and assessed their importance in relation to military service in each individual—whether in spite of his disability a man would stand up to military service anywhere in the world, or whether such service might aggravate it. The result of all this was that though the Army ready to take the field was small it had been carefully selected and contained few misfits.

With the coming of war recruiting was entirely taken over by the civil authorities. There were errors of course, due probably in some cases to a lack of proper assessment of the importance of a disability and also to the desire to admit only the cream of the population. Alas, there is not and never was enough cream to go round. When it was decided to categorize the whole Army this lack of appreciation of the importance of disabilities again became evident, and young and inexperienced medical officers were getting busy again actively to dissipate the Army.

The influence of the C.O. is naturally very great and an enthusiastic C.O. anxious for the very best material often finds or tries to explore the medical route for the discharge of what to him are undesirable. This brings me to the matter of selection of material—a subject which assumed an importance much greater than ever after Dunkirk. We were thrown almost entirely on our own resources so that practically all labour for our industrial concerns—notably munitions, agriculture, &c.—had to be found within these islands, while, at the same time, an army had to be raised for civil defence, including fire services—conditions which had never before existed on a similar scale. The standards in the Army inevitably suffered and while certain of its sections had to have first-class material, such as R.A.C. and R.A., material of lower category, mental and physical, had to be absorbed into other Corps. It has been and is very difficult to make C.O.s appreciate this. Naturally everybody wants the best. Remember too that the

The advantages of this method may be summarized as follows:

- (a) Absolute control and fixation without discomfort.
- (b) Retention of mobility of the temporo-mandibular joint with the maintenance of function and the relative absence of feeding problems.
- (c) Complete absence of all intra-oral fixation with consequent cleanliness of the mouth.

In our experience with these pins, we have come across a few difficulties. In one case we were unable to get retention of the pins in the posterior fragment. This was probably due to two factors: the patient was an emaciated elderly man, who had been edentulous for many years, and there was marked atrophy of the bone. The other factor—probably less important—was the leverage exerted by the weight of the appliance. This has since been reduced from approximately $1\frac{1}{2}$ oz. to less than $\frac{1}{2}$ oz.

A second difficulty was experienced in an airman who had suffered from an ununited fracture at the angle for many months. The bone ends were freed and freshened and the pins applied. On the third day he complained of increasing and intractable pain in the region of the fracture. No obvious cause could be found. The X-ray appearances did not suggest that the pins in the posterior fragment were near the inferior dental canal. It was thought, however, this must be the case, and that either œdema or a small hæmorrhage was exerting pressure on the inferior dental nerve. The posterior pins were therefore removed, and reinserted nearer to the posterior margin of the ascending ramus with immediate relief. The appliance was subsequently left in position for three and a half weeks without discomfort.

A third difficulty was encountered, though this was when we were endeavouring to assess the potentialities of the method. There was a bilateral fracture at the angle with gross downward displacement of the central fragment. We inserted three sets of pins, one in each fragment. This meant that the weight of the horizontal ramus, together with the stresses of the infra-mandibular muscles and the tongue, were being borne by the torsional resistance of the pins in the posterior fragments. Naturally enough, we did not obtain absolute control, and it was necessary to provide anterior splint support in addition. Thereafter the fractures were treated separately by the same method, with a good result.

So much for difficulties. Other questions arise. Can the method be applied in the presence of infection in the fracture line? We have used it on at least six such occasions with good results, and we have also used it to bridge compound soft tissue injury, without any disturbance. Is the method painful? Does it leave appreciable scars? To these the answer is no. We feel, therefore, that here we have an additional appliance which offers an excellent chance of success in those fractures which have previously been least satisfactory. Pins can be applied either alone or in combination with other methods. Fig. 9 shows pins in position in the posterior fragment; this patient had lost considerable bone in the region of the angle of the mandible following upon an infected compound comminuted fracture due to a bomb injury. Flexion of the short posterior fragment was so marked that function of the temporo-mandibular joint was seriously reduced. This is well shown in the photographs of the patient in fig. 5. The posterior fragment was mobilized and held in position by distraction via a rod to a Gunning splint wired on to the mandible. At first the mandible was fixed to a plaster cap by a central bar; this was later removed and the mandible and its graft allowed to move as one piece. I can think of no other method which would give such efficient control, and it will be noticed that this again is an example of the frequency with which more than one basic method is employed on the same patient.

In giving this brief résumé of various combinations of new and old methods, I have drawn freely upon cases treated by other members of the Unit. To my dental colleagues, Mr. Dudley Buxton, Mr. B. W. Fickling and Squadron Leader A. B. MacGregor, R.A.F.V.R., and to my surgical associate, Mr. Barron, I would like to express my sincere thanks, not only for their courtesy in allowing me to quote their cases, but also for their part in the discussion and planning which has gone towards the obtaining of good results in difficult cases.

Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

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call-up age for the Army is from 19 to 45. The R.A.F. takes boys of 17½. This service is much more popular than the Army so we lose the best of the youngsters.

It is more difficult to assess mental than physical ability and the psychologists must try to estimate what a man will be like in three or four months' time. Dull and backward men are undoubtedly a problem, but the rejections are greatly reduced in number of late owing largely to a better understanding on the part of psychiatrists of how such men can be utilized in the Army. The great majority are transferred to Pioneer Units and do well.

Specialists in all branches of medicine and surgery should know something of the duties and work of the soldier otherwise they cannot advise properly upon disabilities *vis-à-vis* such duties. It is interesting to note that in the German Army the psychologists live in much closer contact with the fighting troops, so that they are better able to assess a soldier's mental outlook in that particular arm of the service. Also in Germany each man has a dossier which gives his whole life-history (including medical) from birth; this is of great value to the psychiatrist in summing up the recruit's character and health.

Some years ago I was instructed to observe the effects of prolonged tank life upon physical efficiency. The object of training is to produce physical and mental fitness so that work is carried out at the most economical cost and with a reduction in the time "lag". This is the time between the reception of an order or a stimulus and the correct reaction thereto. The scope of training is not wide even in the R.A.C. and the movements and co-ordination required are not very complicated. A situation is presented which has to be appreciated and then the execution of the appropriate action has to follow. Speed is the all-important factor and training ensures this. I was told then that if officers, trained N.C.O.s, and men, all of whom had completed their training, were pitted against each other in snap-shooting the N.C.O.s would win. If this was so, it excludes education as a dominant factor—education, as we normally accept the term—and puts training first in importance. The best that we can say is that our various tests are an aid in assessing the "trainability" of the men. There are two factors which govern selection of men: (a) The increase in mechanized technical units requiring a higher standard of intelligence, and (b) Speed of training—two months instead of six. There is no time to train the slow and backward.

When the recruit is accepted for the Army his accommodation is good—not up to peacetime standards, but the reduction in floor space from 60 to 45 sq. ft. per man and in cubic from 600 to about 400 has had no appreciable adverse effects. In the defended localities in this country—coast defence, gun positions, A.A. sites and the like—it has not always been possible to obtain these standards, but on the whole we can view the accommodation of troops with equanimity. Even tentage in winter, provided double tents are issued with linings where possible, impervious floors, fireplaces and plenty of blankets, is much less spartan than it sounds, and the men's health in such tentage in my own experience in the first winter of the war was markedly better than that of men in adjacent barracks. In the Army we endeavour always to ensure separation of heads, and double bunking of barracks has been of great value. I am unable to give many figures of sickness but in my Command the total admissions to military hospitals for tonsillitis, bronchitis, pneumonia, and influenza in the first six months of 1941 amounted only to about 8 per 1,000 of the population. This figure excludes epidemic catarrh which in the mild form experienced was treated at reception stations.

Feeding of the Army continues satisfactorily with the advice of nutritional experts. Deficiency diseases are practically non-existent in the Army in any part of the world and the issue of vitamin preparations has been made only in very few instances. Recently tests to ascertain the degree of vitamin C saturation in groups of soldiers living under various conditions have been satisfactory. Improvements in feeding and messing have resulted from the appointment of catering advisers and the establishment of an Army Catering Corps, but it is constantly stressed that the Medical Officer should be the scientific adviser in his own unit. Schools of cookery are established and the students include Commanding Officers who not only prepare food but have to eat it. These schools are concerned with cookery all over the world from Salisbury Plain to the Desert and a lecture on the scientific aspect of messing and feeding by a hygiene specialist is included in each course. Special compact rations for special purposes where lightness and high nutritive value are of supreme importance have also been evolved.

An important part of the organization is the exclusion from kitchen staff of carriers of diseases, notably dysentery and the enteric group, and the strictest precautions are being taken with prisoners of war who may come to this country similarly to exclude intestinal carriers and other disease carriers. This should constitute an effective barrier against infection of the civil population. I may say the incidence of dysentery and enteric fever has been exceptionally low even during periods when there was a considerable incidence amongst the civil population. Inoculation remains a sheet anchor in the Army.

The training of the soldier is supervised by medical officers whose duty it is to impress upon Commanding and Training Officers the necessity of setting a pace for the average and not the super-enthusiast, of watching for signs of breakdown, and of attention to weights, avoiding strain and overloading the man on marching. I consider that the old standard of being able to march 20 to 25 miles in full kit day after day for a week must go, good as the standard was, but on many occasions the soldier will still be called upon to march to a destination and fight. The medical aspect of correct loading of the soldier, carriage of equipment as well as weight and distribution on the man, are constantly being watched and the importance to hygiene of new equipment as well as new developments in military tactics is ever in the minds of the various departments concerned.

I consider every medical officer should have a spell of regimental duty before going on to a hygiene appointment, for the application of hygiene knowledge differs immensely in civil and military practice. Every Field Hygiene Section mobilized on a divisional basis now runs a school which is an Army School of Hygiene in miniature. Instruction is given by the O.C. and his N.C.O.s; many of the latter are ex-civil sanitary inspectors. These schools are attended by the sanitary and water duty men of units. Special courses are held for medical and other officers and lately we have included A.T.S. officers and auxiliaries, and they are essentially intended for Field Service. We are also teaching elementary tropical sanitation, and lectures are given at other schools of instruction and O.C.T. Units, in the hope that if we can instruct these officers as early as possible greater benefits may result.

Propaganda films on such subjects as water purification, malaria prevention and the fly, louse, &c., have been prepared in addition to the issue of certain Army propaganda posters.

Certain organizations have been built up to restore to the Army man-power which has been temporarily lost to it, such as Convalescent Depots, Medical Examination Centres, and Physical Development Centres. Red Cross Convalescent Hospitals, though not under Army administration should be included. I would like to pay a tribute to the Emergency Medical Service. I look on hospitals as a most important element in the conservation of man-power, for on the skill of the staff depends the man's rapid return to duty and his saving to the Army. Our relations with the regional authorities of the Ministry of Health, including Medical Officers of Health are close and cordial and we owe them much.

The Red Cross Convalescent Hospitals receive our patients and perform the first part of their rehabilitation. From there or directly from a hospital, civil or military, all patients—with a few exceptions—proceed to a military depot where they are trained by stages to return to full duty with their units. The staff includes P.T. experts, masseurs, &c., and the command specialist in physical medicine, as well as the consultants in this branch, watch over the whole of the activities. The training programme is carefully graduated and work of first-rate importance is achieved.

The Medical Examination Centre is a new feature and it is as yet too early to assess its value. It is intended that men who are considered unfit for any form of military service, either while in their units or after being in hospital, should be sent there for medical boarding. The hospital cases are men fit to walk, able to look after themselves, and on ordinary diet. The real hope is that careful examination and medical boarding by experts may result in a saving of personnel by placing them in suitable jobs in the Army. As regards such men in units, careful boarding by the Civil Panel should exclude them to a large extent at the outset, and discharges from the Service should be rare. Expert knowledge of their possible utilization in the Army in some capacity is essential on the part of the examining board. The bed situation in hospitals is also helped by such a centre. It has been separated from Convalescent Depots, as it seemed undesirable to introduce an element where discharge from the Army was dominant into a unit where the desire for fitness to carry on should be the all-important outlook.

The Physical Development Centre is a revival of the centres which existed at Canterbury and Scarborough before the war, and which were so conspicuously successful. They received the "underweights" from depressed areas and men with various defects, mainly foot disabilities, spinal defects and other minor curable afflictions. Similar work is being done at this new centre with considerable success, especially in the case of undernourished and underdeveloped individuals. Flat-feet cases are responding to treatment. Much of the success of the centre must depend on the selection of the material sent to it—70% of the cases have their category raised. The ideal of course, is some centre where all the intakes would come and where they would be examined, questioned, analysed, scrutinized, categorized, and where every man would be put into that particular niche which fitted him. This is perhaps Utopian, but even with the present system, a man should ultimately find that niche and stay there, provided always, C.O.s would realize that the stream of man-power is not a mighty torrent and that the quality is intimately bound up with the many calls upon the man-power of the country.

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Section of Urology

President—G. E. NELIGAN, M.C., F.R.C.S.

[February 26, 1942]

Experiences with Phenothiazine as a Urinary Antiseptic

By WILLIAM H. GRAHAM, F.R.C.S.

(*In absentia* read by T. J. MILLIN, F.R.C.S.)

INTRODUCTION

PHENOTHIAZINE is the parent substance of the thiazine dyes, and has a chemical formula closely related to methylene blue. Oral administration results in the urinary excretion of phenothiazine and its two oxidation products, thionol and leucothionol, part of the leucothionol being in some loose chemical combination. Leucothionol is spontaneously oxidized to thionol, which imparts the characteristic red colour to the urine, and gives it its bactericidal properties. Thionol-leucothionol is a reversible oxidation-reduction system similar to methylene blue and its leuco base.

Thomas, DeEds and Eddy, of San Francisco, showed the bactericidal properties of urine containing thionol, and because of the low toxicity after oral administration of phenothiazine, as shown by growth curves and blood picture changes, DeEds, Stockton and Thomas (1939) considered it justifiable to undertake clinical trials with it. They carried out this trial on two groups of patients. The first group of 61 patients was given phenothiazine alone. The second group of 31 patients was given phenothiazine in combination with ammonium chloride. The acid reaction of the urine, especially in the pH range of 4.5–5.5, facilitated the liberation of the leucothionol held in combination, and this oxidized to thionol increased the active bactericidal properties of the urine.

In the first group 33 had chronic urinary tract infection complicated by obstructive lesions. Many had previously received prolonged courses of treatment, both local and general. Sixteen had acute urinary tract infection. The types of bacterial infections were varied, covering *B. coli*, streptococci and staphylococci and the typhoid dysentery group. This could be taken as a good test of the efficacy of phenothiazine as a urinary antiseptic. No patient was considered as cured who had not been observed for a minimum period of four weeks following the cessation of administration of the drug. Others with temporary freedom from symptoms, or apparently cured and observed for less than four weeks were classed as "improved".

10 of the 16 acute infections were cured, 5 of the 35 chronic infections were cured and 20 more improved. Only eight of the 49 patients in this group using phenothiazine alone failed to secure relief from their symptoms. The average daily dose of phenothiazine was 1.33 g. during an average period of 7.4 days. The average total dose was 9.99 g.

In the second group of 31 patients, ammonium chloride was administered with the phenothiazine. A daily dose of 1.9 g. of ammonium chloride was found sufficient to maintain the optimum pH value of the urine, namely between 4.5 and 5.5. The increased thionol content was shown by the intensity of the red colour. 20 of the patients had acute urinary tract infection, a higher percentage than that in the first group. The authors summarized their findings, stating that 25 out of the 31 were symptomatically cured in an average time of three days. The dose of phenothiazine was approximately one-half that used in the first group, who received phenothiazine without ammonium chloride.

During this investigation of 61 patients, no undesirable effects or complications arose which could be attributed to the drug. There were no signs of gastro-intestinal upset nor of renal irritation. The direct Van den Bergh reaction was never elevated, and any rise in the indirect Van den Bergh and icteric index could be attributed to the colour of the dye. Liver function tests showed no change. Blood-counts were done in 19 of the patients. Three patients developed a secondary anaemia after receiving an average total dose of 23.5 g. In one patient this anaemia was marked after nearing 19.9 g. The red

The response to inoculation against typhoid, and paratyphoid A and B, in this country is satisfactory, and its value is proved. The same may be said of vaccination against smallpox, but here we have the complicating factor of large numbers of the entrants unprotected by previous vaccination in infancy, or at any other time. Their reactions are in many cases severe, with consequent interruption in training. Here again the response is good, and this is of great importance considering the fields of action in which the Army may find itself. When troops go to yellow fever countries, they have the advantage of protection given in this country against the disease. Special malaria units have gone to the East. Special arrangements for disinfection will accompany units where there is a risk of typhus. Protection against tetanus is now a routine measure, and cholera inoculations are available whenever specially called for. We are carefully watching typhus inoculation experiments. Every factor for the spread of the commoner zymotic diseases was present in the first year of the war, and if we except German measles we have had practically nothing in spite of all forebodings. Our cerebrospinal fever figures compared favourably with civilian figures, and we have never had anything more than sporadic and widely separated cases. Now we have a good organization for the treatment of major infectious diseases, and should it be necessary we are prepared to convert some of the smaller hospitals for use as infectious hospitals. Every Station has a plan for dealing with outbreaks of the milder infections.

We have established centres for treatment of scabies all over the Command, and these are supervised by our skin specialists. A large number of beds are continually occupied on this account. The recent work of Kenneth Mellanby is most interesting and bears out the Army view that scabies is a disease of very close contact. We are convinced that our men and women are acquiring the infection while on leave. I can sense my civilian confrères' eagerness to tell me that the reverse is the case, but here my American colleagues of the Harvard Red Cross Hospital Unit tell me they have been following up cases in their homes and have found entire families affected.

This subject of contact between the civil and military assumes supreme importance when the question of louse-borne diseases is discussed. We should conduct a most strenuous campaign in all classes of the population on the danger of lice infestation. There seems to be an appalling ignorance of this; and the incidence of head infestation by lice, and nits, amongst our Service intake, i.e. recruits, has been, at times, alarmingly high. If the cause of this, as it well may be, is partly the fact that the modern method of women's hairdressing prohibits the washing of the sides and back of the head, because of the danger of spoiling a coiffure, then surely it is time that this style was laughed out of existence. I feel that we are losing a first-class method of propaganda.

We cannot disregard the head louse as a possible transmitter of typhus. At present we are trying to make a census of cases of pediculosis amongst troops. It is quite obvious that the louse, if not destroyed, can become a much greater dictator than any human we have yet known, and can take a much greater part in altering the destinies of nations than any conference of humans. Every Army M.O. should read Zinsser's book (1935, "Rats, Lice and History", London). Plans must be ready, civil and military authorities must act in the closest co-operation. In the Army, in this country, control should be easy, if conditions remain substantially as at present, but abroad the problem may be vastly different, if populations have—as they well may—to adapt themselves to an entirely different method of life, with all the standards now normally accepted drastically altered. It is only by education of *all ranks* that this can be assured.

The importance of the A.T.S. organization becomes greater daily. The special problems of enlisting bigger numbers of women have been largely overcome, and barracks, camps and reception stations have been adapted for their use. Difficulties arise with the small parties who are attached to units, but the larger depots are easily arranged. Conditions as regards mixed batteries in Southern Command are satisfactory, and the health of the auxiliaries has been good. They are being carefully watched in order to see what, if any, deleterious results accrue from their special duties. It is gratifying to note that the incidence of venereal disease has been negligible in this Command. In these mixed batteries I saw no signs of any deleterious effect on their health, physical or mental, and—perhaps this is a secret—Commanding Officers of the old school, who viewed such innovations with horror, are being completely won over and appreciate the quickness and general suitability of women for this work. Recent intakes of A.T.S. in my Command are working very satisfactorily and smoothly. The services of women medical advisers at Command Headquarters are much appreciated.

The British Army of to-day can only be of a certain size, because of the demands of industry and agriculture, and because of the limitation of the quantity of armaments which can be produced. We have not been faced with a similar problem for nearly two centuries.

depression, the drug was discontinued. There was still a heavy growth of *B. coli* in the urine. There were no blood changes. A total of 12 g. of phenothiazine had been given.

CASE V.—R. G., male, aged 3½ years. Admitted with *B. coli* bacilluria. Phenothiazine was given, 0.25 g. q.i.d. In three days the urine was sterile. Blood examination the same day revealed a relative lymphocytosis. The white cell count was 8,600, of which 69.5% were lymphocytes, 28% being polymorphs. In view of this blood-count the drug was discontinued. The child showed no toxic symptoms and was discharged from hospital. Twelve days later he was readmitted. There was an icteric tinge of the conjunctiva and general wasting. The abdomen was distended and tympanitic and the liver was enlarged 1 in. below the subcostal margin. The blood examination was repeated, showing a white count of 5,900, 40% polymorphs. and 54.5% lymphocytes, the lymphocytosis being less marked. The red cells appeared to be unaffected. Glucose was administered, and in seventeen days the condition had completely cleared up, but the relative lymphocytosis remained unchanged. This was considered to be a toxic jaundice and was directly attributed to the phenothiazine medication.

CASE VI.—C. W., male, aged 59 years, suffering from infected bilateral hydronephrosis. Both kidneys were enlarged and tender. The urine was thick with pus and contained a heavy *B. coli* infection. The patient had a persistent hiccup and became very drowsy. Phenothiazine 0.5 g. q.i.d. was given. The blood urea was 201 mg. per 100 ml. Phenothiazine 0.5 g. q.i.d. was given. He showed a steady improvement, the blood urea falling day by day and the urinary infection decreasing. The phenothiazine was continued for seven days, a total dose of 14 g. having been given. Blood-counts showed a fall in red cells to 2,030,000 and a 40% hæmoglobin estimation. The drug was discontinued. Blood transfusion was given and with iron medication the blood picture returned to a satisfactory level. As far as one could judge, the urinary infection responded to phenothiazine, but a marked toxic æmia followed.

CASE VII.—E. H., female, aged 37 years. A *B. coli* bacilluria complicated this patient's convalescence following subtotal hysterectomy and bilateral salpingo-oophorectomy for bilateral tubo-ovarian abscesses. The routine phenothiazine medication was carried out over eight days. A total dose of 16 g. was given with no effect on the urinary infection, which still showed a heavy growth although the symptoms were alleviated. There were no blood changes in this case.

CASE VIII.—M. H., female, aged 38 years. Repeated attacks of left loin pain associated with frequency and dysuria had given this patient much distress over the past twelve months. Investigations revealed a double ureter and pelvis on the left side, complicated by a *B. coli* infection. Phenothiazine and ammonium chloride were given for seven days without any effect on the urinary infection, as shown by repeated cultures. In all, 14 g. of phenothiazine were given. There were no blood changes of any note.

CONCLUSION

In this small series of 8 cases, only 2 showed improvement in the urinary tract infection. 6 cases sustained a severe degree of R.B.C. destruction. Two cases of relative lymphocytosis with a low polymorphonuclear cell-count suggested that the bone-marrow had not escaped damage. Toxic hepatitis followed in 1 of these 2 cases. The conclusion was reached that phenothiazine has no place in the treatment of urinary tract infection.

[I should like to express my thanks to Messrs. Burroughs Wellcome, who supplied the drug, and to Dr. Prescott, of the Wellcome Research Laboratory, for his support in initiating the trial. My thanks are also due to the Medical Superintendents of St. Mary Abbots and Fulham Hospitals, and the staff of the Group Laboratory, for their kind co-operation.]

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In the subsequent discussion it was agreed that many advertised urinary antiseptics were spurious. The only effect they had was to show some colour in the urine and as antiseptics they were useless.

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cells had fallen from 4,410,000 to 1,510,000 and the hæmoglobin from 84% to 34% with a white cell increase from 11,250 to 21,650. The authors concluded that anæmia produced by phenothiazine is an exceptional and rare occurrence, but advised that the blood should be examined periodically and any evidence of anæmia should be the signal for stopping medication. After a maximum total dose of 15 g. a rest-period was advised. These findings led to the hope that an alternative to sulphonamide (with its subjective symptoms of nausea and depression) had been found to overcome those chronic infections resistant to the usual line of treatment consisting of increased fluid intake and change in the reaction of the urine, to which many cases of urinary tract infection respond. Accordingly it was decided to repeat this clinical trial with phenothiazine. With the support of the Research Committee of the London County Council, we had at our disposal a large number of suitable cases which could be kept under close observation.

Phenothiazine in tabs. 0·5 g. were given three to four times daily. The urine was maintained at a pH 4·5 to 5·5 by tab. ammonium chloride 0·5 g. q.i.d. before administration of the phenothiazine, and then on alternate days. Bacteriological examinations were made during the course of treatment. Blood-counts were done, and after six days of treatment were repeated unless symptoms indicated an earlier examination. Only 8 cases had been subjected to the trial treatment when the alarming blood pictures and the same small percentage of improved cases showed that continuation was unjustified, and the trial was abandoned.

CASE I.—F. A., female; aged 20 years. Gave a history of dysuria and frequency at intervals for the previous twelve months. Bacteriological examination of the urine and culture showed numerous colonies of paracolon bacilli. Pyelography showed no abnormality. Cystoscopy revealed slight trigonitis. Guinea-pig inoculation for tubercle bacilli was negative. A course of sulphapyridine gave no relief. Phenothiazine was given a trial, 4 tablets being administered daily with ammonium chloride. After seven days' treatment the appearance of the patient suggested an increasing anæmia. Blood examination showed that the red cells had fallen from 5,680,000 to 2,960,000 and the hæmoglobin from 108% to 54%. In all, 16 g. of phenothiazine had been given without improvement in symptoms or in the bacteriological content of the urine. The anæmia responded well to iron medication, the blood-count and hæmoglobin percentage returned to normal within one month. This suggested that the anæmia was of a toxic nature and that the bone-marrow was unaffected.

CASE II.—C. J., male, aged 67 years. Admitted suffering from acute retention of urine due to prostatic enlargement. Catheter drainage was instituted *per urethram*. The urine was heavily infected. Bacteriological examination showed a heavy growth of coliform organisms. Urethritis developed, and as the general condition of the patient necessitated it, suprapubic cystostomy was performed and the vasa deferentia divided.

The wound became infected and suppurating, and a profuse discharge came from the site of division of the vasa. The urethritis persisted. Phenothiazine 0·5 g. was given four times daily along with ammonium chloride. After seventy-two hours there was a definite improvement in the condition. The medication was continued for eighteen days, by which time the suppuration had ceased, the wounds had healed, and the urine was sterile. 36 g. of phenothiazine had been given. The patient looked anæmic, and blood examination showed that the R.B.C. had fallen from 4,350,000 before treatment to 3,070,000, the hæmoglobin from 68% to 54%. The phenothiazine was stopped and iron medication begun. The patient became more anæmic, as was shown by blood examination eight days later when the red cells had fallen to 2,880,000 and the hæmoglobin to 42%. A blood transfusion was given, after which the patient made a rapid recovery. The infection in this case apparently responded to the phenothiazine.

CASE III.—W. O. F., female, aged 23 years. This woman was symptomless, but had a heavy *B. coli* bacilluria persisting after an attack of pyelitis. Pyelography and cystoscopy revealed no abnormality in the urinary tract. A daily total dose of 2 g. of phenothiazine was given with ammonium chloride. Repeated bacteriological examinations showed no change in the growth of *B. coli*. Treatment was continued for thirteen days, 26 g. of phenothiazine having been given. Blood-count examination on the twelfth day of treatment showed a relative lymphocytosis and W.B.C. of 5,600 with polymorphs. 29% and lymphocytes 61·5%. The treatment was discontinued. The *B. coli* infection had not responded to phenothiazine.

CASE IV.—M. N., female, aged 51 years. Complaining of frequency and dysuria. Urine gave a heavy growth of *B. coli*. On cystoscopy there was well-marked trigonitis. Phenothiazine was given, 0·5 g. four times daily, along with ammonium chloride. After three days the symptoms definitely improved, but bacteriological examination showed no difference in the urinary infection. In six days, owing to nausea, sweating and

depression, the drug was discontinued. There was still a heavy growth of *B. coli* in the urine. There were no blood changes. A total of 12 g. of phenothiazine had been given.

CASE V.—R. G., male, aged 3½ years. Admitted with *B. coli* bacilluria. Phenothiazine was given, 0.25 g. q.i.d. In three days the urine was sterile. Blood examination the same day revealed a relative lymphocytosis. The white cell count was 8,600, of which 69.5% were lymphocytes, 28% being polymorphs. In view of this blood-count the drug was discontinued. The child showed no toxic symptoms and was discharged from hospital. Twelve days later he was readmitted. There was an icteric tinge of the conjunctiva and general wasting. The abdomen was distended and tympanitic and the liver was enlarged 1 in. below the subcostal margin. The blood examination was repeated, showing a white count of 5,900, 40% polymorphs. and 54.5% lymphocytes, the lymphocytosis being less marked. The red cells appeared to be unaffected. Glucose was administered, and in seventeen days the condition had completely cleared up, but the relative lymphocytosis remained unchanged. This was considered to be a toxic jaundice and was directly attributed to the phenothiazine medication.

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Refractory Ulcerative Cystitis.

By A. WILFRID ADAMS, M.S.

Terminology.—The condition of refractory ulcerative cystitis has been called after names of surgeons and by somewhat poetic titles. It would be advantageous for the sake of popularizing the diagnosis in the medical mind to group together under one clinical title—such as heads these remarks—the lesions known as Fenwick's solitary ulcer, Hunner's elusive ulcer, pan-mural, interstitial cystitis, &c. The disease may be uncommon but early recognition is important, if we are to save its victims from a pernicious scourge. It is well to remember that the rarity may be more apparent than real. If the possibility of the disease is not in his mind the doctor will not, in milder cases, feel moved to send the patient for the revealing cystoscopy. A suitable title will help the doctor to recall that there is an organic cause of refractory cystitis which is liable to rank as trivial or functional disease because the customary changes in the urine are wanting and, instead of physical deterioration, there is often increase in weight. The simple truth is at present so obscured by a multiplicity of names that we can scarcely blame the practitioner for the protracted histories of suffering which the patients usually tell us.

Symptoms.—The multiple type of ulcer (Hunner [1]) occurred in five of the present seven cases reported, all women, ages 36, 21, 68, 58, 44 years. The solitary ulcers (Fenwick [2]) were in men, ages 62 and 66 years.

The complaints start rather abruptly with frequency and, usually, pain at the end of micturition. Hæmaturia, which may be heavy, occurs at times in about half the cases. The dysuria is apt so to increase that the patient is tied to the house. In addition, spontaneous stabbing pains may be felt through the pubic and rectal regions and be aggravated by mechanical causes, e.g. movements or by lateral recumbency. The patient may sense the exact locality of a sore or sores in the bladder itself. One, F. W., calls such "ulcers", as the pain derived reminds her of the sore ulceration in the mouth to which she is also prone. In the sixth year of her illness the pain extends as an ache through both legs to the great toes, especially the left.

All the patients were of average physique and a psychopathic taint only manifested in one (D. M. A.).

The urine looks inoffensive and the pathologist merely reports pus and red blood cells. Later, when necrotic changes are going on, *B. coli* and streptococci appear. Guinea-pigs were inoculated in four cases with negative results.

Urography.—In six out of seven patients pyelography was used. The two cases of solitary ulcer showed right hydronephrosis, slight in W. B. (pelvis $\times 2$), and in the man R. P., it was definite (pelvis $\times 4$). In none did it suggest a causal relationship, and is discussed later.

(A) THE MULTIPLE TYPE OF ULCER

Cystoscopy is facilitated in these irritable bladders by low spinal anæsthesia.

Stretching the ulcers is wont to provoke bleeding and, therefore, calls for cautious filling of the bladder.

The posterior hemisphere well above the trigone is the site of the lesions. In one case (D. G.) there was an appearance akin, but not due to, Koch's infection at the left ureteric orifice. This was swollen and appeared fissured, whilst round it the mucosa was studded with little tubercles. In moderate cases two or three little irregular patches of slightly raised hyperæmic mucosa are visible on a background of ordinary straw colour. They may amount to little more than trivial marks resembling spider nævi, even in cases with long histories. With time or subsequent to diathermy the inflammation tends to develop into ridges on which ragged linear ulceration develops. Attached to the ulcers fibrinous strands are often seen, looking like cobwebs. Issuing blood may appear in the form of waving streamers and in one instance on pressure with an electrode, creamy pus was seen. In advanced cases the areas are broad with shallow ulcerative erosions, as if a mouse had nibbled the bladder wall. Phosphatic incrustation may be seen.

Case Reports: I.—D. M. A., 36 years, spinster, gave a history of acid water for two years and marked frequency, day and night. There was an interval of six months' return to normal between the onset and the subsequent symptoms. She felt "acid run round the bladder after meals". Bladder washes greatly aggravated the symptoms. The urine was faintly red, although the patient had not observed any abnormality. When, in November 1940, diathermy was started, two ragged granulating ulcers were present $\frac{3}{4}$ in. in diameter, on the posterior wall. The dysuria abated to negligible proportions but there was recrudescence, which tonsillectomy in the spring of 1941 did not benefit. By 23.7.41, three ulcers were seen and more radical measures were likely to be necessary. She developed mild delusions for a time, but on 23.2.42, the doctor reports "much improved and happily teaching".

Conclusion.—One is left with the impression that the condition is barely controllable by diathermy, although the last application has effected notable improvement.

II.—The woman D. G., 21 years, came to me first on 31.3.37. Like the preceding case, she was single and well nourished. There was slight suprapubic tenderness. Frequency had developed during the previous four years and there had been hæmaturia. A course of salvarsan gave no persistent benefit, so, to a large red jelly-like patch on the dome of the bladder, diathermy was applied. Hers was the ureteric orifice which, at the next cystoscopy, somewhat resembled tuberculous infection, but investigation refuted the idea. By 28.1.38, operation and excision of the affected area in the dome of the bladder became necessary. It was an elevated thickening of the submucosa, like a button about $\frac{3}{4}$ in. in diameter but not ulcerated. The histology conformed with that of Hunner's ulcer. She appeared cured and looked radiantly well on 30.5.38, but two days after marrying (against my advice) in October 1939, she developed a frequency which has confined her to the house ever since. The last cystoscopy was in 1940 when a large part of the posterior bladder wall showed ragged ulceration surrounded by a raised, rolled edge. She still (9.2.42) rejects proffered transplantation of the ureters.

Conclusion.—Her story includes a disappointing relapse and demonstrates the pernicious nature of the lesion, the patient's imprisonment by it and the only likely cure to lie in the transfer of the bladder function to the bowel.

III.—In the case of L. H., 68 years, a widow who came on 8.3.40, pain and frequency had troubled her for four years. Occasional stabbing pains into the rectum also occurred. The bladder showed areas of condensed vascularity with tiny petechial blobs on the capillaries, like terminal buds on twigs, as well as woolly patches of mucosa with central ragged ulcers and tags of granulation tissue. Neither diathermy nor a course of salvarsan prevented the lesion extending round the back of the bladder in the characteristic irregular crescent from about 1 in. above the right to a similar level above the left ureter. The frequency is aggravated by jolting in buses or by agitation. She was still able to get out of doors a little, till a month ago.

Here again, ureteric transplantation seems preferable to leaving the patient in her miserable state.

[POSTSCRIPT (15.4.42).—Since writing the above bladder lavage with silver nitrate has been followed by marked relief.]

IV.—Mrs. M. C. S., 58 years, similarly for four years suffered from dysuria before reaching me. Hæmaturia had developed and she passed bits of flesh in the water which had a heavy *B. coli* infection. Cystoscopy showed multiple shallow erosions and an appearance simulating cancer. Suprapubic cystostomy was performed for biopsy and with a view to possible ureteric transplantation. Shallow ragged ulcers without any induration or thickening were found. Dr. A. D. Fraser reported on the excised portion: "non-malignant ulceration conforming with Hunner's type." She refused further treatment and I am unable to obtain subsequent reports of her condition.

V.—Miss F. W., 44 years, gave in February 1939 a history of three years' chronic cystitis and recent inability to lie on the left side. At cystoscopy the patches of cystitis bled and prevented ureteric catheterization but this was achieved subsequently with negative findings. A course of salvarsan was given without benefit. A year later two small hæmorrhagic areas only were seen near the dome of the bladder posteriorly, with a tendency to polyposis but no visible ulcer. On 4.2.42 never having had operative treatment by the cystoscope or otherwise, she said the pain and frequency were getting worse and she had not left the house for about a year. In addition to the dysuria, aching in the bladder spreads down her legs, especially the left one, to the great toes. Her weight has increased despite the suffering.

At cystoscopy, 18.2.42, about 6 oz. of clear urine ran out and the ureteric orifices were normal. An irregular arch of chronic ulceration extended round the posterior wall of the bladder in the usual site. On the right the surface looked greyish-white and irre-

gularly eroded, as if nibbled by a mouse. The margin was deep red in places. The central region more resembled icing sugar, while to the left was a white area decorated by bare interlacing bars of muscle, denuded as by exfoliative cystitis and neatly bounded by an even, rather geometrical margin. This part simulated an abiotrophic lesion rather than an advancing infective or malignant ulcer. Inspection was partly hampered by instrumental defect and, at the close, streamers of blood suddenly appeared from the white ulceration. The patient complained of pain and shortly after vomited. As this was happening a rent became visible through which the shreddy margins tended to be carried into the dark space beyond the bladder. Evidently erosion had thinned the viscus which could not withstand continued distension. Prompt laparotomy revealed the entry of lotion into the abdomen but no tear was visible in the peritoneum over the bladder. The latter was opened, the ulcerations were probed and a hole found on the left side, but how it communicated with the peritoneum was obscure. The perforated area of the bladder was excised and a suprapubic drain installed. Dr. Fraser reports the presence of pyuria obviously originating in some other process than the mild infection with *B. coli* and streptococci would explain. Histology showed no evidence of neoplasm and no organisms could be demonstrated.

Here is another instance of the protracted nature of this disease, which has culminated in pathological rupture of the bladder and provides a warning of the risk of this very rare complication of cystoscopy. It further emphasizes a pathological feature notable in two other of these bladders exposed at operation, viz., the absence of any protective thickening of the bladder wall or peritoneum in advance of the ulcerative process.

[POSTSCRIPT (9.4.42).—This patient's wound is healed. Micturition is painless and occurs only eight times in 24 hours. She has acquired a vigorous, healthy, happy appearance and walks well, which is a remarkable sequel to years of invalidism culminating in a grave calamity.]

Review of Five Cases of Multiple Type of Refractory Ulcerative Cystitis

(1) All are women, three being single.

(2) Ulceration tends to develop an irregular crescentic distribution across the upper zone of the posterior bladder wall. What bearing has this on aetiology? Is it a local peculiarity of vesical blood supply that renders this part prone? The contiguity of the uterus is conceivably influential but no gynaecological disorders were noted.

(3) The usual absence of thickening in advance of the ulcers received poignant proof by pathological rupture during one cystoscopy.

(4) Salvarsan has not helped. Cystoscopic diathermy relieves but relapse is common. This may also occur many months after an apparently successful excision.

(5) In consideration for the patient's feelings and lest her healing powers become exhausted, there should be timely resort to ureteric transplantation.

(6) Though uncommon, the disease demands earnest attention, for it is apt to impose a sentence of most painful penal servitude on its victims.

(B) SOLITARY ULCER

For clinical purposes, and since neither the multiple nor solitary type is common, this variety is well included under the generic clinical title of "refractory ulcerative cystitis".

Two examples are reported—men in the sixties. They complain to the doctor of the same protracted symptoms as the women, viz. relentless frequency at the onset and, later, scalding micturition. There was intermittent hæmaturia in one (W. B.).

At cystoscopy the pathological distinction from the multiple type becomes apparent. As the appearances of the lesions were not uniform it seems better to describe them with the individual case reports.

Mr. W. B., 66 years, came in September 1940 with a history of half-hourly frequency and hæmaturia on four occasions. Pain had only supervened since instrumentation at another hospital a month before, up to which time he was working. There was nothing in the family history bearing on the illness. Physique was fair for his age. The urine was that of acute cystitis with an infection of *B. coli* and streptococci. Urography showed competent kidneys but there was a slight suggestion of stasis and kinking in the right ureter which may have a bearing on later complications.

At cystoscopy the bladder capacity was about 6 oz. A sessile plaque of growth, rounded and about 1½ in. broad, with surface half clouded by fluffy material, was seen rising from about the middle of the posterior wall, an inch from the right ureter. The catheter passed readily through the right ureter, the wide mouth of which appeared retracted under an anterior ledge. The prostate was somewhat conical. On 6.6.41 partial cystectomy and vasotomy under spinal anaesthesia were undertaken. The lesion appeared as a slight thickening, notably rough compared to the normal mucosa around. It was removed with a ½ in. belt of healthy tissue and naked-eye section showed the submucosa ⅓ in. thick in contrast to the normal ⅓ in. My cystoscopic diagnosis of carcinoma was invalidated. Fashioning a bladder out of the small remains of the organ was an exacting task. Dr. Fraser reports: "This is a large ulcer with a base formed of exuberant subacute granulation tissue. No organisms can be seen and there is no evidence of carcinoma. At one point there is a suggestion of tuberculous infection but on the whole the appearances are those of a simple ulcer of the bladder and would agree with Hunner's ulcer."

Suprapubic leakage continued and I blamed the prostate. This was treated by resectoscopy in September 1941 and only recently has a suprapubic sinus become dry. Sepsis is present now (15.4.42) in his right urinary tract to whose slight structural imperfections allusion was made above. Right nephrectomy is needed because although his temperature and pulse remain normal between them, he has recurrent rigors and vomiting with some tenderness in the right loin every few weeks. The urine shows a little pus, there is hourly frequency but otherwise he is doing well. Another surgeon who cystoscoped him in November 1941 saw satisfactory healing within the bladder.

The second case of solitary ulcer occurred in Mr. R. P., aged 62, a fitter in aero works, who came in September 1940 with a history of frequency $\frac{D^{20}}{N^{10}}$ for ten years. There was no remission of symptoms, yet he persevered at work. The trouble started a few months after a sudden retention had required relief by catheter. The urine was always clear and micturition painless until about two months before admission. Since then, so acute was the dysuria that his mates had thrice found him faint on the floor of the water closet and thick white jelly appeared in the urine.

He was a spare active man, very tender over the right kidney and slightly so over the bladder. Urography showed right hydronephrosis (pelvis $\times 4$) and a systolic bladder. *B. coli* and streptococci were present in the urine.

At cystoscopy, the small hyperaemic bladder showed a deep ovoid ulcer about the site of the right ureter and measuring ½ in. in its long (vertical) axis. It had a grey necrotic base with an edge raised and, at the lower end, resembling epithelioma. Indigo-carmin was injected but no efflux seen from either ureter fifteen minutes later and the latter defied detection.

16.9.40: Exploration of exterior of bladder and right ureterostomy. The bladder was first completely divested of its peritoneum. It was thickened uniformly but supple. The region of the ulcer did not feel like neoplasm. Right ureterostomy appeared essential and allowed catheterization. At the same time a portion was removed for biopsy. Both a guinea-pig test and histology excluded tubercle, and time carcinoma, for he soon became very well. Two months later the pelvis of the right kidney was normal on pyelography. Ten months later he felt perfectly well, the bladder held ½ pt., and was normal on cystoscopy.

On 16.7.41 uretero-colostomy replaced his external fistula. After a somewhat stormy time he healed and has been working. On cystoscopy, 11.2.42, the right ureteric orifice was both visible and normal. 1 in. above it is a triangular recessed area which is white and smooth and the only vestige of the menacing ulcer which tyrannized over him for ten years. His motion, somewhat soft, does not come away too frequently. Residual urine is clear and less than 1 oz. Micturition is normal but *B. coli* infection persists. He seems perfectly well clinically.

Review of Two Cases of Solitary Ulcer

(1) Presumably the two cases represent the early proliferative and the late non-specific ulcerated stage of interstitial cystitis—in Fenwick's view somewhat analogous with peptic ulcer.

(2) In contrast with the multiple ulcers in women this lesion is solitary, more basal and there is marked proliferation in advance of the ulceration. The different sexual anatomy possibly accounts for this variation in men and women.

(3) It appears to be more amenable to ordinary surgical treatment than is the type with multiple ulcer.

(4) The efficacy finally of resorting to diversion of the urine; even when this was done unilaterally it proved curative in one case.

(5) In other respects the conclusions on the multiple cases apply.

ABSTRACT

(1) Refractory Ulcerative Cystitis is proposed as a title for covering this group of rare but kindred ills obscured at present by a medley of names and apt to be neglected.

(2) From this revised nomenclature that clinical clarity may result which is needed to keep the disease more in the doctor's mind and so shorten the long history of misery these victims usually suffer and which amounts to little less than most painful penal servitude.

(3) Seven cases are reported. Five of multiple variety in women and two solitary in men. Symptoms, pathology and treatment are reviewed.

(4) Outstanding frequency and pain sharply contrast with slight pyuria in refractory ulcerative cystitis.

(5) While it was distended during cystoscopy, pathological rupture occurred in one bladder with advanced multiple ulceration.

(6) A plea is made for research into the ulterior cause of this primary inflammatory infiltration of the bladder wall. Noteworthy are some points of resemblance to peptic and colitic ulceration.

REFERENCES

- 1 HUNNER, GUY L. (1918), *J. A. M. A.*, 70, 203.
- 2 FENWICK, E. HURRY (1900), "Ulceration of the Bladder". London.

DISCUSSION.—Mr. A. CLIFFORD MORSON made a point of the ambiguity of the cystoscopic appearance, the disease simulating malignant ulcer of the bladder. He emphasized the need for research on the pathological side.

Mr. E. W. RICHES mentioned the method of treatment in elderly women beyond the age suitable for operation of repeated cautious distention of the bladder.

Mr. T. J. MILLIN supported this, saying that in his experience he had found it of good effect.

The meeting agreed that ureteric transplantation should be done without waiting too long in the severe case.

Section of Anæsthetics

President—A. D. MARSTON, D.A.

[February 6, 1942]

Resuscitation

By GEOFFREY ORGANE, M.A., M.D., D.A.

Causes of Death on the Table

ULTIMATELY, death is due to heart failure, which may be primary or secondary.

Primary heart failure may be due to sudden stopping of the heart or to ventricular fibrillation. It occurs, most commonly, during light anæsthesia, either during induction or shortly after the start of the operation. Heart failure due, solely, to an overdose of anæsthetic probably does not occur; the dose of ether required to stop the heart is nearly half as much again as the dose which stops respiration. Chloroform anæsthesia, uncomplicated by operative stimuli, may be taken down to respiratory failure, when the heart will continue to beat until the effects of anoxæmia are felt (Meek, 1941).

Sudden stopping of the heart is probably due to vagal inhibition excited reflexly from the respiratory tract or from a painful stimulus elsewhere. It may be initiated, for instance, by the application of concentrated chloroform vapour to the nasal mucous membrane. It is often associated with the presence of lymphatic hyperplasia—I dare not say status lymphaticus! The heart itself may be entirely healthy and will resume its normal rhythm on the application of a suitable stimulus.

Ventricular fibrillation, which appears to be the commoner condition, is usually set off by the same stimulus, but it seems to depend on the presence of an abnormally irritable myocardium. Vagal inhibition of the normal pacemaker, here releases lower ectopic foci of irritability which set off the irregular contractions. Among anæsthetics, cyclopropane and chloroform are the chief offenders; yet sudden death during induction with cyclopropane (that is, without operative stimuli) is comparatively rare, probably because the stimulus provided by chloroform vapour is lacking. It is the present fashion to regard all cases of sudden heart failure as due to fibrillation—a point obviously rarely capable of verification in human beings. Guedel (1937) quotes, as such, three cases of heart failure under light ether anæsthesia. There is no conclusive evidence that ether does appreciably sensitize the myocardium, and it seems more likely that these were instances of cardiac standstill. On the basis of available writings, I question whether ventricular fibrillation occurs under ether anæsthesia except when large amounts of adrenaline have been injected by the surgeon. The role of adrenaline in these cases is not quite clear. Nobel and Rothberger (Meek, 1941) thought it stimulated the vagus, inhibiting the upper centres, and allowing the escape of lower foci in the auricles and ventricles; in large doses, certainly, it also directly stimulates the ventricles. The lower centres, not normally being under any vagal control, respond with a few scattered beats which can rapidly increase, developing into a multifocal ventricular tachycardia and finally ventricular fibrillation. The protective action against adrenaline of atropine, or of cutting the vagi, eliminates the vagal inhibition. The sinus node is accelerated and, to become dominant, the ventricular automatic centres have to be stimulated to a much higher degree if ventricular tachycardia is to develop. Nathanson, quoted by Meek, induced cardiac standstill in the human being and then produced automaticity in the lower centres by an injection of adrenaline. This was attributed to a direct action of adrenaline on the heart muscle.

From all this emerges the fact that optimal conditions for the production of ventricular fibrillation arise with the injection into the ventricle of adrenaline when the heart has stopped.

Substances which decrease heart excitability eliminate the adrenaline effect; such are quinine and quinidine orally (Bardier and Stillmunkes, 1922, 1926), and procaine (Shen and Simon, 1938) and cocaine (Hermann and Jourdan, 1931) intravenously. Burstein and others (1940) reported some remarkable instances of recovery in dogs after the intracardiac injection of procaine, 5-10 mg. per kilo, in 5 c.c. of normal saline.

Secondary heart failure is due to anoxia of the myocardium; failure occurs earlier with ethyl chloride or chloroform owing to the toxic effect of the anæsthetic, which is directly proportional to the dose.

Anoxia of the heart muscle may be part of a generalized anoxæmia due to respira-

tory-failure or obstruction, or to breathing an atmosphere deficient in oxygen; it may be due to anaemia; it may be due to a deficient coronary circulation resulting from peripheral circulatory failure. Anoxaemia plays, also, a secondary part in increasing the tendency to cardiac irregularities leading to fibrillation.

Treatment

The treatment may be discussed as preventive and restorative. Preventive treatment, apart from the avoidance of grosser errors, is of little effect in primary heart failure; restorative measures are likely to be ineffective in secondary heart failure.

Preventive treatment in secondary heart failure.—The main indications in the prevention of secondary heart failure are for the maintenance of (a) blood-pressure, of (b) an adequate volume of circulating blood, and, of course, of (c) an adequate supply of oxygen.

(a) *Blood-pressure*: It seems reasonable, where blood-pressure fall is due to peripheral vasodilatation, as in spinal anaesthesia, to use a drug which will constrict the blood-vessels. The most widely used is ephedrine, which gives good results; neosynephrine, of which I have no personal experience, may be better—of all the sympathetomimetic drugs it is the least likely to produce cardiac irregularities. Both are very closely related, chemically, to adrenaline. Neosynephrine is less active than ephedrine and its effect is relatively transient, but it may be repeated without loss of efficiency and has been found effective where ephedrine had failed (Lorhan and Lalich, 1940, Bittrich, 1939); the recommended dose after a fall in blood-pressure is one or two minims of a 1% solution, intravenously, repeated as necessary. Prophylactically, not more than one grain of ephedrine should be given intramuscularly; with larger doses the blood-pressure often rises to 40-50 mm.Hg above the normal. To combat an already established fall in blood-pressure, ephedrine, gr. $\frac{1}{4}$ to $\frac{1}{2}$, may be injected into a vein. The recovery is dramatic and is fully established in one or two minutes: it is better to repeat small doses than to risk the development of cardiac complications by an overdose. It is probably unwise to allow the systolic blood-pressure to fall below 80 mm.Hg.

(b) *Blood volume*: Lack of an adequate volume of circulating blood is the basic factor in the production of what we call shock and, in that condition, is accompanied by an increased permeability of the capillary walls, allowing leakage of plasma into the tissues; this is aggravated by anoxia. The normal reaction to fall in blood-pressure due to hæmorrhage or similar cause is peripheral vasoconstriction which is present in shock; the use, here, of a vasoconstricting drug still further reduces the supply of oxygenated blood to the capillaries and produces, ultimately, a deterioration rather than an improvement.

Cold and fear are important contributory factors which will require attention.

You will remember that chloroform and ether increase capillary permeability. The use of ether intravenously was discarded nearly thirty years ago, except as a museum piece, on account of the frequent occurrence of grave complications resulting directly from this. Magnus (Marx, 1931) failed to produce œdema with large saline infusions in normal rabbits unless they had been anaesthetized with chloroform, ether or chloral hydrate. It seems clear, on the evidence at present available, that ether and chloroform are to be avoided, except in minimal quantities, where the patient's condition gives rise to anxiety.

Restoration of the blood volume will, if the shock is not too firmly established, result in vasodilatation and a more efficient circulation. Quite a severe degree of anaemia can be tolerated provided the blood volume is adequate; this accounts, in part, for the greater severity of symptoms resulting from acute than from chronic anaemia.

The indication is for the intravenous administration of fluids. Grant (1942) suggests that a neglected systolic blood-pressure of 80 mm.Hg or less may result in the development of that condition of intense vasospasm in which introduction of fluids is difficult and response is poor. We may therefore take as our standard, requiring correction, a systolic blood-pressure of 90 mm.Hg or less, and aim to maintain it at a little over 100 mm.Hg.

Hypodermoclysis is clearly useless in a condition where fluid is being lost from the blood into the tissues. Intravenous saline has an immediate stimulating effect and, in a mild case, may succeed in breaking the vicious circle. Usually, serum or plasma will be necessary; blood is indicated where hæmorrhage has been severe or where anaemia was present before operation; in shock with hæmoconcentration it is clearly unnecessary and even undesirable. There seems to be no limit to the amount that can be given. Grant used from two to thirteen pints of blood (the last in the space of twenty-eight hours). The first pint or even more may be run into the vein as fast as possible (usually taking five minutes). The amount of blood given should be limited to the

estimated amount lost, further fluid required, as indicated by the blood-pressure, being made up with serum or plasma, or even saline. We are now told that the renal failure sometimes ensuing is due to the crush syndrome and has nothing to do with the quantity of blood given.

The use of so-called cardiac stimulants in a patient gravely ill from shock or hæmorrhage may be dangerous.

CASE I.—A man, aged 52, was gravely ill from hæmorrhage during a perineo-abdominal excision of the rectum, the blood-pressure being unrecordable. Coramine, 1·7 c.c., was injected intravenously, whereupon he died, anticipating the natural course of events, probably by ten or fifteen minutes.

(c) *Oxygen supply*: The supply of oxygen to the blood may be reduced by respiratory obstruction; by an inadequate proportion of oxygen in the anæsthetic mixture; by deficient pulmonary ventilation; by impaired gaseous exchange (as after poison gas). In all these, cyanosis will be present and the remedy clear.

The question arises of giving large proportions of oxygen to patients with a diminished oxygen-carrying capacity of the blood. With normal pulmonary ventilation, negligible improvement is produced by increasing the oxygen above 30%. Where breathing is also depressed, as is often the case, higher proportions of oxygen will mask the deficiency. It is questionable whether it is not better to rely mainly on increasing the respiratory movement, incidentally assisting the circulation. This may be done by some form of artificial respiration or by using carefully controlled amounts of carbon dioxide, not exceeding 5%.

Where the respiratory centre remains depressed in the presence of adequate oxygenation, a respiratory stimulant may be considered. For this purpose, picrotoxin, 3 mg., cardiazol, $\frac{1}{2}$ -1 c.c., coramine, 1-2 c.c., or alpha-lobeline, intravenously, are suitable. Intravenous injection is, to my mind, preferable; the dose may be divided and the effect exactly judged. The dose required is less than may be used in barbiturate narcosis for its analeptic effect and should be no larger than is needed, lest a secondary depressant effect come into play. It should be borne in mind that the usual analeptic dose of coramine—5 c.c.—is the same as that used for the production of convulsions in the treatment of schizophrenia.

Clearly, in the gassed patient, with impaired gaseous exchange in the lungs, the oxygen percentage must be as high as possible. I can think of no anæsthetic as suitable as cyclopropane.

As both lowered blood-pressure and oxygen lack enhance the depressant effects of anæsthesia, an essential part in the treatment of a patient whose condition shows deterioration consists of the withdrawal of the anæsthetic. If avertin or a barbiturate is playing any considerable part in the anæsthesia, an analeptic dose of cardiazol or coramine should be given intravenously—sufficient to produce the desired result.

Preventive Treatment of Sudden Heart Failure

The preventive treatment of primary heart failure consists of pre-operative atropine or scopolamine to inhibit the vagus; the avoidance of the chief culprit—chloroform; avoidance of excitement during induction of anæsthesia and of premature operative stimuli; exceeding caution in the use of adrenaline—absolutely contra-indicated with chloroform or cyclopropane.

The direct protective action of barbiturates against untoward effects from adrenaline has lately been questioned (Orth *et al.*, 1941). Their value in by-passing the second stage of anæsthesia is obvious.

Restorative treatment in primary heart failure.—Established methods: Many textbooks of surgery confine themselves to the statement that cardiac massage through the diaphragm has been found of value; others suggest the intraventricular injection of adrenaline, $\frac{1}{10}$ 10, ether, $\frac{1}{20}$ 20, pituitrin, $\frac{1}{2}$ c.c. Hewer (1939) mentions also strophanthus, digitalis, camphor, coramine, strychnine and dextrose, pointing out that their diversity of action suggests strongly that it is the mechanical stimulus of the injection that is responsible for the restoration of the heart beat; to this stimulus the auricle is much more sensitive than the ventricle.

Drugs.—Intravenous, intramuscular or subcutaneous injections are clearly useless without a circulation. For intracardiac injection, the drugs most commonly in use are adrenaline and coramine. Coramine has no direct action on the heart and is not indicated.

Gunn and Martin (1915) found, in dogs, where twenty minutes of direct cardiac massage had failed, that intrapericardial injection of adrenaline restored the heart beat almost at once. It was unsuccessful alone. We know that adrenaline can initiate ventricular fibrillation under chloroform and, in sufficiently large doses, under ether. As it acts directly on the ventricles it seems reasonable to suppose that the intraventricular injection of the usual dose (1:1,000 solution, 10-15 M) may produce fibrillation with ether, especially where the tendency to irregularities has been enhanced by a period of anoxia. Tournade and others (1932) reported recovery from chloroform syncope in the dog by intravenous or intracardiac injection of adrenaline combined with massage and artificial respiration. Meek comments "these must have been a simple toxic depression of the heart, a real chloroform syncope". One in ten of these developed fibrillation.

The following case illustrated the inadequacy of accepted methods of resuscitation:

CASE II.—A boy, aged 13, was anesthetized by a house surgeon for bilateral inguinal herniorrhaphy. Premedication was omnopon gr. $\frac{1}{4}$ and scopolamine gr. $\frac{1}{16}$ (far too large a dose) two hours beforehand. The anæsthetic was open ether, preceded by ethyl chloride, and the course was at first uneventful. Shortly after the incision on the second side, under what was said to be deep anæsthesia, he *suddenly* stopped breathing, was pulseless and apparently dead. Adrenaline, 1:1,000, M 10 was injected into a ventricle. On attempting cardiac massage through an abdominal wound, the surgeon found what he described as irregular, feeble contractions—evidently ventricular fibrillation. The lungs were then inflated at regular intervals with oxygen under pressure. Regular, very forcible heart beats started and his colour returned. The pupils were $\frac{1}{2}$ dilated. After ten minutes of artificial respiration by compressing a closed re-breathing bag, occasionally emptying and refilling with oxygen, breathing recommenced. He was returned to the ward in about twenty-five minutes, breathing quietly and with pupils half dilated. The pulse was slower and the heart beat less exaggerated.

[Two hours later, twitching was noticed in the arms and legs. In the next four hours he had several generalized fits. It was found that he had had continuous intranasal oxygen with carbon dioxide, which was at once stopped; there were no more generalized fits. Five hours later he was less deeply comatose and definitely irritable, responding to manipulations by whimpering and mild convulsive movements, mainly in the limbs, especially the arms, but also in the face. The oral airway was removed with the aid of a gag, and he became quieter. The plantar reflexes were extensor. The patellar, biceps and triceps jerks were uniformly increased. During the fits, the eyes were turned upwards, with nystagmoid movements. His temperature was 103° F. Tepid sponging was ordered and a rectal drip saline started. Three hours later he was more spastic and his legs were crossed at the thighs.]

He died in twenty-three hours after operation, cyanosed, with a temperature of 106° F.

At post-mortem the thymus was found markedly increased, with generalized lymphatic hyperplasia. There were the usual signs of an asphyxial death.

The period of circulatory arrest was about five minutes.

The history of the case and the presence of status lymphaticus suggest to me that the heart stopped from vagal inhibition. It is my opinion that the fibrillation was induced by the injection of adrenaline, and that cardiac massage, first, might have had a more favourable result.

Mechanical stimuli.—Lowering the head of the table is widely practised and may be successful in reviving the patient. Artificial respiration alone may be sufficient to restore the heart. Auricular puncture, if done before the irritability of the auricles has been lost from anoxia, is an adequate stimulus.

CASE III.—Some years ago I anesthetized a girl, aged 17, for tonsillectomy. The surgeon liked chloroform and, as he had given many more anæsthetics than I had myself, I was not disposed to argue. She had atropine, gr. $\frac{1}{6}$. I induced anæsthesia with ethyl chloride and chloroform-ether mixture to the third plane of the third stage and took her into the theatre, where she had chloroform and oxygen through a Davis gag. Shortly after the surgeon had begun I noticed that she was not breathing; she was pulseless and I could not hear the heart. The auricle was pierced with a handy lumbar puncture needle: to my relief I saw it ticking to and fro, the pulse had returned, and breathing started spontaneously. The operation was completed with oxygen and ether, and all was well.

CASE IV.—A healthy man of 52 became obstructed, under pentothal anæsthesia, by an intense laryngeal spasm which I could not overcome with artificial respiration. His face, which was cyanosed, became blotchy white, intra-ocular tension dropped and there was no sign of a heart beat. Immediate auricular puncture restored the heart. Intermittent inflation of his lungs through an oral endotracheal tube restored his

colour, and the pulse gradually returned. The operation—removal of a malignant ulcer of the tongue by diathermy cautery—was completed with nitrous oxide and oxygen. Respiration was still rather depressed at the end but was satisfactorily stimulated by intravenous injection of 2½ c.c. of coramine. He was well the next day.

Ventricular puncture without injection is ineffectual.

Cardiac massage through the intact abdominal wall is a waste of valuable time in adults. Massage through the intact diaphragm, though less effective than direct massage, involves less drastic surgery and is often adequate. Ogilvie (1929) states that only direct massage can maintain the circulation artificially. In one case of his there was no spontaneous heart beat for seventy-five minutes, though the patient remained a good colour, and was breathing, groaning and moving spontaneously.

Gunn and Martin suggest that massage should be carried out at less than half the normal rate to allow filling which, in the absence of auricular contractions, is entirely passive. Compression should be gradual to avoid injury, and relaxation rapid. They say that continuous massage, while helping the circulation and the elimination of anaesthetic, may cause feeble spontaneous beats to disappear again. This may have happened in Ogilvie's case. There should, therefore, be pauses as soon as even feeble beats have begun.

Irritability to mechanical stimuli is soon lost and speed is the essence of the matter.

Where irritability is so far lost that no response is found to direct massage, the violent stimulus of intraventricular adrenaline may still produce results. Massage should be resumed as soon as the injection is completed.

I am indebted to Dr. Nosworthy for an account of the following case:

CASE V.—A soldier, aged 25, was wounded in the chest at point-blank range by a single machine-gun bullet, entering between the 5th and 6th ribs near the sternum. The wound was excised at the C.C.S. On arrival at the Base his general condition was poor, pulse 120, with right hæmothorax. He developed bronchopneumonia on the opposite side and was dyspnoeic, with a pulse of 130. A further exploration was undertaken three days after the injury.

Anæsthesia: endotracheal cyclopropane and oxygen with bronchial suction. There was a hæmatoma of the mediastinum and right hilar region. The damaged upper and middle lobes were removed by tourniquet.

Suddenly, while suturing the hilum, the heart stopped and his colour rapidly deteriorated. The heart was massaged with difficulty through the mediastinum for eight minutes without response. Adrenaline was injected, with almost immediate effect: at the end of twelve minutes the heart was beating satisfactorily. He took a deep breath and the colour flooded back to his face. The operation was completed without further anaesthetic.

At the end of the operation the temperature was 105° F. Left on oxygen, he recovered to the stage of eye movements, but he had gasping respirations. There was no further improvement in two hours and he died within a minute of withdrawal of oxygen. His temperature was 106° F.

Post-mortem findings: Bronchopneumonia of the left lower lobe. Œdema and asphyxial signs in the brain.

Treatment of the resuscitated patient.—After even a short period of cardiac arrest, there is usually a degree of general, but especially of central nervous system depression.

Cortical depression is marked by prolonged coma which, though causative of anxiety, does not require treatment. Any remaining stages of the operation can be completed with little or no anaesthetic. Medullary depression shows itself by a lowered blood-pressure and, more important, by apnoea. It may be necessary to keep up artificial respiration for some little time, seeking relief by cautious injection of respiratory stimulants already mentioned. The patient must be carefully watched until recovery is complete, as any minor accident might prove fatal. Intravenous glucose saline may usefully be given as a circulatory stimulant.

Suggested Routine for Resuscitation

I suggest the following routine measures for resuscitation after sudden heart failure, which differ in one or two important respects from the procedure accepted generally among surgeons.

(1) Clearing of the airway and establishment of artificial respiration with oxygen. Sylvester's method may be used, rhythmical inflation with oxygen from a suitable machine, or rhythmical compression of a closed re-breathing bag. Care must be taken when an endotracheal tube is in use: I have seen, at post-mortem, widespread surgical emphysema from rupture of the lung due to over-enthusiastic insufflation of oxygen.

Gunn and Martin (1915) found, in dogs, where twenty minutes of direct cardiac massage had failed, that intrapericardial injection of adrenaline restored the heart beat almost at once. It was unsuccessful alone. We know that adrenaline can initiate ventricular fibrillation under chloroform and, in sufficiently large doses, under ether. As it acts directly on the ventricles it seems reasonable to suppose that the intraventricular injection of the usual dose (1:1,000 solution, 10-15 M) may produce fibrillation with ether, especially where the tendency to irregularities has been enhanced by a period of anoxia. Tournade and others (1932) reported recovery from chloroform syncope in the dog by intravenous or intracardiac injection of adrenaline combined with massage and artificial respiration. Meek comments "these must have been a simple toxic depression of the heart, a real chloroform syncope". One in ten of these developed fibrillation.

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JOINT DISCUSSION No. 3

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DISCUSSION ON METHODS OF RESUSCITATION IN SHOCK

Dr. R. T. Grant: In comparing the efficacy of different methods we are at once in difficulties because of the confusion in the definition of the word "shock" even when qualified by the adjective "traumatic". In practice there is a wide variation in the application of the term. Some medical officers apply the label "shocked" to any patient, however slightly wounded, who seems ill, is pale and has a weak pulse. Others restrict it to cases displaying low blood-pressure, a rapid thready pulse, pallor and cyanosis, a cold and sweating skin, shallow rapid respirations and often vomiting, restlessness, a lessened sensibility and a dulled mental state. To the majority, the criteria of "shock" are severe injury and low blood-pressure. The lack of a common basis of diagnosis renders it difficult to assess the efficacy of the various methods of treatment adopted. In dealing with air-raid casualties our first job is to pick out the cases which are seriously ill, our second is to recognize those who are likely soon to become seriously ill if left without rapid treatment. We have then to decide on the remedies to be adopted and later to decide when the patient is fit to undergo surgical operation. This sorting of the patients is determined by the nature and extent of their injuries, by the amount of blood lost and, lastly, by the state of the circulation as judged chiefly, but not only, by the level of blood-pressure. The remedies available are the simple measures of rest, warmth and the head-down position, drugs such as morphia, adrenaline and pituitrin, oxygen, the giving of fluids by mouth and rectum, and, finally, the intravenous administration of blood or blood substitutes.

Only too often we are uncertain whether the patient has lost much blood or little; only too often we can merely guess as to the nature and extent of his injuries and what part nervous influences are playing in his collapsed state.

In cases of serious injury with severe loss of blood the simple restorative measures, while they may bring about a temporary improvement, are usually insufficient for recovery. Where there are severe injuries and possibly severe blood loss, transfusion should be begun at an early stage. This does not mean, however, that the simpler measures should be neglected, for they are of value even in serious cases. In milder cases they may be all that is required.

The term rest does not imply that the patient should be kept still. It means making him feel comfortable by transferring him as soon as possible from stretcher to warm bed, removing his clothes, examining his wounds as quickly and as smoothly as the circumstances allow, allaying his fears and his pain, and attending to his wants. I stress these points, for they are often neglected. To allay pain, fear, or apprehension, morphia is specially useful. It is given almost as a routine, and we have not seen bad results follow the injection of $\frac{1}{4}$ gr. We have seen instances in which the giving of morphia has been followed by the subsidence of a raised blood-pressure to normal levels, and others in which a low pressure has risen to normal levels coincident with the relief of pain. It should be remembered, however, that the drug is not well tolerated by some patients and that it may induce or aggravate nausea or vomiting. When the peripheral circulation is greatly reduced, as it often is in these cases, a subcutaneous injection may be but slowly absorbed. A first subcutaneous injection, producing no effect, is followed by a second. If the peripheral circulation meanwhile speeds up, the morphia is more rapidly absorbed. We have seen at least one instance of what we took to be morphia poisoning, probably due to this factor. It should not be forgotten that there are other remedies that may be usefully employed, such as chloral and the bromides by mouth and rectum.

Warmth is also useful in promoting comfort, but heat must be applied with care and moderation. The patient must be watched and the heating reduced or withdrawn when he begins to feel warm. I doubt if it is wise to attempt a rapid warming of the patient and it is certainly not good to leave a cradle on with all its lamps alight for

Insufflation of oxygen through a narrow-bore endotracheal tube is not recommended: the movements of respiration are of valuable assistance in re-establishment of the circulation.

(2) As soon as artificial respiration has been established, auricular puncture should be made with any available needle of $3\frac{1}{2}$ in. or more in length—Hewer's cardiac puncture needle or a lumbar puncture needle—piercing the chest wall in the third right intercostal space $\frac{1}{2}$ in. from the sternum and passing inwards and towards the mid-line. The risks attendant on this are small compared with those of delay.

(3) Should auricular puncture fail to produce an immediate response, cardiac massage must be started through an upper abdominal incision, sterility being secondary to the necessity for speed.

(4) If massage through the diaphragm is not effective within four minutes of the onset of heart failure, a 3 in. incision must be made in the diaphragm and an artificial circulation established by direct massage at a rate of forty compressions a minute.

(5) If a satisfactory artificial circulation is not established, or if spontaneous beats have not appeared in ten minutes, then, and only then, adrenaline 1:1,000, m 10, may be injected into the ventricle through the fourth left intercostal space two inches from the border of the sternum, as a measure of desperation.

(6) It seems a justifiable risk, if the surgeon's hand confirms ventricular fibrillation, to inject into the cavity of the left ventricle 10 c.c. of 2% procaine.

If a satisfactory circulation is not established within four or five minutes, permanent damage to the brain is likely to occur, leading to death at a later period, as in Case II.

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The raising of the foot of the bed is a measure always worth trying in cases where the blood-pressure is low. It is often followed by a more or less rapid rise of blood-pressure and a general improvement in the patient's condition, which, however, is often not maintained. Sometimes the simple measure of raising the end of the bed leads to a remarkably rapid recovery.

In the severely injured patients these measures are usually insufficient to effect lasting improvement, and transfusion is required. Of the 100 cases which Dr. Reeve and I reported recently only 17 did not receive transfusion; none of these was suspected of having lost much blood and in the majority the injuries were not regarded as severe. Moreover, I think that in several of them transfusion ought to have been given and would probably have hastened their recovery.

In assessing the need for transfusion I would place emphasis on the severity of the injury and blood loss rather than on blood-pressure. In severely wounded cases, therefore, with evidence of blood loss, transfusion should be begun without delay, no matter what the blood-pressure is, low, normal, or raised. It must be remembered that a normal blood-pressure and pulse-rate and even a good facial colour are compatible not only with severe injury but also with much loss of blood. Such cases are not uncommon—there were 10 such in the group of 28 with normal blood-pressure in our series. It should also be remembered that low blood-pressure, pallor, sweating, nausea, and other signs of collapse are not necessarily associated with severe injury or blood loss, and that not all cases displaying these signs require transfusion; they may recover well and rapidly without it.

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I have no experience of transfusion fluids other than blood and plasma. If choice of these two is not restricted by supply, I use blood rather than plasma because hæmorrhage seems to play such a large part in producing the depressed state of our patients. Of the 63 cases with low blood-pressure in our series, at least 45, or 85%, were judged to have lost much blood. With small transfusion, not more than 1 or 2 pints, I do not think it matters much whether blood or plasma is used but with larger transfusions more blood than plasma should be given. In any case it is well to observe the changes in the patient's hæmoglobin resulting from transfusion, for it does not seem wise to allow the hæmoglobin level to fall below 50%. It is to be remembered that we have not only to restore blood volume but also to ensure an adequate supply of oxygen to damaged and infected tissues. I have in mind an instance where the femoral artery was cut, and the foot though not entirely devoid of collateral circulation became gangrenous. I think it likely that the loss of the foot can be attributed to the low hæmoglobin content of the blood, which for days remained about 45%.

The amount required for resuscitation varies so greatly in different patients that no definite ruling can be given on this point. Of the 83 cases in our series 23 received 2 pints or less, 29 from 2 to 4 pints, 18 from 4 to 6 pints, and 13 more than 6 pints. The largest amount was 13 pints given over a period of twenty-eight hours, and this was called for because of continued bleeding. In general the amounts are related to the degree of blood loss, the larger transfusion being given mainly where bleeding was renewed either before or at operation. But as a guide we may say that restoration and maintenance of normal systolic blood-pressure (100 mm.Hg and over) can be taken as the chief indication that an adequate amount has been transfused. If there is doubt about how much fluid should be given it is a good plan, if improvement has begun and continues, to cut down the rate of transfusion to see if improvement is maintained.

As regards the rate of transfusion, we have found that for early transfusions a rate of 1 pint in a quarter to half an hour is satisfactory, and for later transfusions a slower rate suffices—one pint over one or more hours, except in instances where these later transfusions are required to compensate for renewed bleeding. It is to be remembered that in some cases when the veins are tightly constricted the transfusion fluid will not run freely into the veins and must be given under pressure, either by means of a bulb attached to the bottle, or by a two-way syringe.

More rapid transfusions have been given, apparently with good effect; for example, in one case two pints were administered in nineteen minutes with general improvement and a rise of blood-pressure from 40/20 to 130/70 mm.Hg. But the rise of pressure accompanying a rapid transfusion is not always maintained, whereas with a slower injection of fluid the rise of blood-pressure is more gradual, but is sustained. Further,

rapid transfusion is not always well tolerated; in one case restlessness developed during the transfusion of one pint of plasma in five minutes, and one pint of blood in eight minutes; in two others restlessness and increased rate and depth of respiration were noted to come and go with the speeding and slowing of the transfusion. On the other hand a slow transfusion may be ineffective, the blood-pressure remaining low or continuing to fall until the rate is increased.

In most cases improvement is pronounced within a few hours of beginning transfusion, being shown by a lessening of pallor (sometimes restoration of a good colour), the cessation of sweating, a feeling of warmth, and a rise of blood-pressure to over 100 mm.Hg; slow pulses tend to increase and rapid ones to fall towards normal rates. In some the improvement is dramatic in its rapidity. In a few the response is slow; thus in one instance transfusion of two pints of plasma in the early stages raised the systolic blood-pressure from 50 to 90 mm. but no further, though recovery took place later. This case also illustrates that one factor which slows improvement is delay in beginning transfusion. The patient was not admitted to hospital until five and half hours after injury; transfusion was begun seven and a quarter hours after injury. Blood-pressure was not established at normal levels until some time after operation—about thirty-nine hours after injury.

As a general rule, in the absence of continued bleeding, if no material improvement takes place after giving two or three pints of blood or plasma at an adequate rate, later improvement is unlikely. Amongst our series there were only 6 patients who failed to respond after transfusion of amounts varying from two to six pints and all soon died; in 3 bleeding was uncontrollable; this factor was not evident in the 3 others.

According to our experience, operation should be carried out as soon as possible after the patient is revived. At operation as little as possible should be done as quickly as possible, and blood loss must be kept minimal. The patient requires to be watched for signs of collapse, and transfusion ought to be continued at drip rate during operation so that at any time it can be speeded up to compensate for further hæmorrhage.

Mr. R. Vaughan Hudson: The experience of major surgery in peace time demonstrated that the use of posture, avoidance of movement, warmth; relief of pain and fluids by mouth, although of greatest value, were not always in themselves sufficient to combat shock, or to prevent its occurrence. On searching for additional aids, it was soon found that intravenous salines were of temporary value only, and it was not until whole blood was used that definite improvement occurred.

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The anæsthetist has played his part. The occurrence of shock on the operating table was manifested to him as a result of trauma, and he noticed a change in colour, a change in respiratory rate and a fall in body temperature. Later the interest and energy of the anæsthetist, making use of continuous blood-pressure readings, demonstrated that the progressive change in shock was the fall in blood-pressure preceding or associated with a rise in the pulse-rate. This use of the blood-pressure apparatus enabled us early to combat shock soon after its commencement, but in fact the surgeon was still waiting for shock to occur before he treated it.

Thus the results did not markedly improve until an attempt was made to anticipate shock, so that the employment of posture, avoidance of movement and use of warmth, were supplemented by whole blood transfusion, commenced in the anæsthetic room, maintained during the operation and post-operatively. In the road casualty these methods were employed as soon as the patient was admitted to the ward, in order to control and combat shock.

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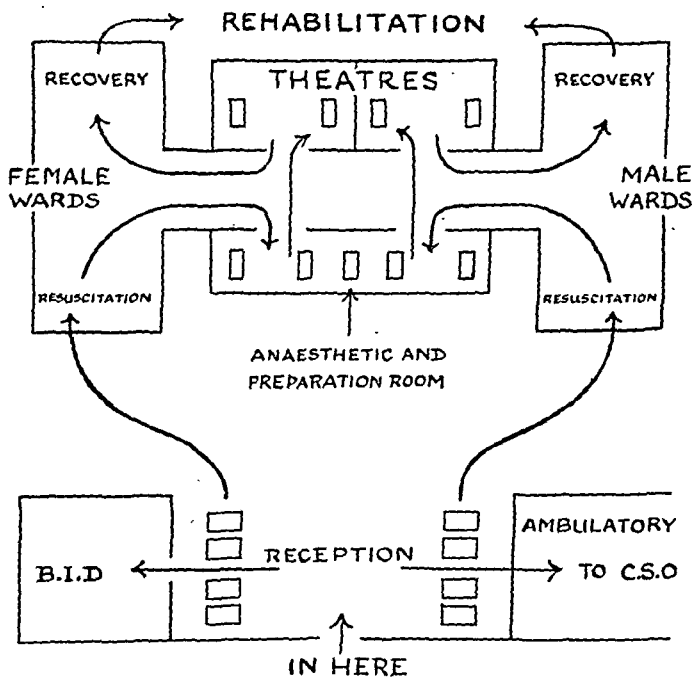
The civilian war casualty has added two main items which were not seen in peace time:

the incredible dirtiness of the casualty due to debris, and the condition known as "blast", the effect of which depends upon the proximity of the patient to the explosion. As a consequence the casualty exhibits a degree of shock which is more profound than that seen in peace time, because the total area of injury is greater although its severity is not necessarily evident upon the surface, but concealed within the ruptured deeper tissues.

Certain general conclusions based upon experience and reasonable conjecture may be drawn as a working basis. Shock inevitably accompanies an injury, it is the expression of an injury, the response to injury, the injury being the causative agent. The fundamental changes are manifested by the cortical centres and the vegetative nervous system.

The cortex expresses either undue exaltation, or confusion and depression, either of which may accompany the changes in what may be termed the automatic nervous system, which in its turn exhibits a prodromal, an intermediate, and a conclusive phase.

These phases may succeed one another with appalling rapidity. In the prodromal period the compensatory mechanism of the nervous system exhibits its conservatism by a slow pulse and normal blood-pressure. This is the dangerous period, and is frequently



Organization and method of dealing with air-raid casualties at the Middlesex Hospital.

[B.I.D.=Brought in dead. C.S.O.=Casualty Surgical Officer.]

ignored or unrecognized. The intermediate stage is the early failure of the compensatory mechanism, in which the pulse may be slow, but the blood-pressure falling. This stage is the inauguration of circulatory failure. In the conclusive stage the mechanism has failed, the pulse-rate is rising, the blood-pressure low and circulatory failure established.

Shock, therefore, appears to be a disturbance of the circulatory mechanism.

Clinically all these phases are accompanied by coldness, pallor of the whole body, peripheral spasm of arteries and veins, and tonelessness of the tissues.

Recovery passes through these phases, but in the reverse order, though the pulse-rate may still be raised in spite of the fact that the blood-pressure is within normal limits. This condition of shock can be mimicked by any cause of circulatory failure, originating in the cortical mechanism, the vegetative mechanism or organic lesions of the circulatory mechanism itself.

The diagnosis rests upon the clinical signs and aid of the blood-pressure apparatus.

The treatment is directed to the mechanism of the patient as a whole, and the nature of the injury.

Although whole blood is considered by us to be the best anti-shock measure, we have relegated its use in war time to exsanguinated patients and employed the more practical plasma, and, in a few cases, serum. These substitutes have been highly efficacious.

The treatment of the wound is of urgency to prevent and control infection. By the removal of dead and dying tissue and their products, by relieving hypertension and correcting hypotension of the damaged tissue, by immobilization of, or resting the injured area, we are preventing further injury and minimizing absorption of the products of damaged tissue. In fact we are stabilizing the patient, relieving pain and preventing further shock.

In the past treatment was focused upon the wound and the general condition of the patient ignored, a phase then occurred in which too much attention was paid to the general condition of the patient and the wound relegated to the background. Now we appreciate that both patient and wound must have simultaneous treatment.

Organization and training in reception and dealing with the injured are of the greatest importance. The personnel, the accommodation, the equipment are kept ready for immediate work. All concerned understand the definite plan, where the equipment is kept and how to use it.

In the Resuscitation Ward a plasma drip is put up on all patients with wounds. In addition it is put up on all those who are considered to be likely to need or benefit by it. Its rate and its quantity is controlled by the blood-pressure reading. As soon as possible the patient is stripped and examined from head to foot and his theatre priority denoted.

In the Anaesthetic Room all preparation of the skin is performed under gas and oxygen anaesthesia preceded by a minimal dose of pentothal injected into the "drip" tubing.

In the theatre foreign bodies are removed and the wound widely decompressed, but dead and dying tissues are sparingly removed.

In glass wounds primary suture is often possible but irregular wounds caused by foreign bodies are left open and packed lightly with vaseline gauze and sulphanilamide.

Wherever possible plaster slabs are made use of for immobilization.

In the recovery ward the strictest attention is paid to the fluid intake and output with special reference to the urinary flow.

In burns the routine differs little, but we cut down, if necessary, through burnt tissue to establish the plasma drip, and we have found of value the Stannard sheeting and envelopes in the treatment of these cases.

The onus of decision as to operation, resuscitation, transference to the theatre and advice in the theatre, rests with the most experienced surgeon present, who should walk and keep walking, continually collaborating with his fellow physician in order to carry through without delay the treatment of the injured. The value of the help of a physician, particularly in thoracic and cardiovascular problems cannot be over-emphasized.

We believe that the policy of preparation and organization for immediate surgery has benefited our patients. The teams of apprentice surgeons, nurses and dressers have borne the burden of the actual work, loyally supported by lay helpers. The following are the results of these labours:

Of the total cases admitted 4% died without surgical intervention. This small group consisted in the main of the crush syndrome and severe fracture of the skull. Of the remainder, consisting of 96% of all cases admitted, the final mortality was 3.8%.

We have been fortunate to work in a soundly constructed building, which, so far, has not been too severely damaged, although we had to make use of supplementary heating, water and lighting arrangements on more than one occasion.

Dr. J. McMichael: In order to have a satisfactory conception of the type of case which we are attempting to treat, I think it is necessary to try to systematize the facts so patiently collected by Grant and others into some scheme which reveals more clearly the various clinical types of shock. The reactions to injury which we can now recognize are shown in the following table:

CLINICAL TYPES OF "SHOCK".

| | | |
|-------------------------|-----|------------------------------------------------|
| Effects of injury | (1) | Fright (emotional "shock"). |
| | (2) | Post-traumatic hypertension. |
| | (3) | Neurogenic shock (pulse slow). |
| | (a) | Vasovagal. |
| | (b) | Brain injury. |
| | (4) | Oligæmic shock. |
| | (a) | Blood loss. |
| | (b) | Plasma loss. |
| | (c) | Blood and plasma loss. |
| | (5) | Crush syndrome (renal failure). |
| | (6) | Circulatory collapse due to mechanical causes. |
| | (a) | Fat embolism. |

| | | |
|-----------------|-----|--------------------------------------------|
| Medical factors | { | (b) Pulmonary embolism. |
| | | (c) Coronary thrombosis. |
| | (7) | Toxaemic shock. |
| | (8) | Bacterial toxins: Diffuse peritonitis, &c. |
| | | Dehydration collapse. |
| | | Gastro-enteritis. ? diabetic coma, &c. |

This table includes a considerable range of conditions, the least severe of which is simple emotional "shock" or collapse associated with nothing more than the distress occasioned by the experience of an accident or bomb incident. It is now well recognized that renal failure may be a remote effect of crushing injury, and it appears that the "condition of lowered vitality due to injury" may result from changes in organs outside the circulatory system. It seems, therefore, that we must admit the wisdom of the definition of shock in the older textbooks as "a condition of depressed vitality due to injury", and we must include under the heading of "The remote effects of injury" such conditions as renal failure. We must also recognize such conditions as *post-traumatic hypertension*, which may be associated with pallor and a rather thin thready pulse; this condition was described by Grant and Reeve in their recent analysis. *Neurogenic shock* includes those cases in which the pulse is slow. This may be a simple vaso-vagal type of faint, although rather prolonged, or it may occur in the more serious condition of brain injury. In either case the treatment consists of rest in a recumbent position. The work of Wallace and Sharpey-Schafer has shown that the vaso-vagal type of collapse is a frequent sequel of hæmorrhage where the blood loss is in the neighbourhood of 1-1.2 litres. Vaso-vagal collapse, therefore, does not exclude the possibility that there may be a considerable reduction of blood volume and these cases have to be very carefully watched. *Oligæmic shock* may result from hæmorrhage, from loss of plasma fluid from the circulation as in burns and crushing injuries, and occasionally these two factors may be combined. Mechanical causes, such as fat embolism, may also complicate the picture.

At the other end of the scale there are the various medical factors which may also produce circulatory collapse, such as massive pulmonary embolism and coronary thrombosis; the latter condition we have seen in an elderly patient who also had oligæmic shock. Presumably the slow blood flow occasioned by blood loss is the factor determining the development of thrombosis on previously diseased coronary arteries. *Toxaemic shock* is a term which we can apply to the collapse which follows sudden flooding of a serous cavity with infected material, as, for example, by a ruptured appendix. *Dehydration* may also play a part in the production of collapse, as, for example, in prolonged diarrhœa or vomiting.

Thus the word "shock" when unqualified has no well-defined meaning, and I suggest that we should always qualify the word in the above manner to indicate clearly the type of case under discussion. The remainder of my remarks will be concerned with the treatment of "oligæmic shock".

I intend to emphasize the more important points in the practical management of cases of wound shock by discussing the mistakes made in the management of some of our earlier cases in the light of advancing knowledge.

A woman of 56 (see *Edinburgh M. J.*, 1941, 48, 160, fig. 3) was our first air-raid casualty. She had sustained multiple injuries of which the most serious was a compound fracture of both bones of the left leg, by which she had been pinned under a fallen beam for many hours. On admission she was cold and collapsed, blood-pressure being under 70 mm.Hg. A serum transfusion of 1,620 c.c. was given in fifteen minutes, with an excellent immediate result, the blood-pressure rising from 70 to 140 mm.Hg. Speed of transfusion is important. The normal heart pumps four litres of blood per minute at rest, and an extra 100-200 c.c. per minute imposes no appreciable difficulty. In oligæmic shock, therefore, we have confidence in giving transfusions as rapidly as they can be made to run until the blood-pressure is restored to normal; only then should the transfusion be slowed to a drip. In this case a mistake was made in dismantling the apparatus after the blood-pressure had been restored and missing the optimum time for operation. Two hours later the patient had collapsed again, and in spite of further massive transfusion (1,440 c.c.) the blood-pressure response was much less satisfactory. The deleterious effect of delay is thus demonstrated. A further fault was to allow the patient to go to the operating theatre where she was subjected to extensive operative procedures without further transfusion; on return to the ward the pulse was imperceptible, the blood-pressure 40 mm.Hg, and a further blood transfusion could not be given sufficiently rapidly to restore the blood-pressure, which only rose to 80 mm.Hg after 1,620 c.c. blood. After three hours it was apparent that further efforts were useless and the patient died shortly afterwards. It is thus important to realize the optimum

time for operation is the earliest moment after restoration of the circulation, the more prolonged the collapse the more difficult it may be to get an adequate effect from transfusion alone, and partial restoration of the blood-pressure is wholly inadequate to save life. When the blood-pressure has been too low for too long, the circulatory collapse becomes irreversible. There may be difficulty in getting blood to flow when collapse is extreme, as the superficial veins seem to go into spasm. Useful measures in such cases are warming of the limb along the line of the vein and ensuring that the blood is warm as it runs in.

When the blood-pressure is low as a result of oligæmia, the critical level at which transfusion is necessary seems to be about 80-90 mm.Hg. Our belief that this is the critical level is based on records of two cases in which transfusion was given and in which the transfusion was stopped when the blood-pressure had reached 85 or 90 mm. Hg. The cases were left with blood-pressures at this level for four or five hours. Collapse occurred at the end of this time, and in spite of further transfusions, death occurred. In one patient who had had a severe hæmatemesis I measured the cardiac output by the acetylene method when the blood-pressure was 90 mm.Hg. The figure obtained indicated a heart output less than half the normal and therefore the oxygen supply to the tissues in such a case was in serious jeopardy. I regard this level of blood-pressure as dangerous only in cases of oligæmic shock. Where circulatory collapse has resulted from other causes, for example coronary thrombosis, and in many cases of post-operative shock where the condition is complicated by the effect of anæsthetics, recovery can occur perfectly well from blood-pressures which are lower than 80 mm.Hg. The reason for this difference is obviously a matter requiring further research.

A second case (*Edinburgh M. J.*, 1941, 48, 160, fig. 2) was that of an Army officer who had sustained punctured wounds of an arm and a leg together with a compound fracture. Subsequent operation showed considerable blood loss into the lacerated soft tissues. On admission his blood-pressure was 110 mm.Hg and he was cold. He was warmed under a shock cage with considerable zeal. An hour later he was found to be hot and perspiring, while the blood-pressure had fallen to 70 mm.Hg. He responded well to transfusion which was maintained through the operation, and his subsequent course to recovery was uninterrupted. I think a major error was overheating. It should be remembered that the warm skin holds at least 500 c.c. more blood than the cold, and when blood is scarce in vital organs, overheating is certainly undesirable. Blalock and Mason have recently shown that dogs bled to a blood-pressure of 75 mm.Hg survive about four times as long when cooled as they do when warmed. Results would improve if heating was not carried out with such enthusiasm. Adequate cover for comfort is probably all that is required.

Pulse-rate.—It has been repeatedly stressed that pulse-rate is a poor guide in the assessment of a case. In the instance of a small boy (*Lancet*, 1940 (ii), 774, fig. 9) whose right kidney had been crushed by a lorry wheel, transfusion and restoration of the blood-pressure were accompanied by acceleration of the pulse from 130 to 180 per minute. Two days elapsed before the pulse subsided to a normal rate. In spite of this, the patient made an uninterrupted recovery.

The volume of transfusion required.—Blood loss slightly over a litre is well tolerated (Wallace and Sharpey-Schafer). We have seen and measured directly blood loss of over two litres and, if our methods are correct, circulating blood volumes under two litres are not uncommon in severe shock. We therefore consider that transfusions of two litres or more are usually essential if the blood-pressure is 60 mm.Hg or below.

Morphine may conceivably be harmful in cases of severe oligæmic shock. It has been our experience that patients who are severely collapsed do not complain of pain, but as the blood-pressure recovers the patient tends to become more restless and pain perception is increased. We therefore do not give morphine in the stage of profound collapse, but as the patient recovers and pain is complained of, morphine is given in ordinary therapeutic doses.

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 GRANT, R. T., and REEVE, E. B. (1941), *Brit. M. J.* (ii), 293.
 WALLACE, J., and SHARPEY-SCHAFER, E. P. (1941), *Lancet* (ii), 393.

Dr. M. D. Nosworthy: It is now agreed that it is seldom wise to operate upon a patient while he is in shock; there being no doubt that effective resuscitation will convert such a patient from being a bad operative risk into a fair one. In fact, first-class resuscitation has given excellent results in spite of second-class surgery and third-class anæsthesia. There is some evidence, however, that, when a limb is so mangled that it will have to be amputated sometime, unless resuscitation produces an immediate and dramatic improve-

| | | |
|-----------------|-----|--------------------------------------------|
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| | | (c) Coronary thrombosis. |
| | (7) | Toxaemic shock. |
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from the ground. This applies also to patients with chest injuries; if such a patient is more comfortable and less dyspnoic when propped up, then his head and shoulders can be raised while the foot of the bed is still blocked. During operation the table should be tilted into a slight Trendelenburg position. Hot drinks may be given with advantage right up to the time of operation except when laparotomy is to be performed.

The blood-pressure level is often a most unreliable guide to the patient's true condition. Patients can pass into the shocked state in a variety of ways. Not all, by any means, show the progressive fall in blood-pressure with increasing pulse-rate which is classically described. For example, the circulation in some way may quickly collapse, the pulse becoming impalpable and the blood-pressure unrecordable. This condition is often seen when a patient loses a considerable amount of blood rapidly—as for instance, when the surgeon during a nephrectomy drops the renal pedicle. As a rule, the blood-pressure soon climbs up again, even without treatment. When, however, the loss has been excessive or recovery has been too slow, the vicious circle of shock gradually becomes established. If injured patients are admitted to hospital in the collapsed state their condition is such that they are likely to be treated at once, and energetically, by the most inexperienced. Although, as I have said, many would pick up on their own, the group as a whole are probably more fortunate than some others who may not be treated seriously until the shock cycle is already well under way. Other patients, who have lost perhaps two pints of blood more slowly, may show no fall in blood-pressure and no increase in pulse-rate. But if patients, who—by their own defence mechanism only—have maintained or regained their blood-pressure in spite of considerable hæmorrhage, sustain further blood loss, they are likely to pass straight into “shock”—the fall in blood-pressure sometimes being almost a terminal event.

After a patient has been resuscitated by intravenous therapy prior to operation, it is a wise precaution to keep the drip running if the nature of the proposed surgery is likely to be severe, because it may be impossible to resuscitate the patient a second time if shock is permitted to return.

Blood and plasma transfusions are so easily obtainable nowadays that they are frequently given when they are not essential. When I judge, however, that the particular patient's powers of compensation are being, or are likely to be, overtaxed by blood loss or trauma, I always give intravenous therapy irrespective of the blood-pressure readings. The vicious circle may then be stopped before it has gathered momentum. I have observed on several occasions during operation deterioration and subsequent improvement in the feel of the pulse without change in either the systolic or diastolic pressures, or change in the pulse-rate. I cannot explain this finding, but I think that it may be of some importance since this change in the feel of the pulse has coincided with my appreciation of a change in the patient's condition on other grounds.

When it has been decided to give intravenous therapy, it will be found that—even if blood is ultimately to be used—it is better to start with plasma which runs much more readily into the veins of a shocked patient. Rapid resuscitation has two great advantages—it may relieve tissue anoxia before irreversible capillary damage has taken place, and it may also permit the patient to be operated upon within the “safe” period. The rates of flow and the amounts of transfusion have already been discussed by Dr. Grant. It is obvious that a patient who has recently lost much blood may safely be given a larger transfusion at a quicker rate than one who has lost little. When the rate of improvement, however, becomes disproportionately slow to the quantity of fluid being introduced, I think it may be wiser to discontinue intravenous therapy even though the plateau reached by the blood-pressure is still on the low side.

With regard to the critical blood-pressure level about which Dr. McMichael spoke I think the pulse pressure is important. I should be much happier about a patient with a pressure of 80/50 than with one of 90/75 mm.

Much has been written by laboratory workers on the effects of different anæsthetic agents in shock. Their conclusions, however, are often misleading since they do not always possess the necessary clinical experience to avoid confusing drug effects with shock. Furthermore, the condition of low blood-pressure which they produce in their experiments does not necessarily reproduce all the changes which occur in shock. It would appear reasonable to suppose that anæsthetic techniques which increase the area of the vascular bed would be harmful, and likewise those during the administration of which the alveolar oxygen tension is low. The deleterious effects of one agent at a certain depth, however, may not be operative at a lighter plane of anæsthesia, so that this agent, although bad for one type of operation, may be satisfactory for another. Again, there are probably several initiating factors in shock, and a different factor may be the chief one in different cases. Anæsthetic agents may produce different results depending upon which factor is uppermost in the particular case, and also upon the degree of shock present.

ment in the patient's condition the operation is better performed at once while the intravenous therapy is continued.

When immediate anaesthesia is called for, the lightest possible plane of narcosis—compatible with the nature of the operation—must be employed. This degree of anaesthesia will be obtained with a very much smaller concentration of *any* anaesthetic agent than would be required for the same individual in normal health. The concentration must also be built up very slowly in order to avoid overdosage which would be likely to result in the immediate or ultimate death of the patient.

Clinical experience is, at present, the best guide as to what resuscitative measures should be taken, and is essential for the correct interpretation of scientific data. This clinical judgment will be more quickly gained by personally following patients all through their illness, than by treating a far greater number merely during the pre-operative phase. The anaesthetist is in the best position to determine what supportive measures should be adopted during the operation itself. His responsibility, however, should not begin and end in the theatre; his opinion is of value in the resuscitative and post-operative phases as well. I am, therefore, doubly in favour of the resuscitation and recovery wards being adjacent to the theatre. Continuity of treatment by the same individual is, I believe, very important in the interests of the shocked patient.

It is obvious that in the case of a young soldier who was hungry, dehydrated, cold, wet, tired and very possibly frightened before he was wounded, there will be more response to simple treatments like hot drinks, warmth, rest and reassurance than there would be in the case of an older civilian whose injury alone—without any predisposing or contributory factors—has produced an identical degree of shock. I am going to confine my remarks on resuscitation to points which I think should be stressed by anaesthetists.

Morphine is often prescribed empirically. Pain, of course, must be relieved; patients with the most extensive injuries, however, are often in no pain, and yet they are well-meaningly given large and repeated injections of morphine. I do not believe that morphine has any specific anti-shock value. So long as the patient is in no pain and is at rest the advisability of giving sedatives is, in fact, doubtful. The use of morphine in such a case is likely to increase any anoxia present since the undesirable results of respiratory depression more than offset any decrease in metabolic rate. Frail patients have been admitted to hospital having had as much as one grain of morphine, and have really died of morphine overdosage. The dose should be individualized. Half a grain is the standard dose at some first-aid posts, and this is too much for many patients. An adequate sedative effect can be obtained, without the same respiratory depression, by giving an injection of scopolamine with a much smaller dose of morphine. Scopolamine, in contrast to morphine, helps to allay fear, and it is common knowledge—to anaesthetists anyway—that anxiety makes a patient much more susceptible to shock-producing factors. Reassurance of the patient is an important point which is usually neglected. Since the actual time of operation is often doubtful it is better to omit premedication; the anaesthetist can then give an intravenous injection of whatever morphine and scopolamine he may think fit in the theatre.

Restlessness is a common sign of oxygen want. Since other methods of resuscitation are always used concurrently the exact value of oxygen therapy in shock is difficult to assess. I have personally, however, so often seen during operations incipient circulatory depression relieved by an increase in the alveolar oxygen tension, that I believe that efficient oxygen therapy may be invaluable in breaking the vicious circle which develops in shock. It is, after all, anoxia that finally kills the shocked patient. Oxygen therapy offsets to some extent the disadvantages of heavy morphine sedation. When restlessness, unassociated with pain, persists in spite of resuscitation, it is better controlled, in my opinion, by intravenous paraldehyde than by morphine. Enough has been written already on how to administer oxygen. All I will say is that the earlier efficient oxygen therapy is started the greater and quicker will be the benefit therefrom. After starting with a high concentration in order to ascertain how great an improvement in the patient's condition can be obtained with oxygen, the alveolar tension should then be lowered little by little—provided the maximum benefit is still maintained—until atmospheric conditions have finally been restored. This procedure should also be followed during anaesthesia. When a high tension of oxygen has had to be used all through an operation, oxygen therapy should be fixed up in the theatre and continued in the recovery ward, until such time as a reduction in oxygen tension is no longer followed by an increase in pulse-rate. Economy in oxygen consumption is obviously desirable in these days, but it is not only for this reason that the minimum increase in oxygen tension which produces the maximum benefit is advised.

The importance of moving a shocked patient as little and as gently as possible cannot be over-emphasized. The foot of the bed should be raised a good eighteen inches

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There seems to be no general agreement on the definition of shock. What one observer calls shock, another will not. Until we are all talking about the same entity, or distinguishing between different conditions, which ultimately present much the same clinical picture, we are attempting treatment before making an accurate diagnosis. If I were asked what anæsthetic technique I would use for thyroid surgery, I should first find out what was the matter with the patient. Thyroid operations are performed for various conditions, and each condition—apart from its individual variations—presents a different anæsthetic problem, and, in my opinion, each calls for a different method of approach. I believe much the same applies to shock. I want to know what has set the vicious circle of capillary atony in motion, and how far the process has progressed; whether the shock cycle is now, so to speak, self-generating, or whether initiating factors are still operating. In my view, we must first—making use of every means at our disposal—analyse cases and try to decide what factors are at work, and which is the predominant one in a given case—whether it be straight blood or fluid loss, the effects of trauma or afferent stimulation, exposure or anxiety, asphyxia or other factor. We also badly need criteria by which different degrees of shock development can be uniformly recognized. Then, in time, we may be in a position to say which is the best anæsthetic technique—and incidentally, the best method of resuscitation too—for a particular type of case.

Here is the anæsthesia record of one patient which may help to emphasize a few points.

A nervous man of 38 underwent a partial gastrectomy one morning in 1933. During the operation, which took just under two hours to complete, nothing of note occurred. The patient sustained little blood loss, was subjected to the minimum of trauma, and required no supportive treatment. At 11 o'clock, after the anæsthetic had been stopped, his blood-pressure was 150/100 and his pulse-rate 72. During the course of the afternoon he vomited bright blood on three occasions, the total quantity of blood lost being estimated at between 2½ and 3 pints. Treatment consisted of rectal salines and three injections of morphine gr. ½. At 5.30 he was taken back to the theatre for exploration, and I was called to anæsthetize him. At that time he had a pulse-rate of 140 and a blood-pressure of 70/50. The patient was transferred from his bed to the operating table, and this handling made his blood-pressure become unrecordable and increased his pulse-rate to 170. I gave him 100% oxygen to breathe, and by the time the blood transfusion had been set up—and before any benefit could have been derived from it—his pulse-rate had dropped to 130 and his blood-pressure had risen to 90/70. Of course, he had had time to rally from the effects of being moved, but since his pulse-rate was now lower and his blood-pressure higher than they had been in bed I think the oxygen was in no small measure responsible for his improvement.

At this point, making use of the carbon dioxide absorption technique—which several of my correspondents have also found beneficial for shocked patients—I anæsthetized him with cyclopropane and oxygen. The anastomosis was unpicked and resutured in thirty-five minutes and, as you can see, the improvement in his condition was maintained. To guard against possible restlessness I kept him anæsthetized for a further twenty-seven minutes, until he had received two pints of blood. During the last half-hour I was able steadily to lower his alveolar oxygen tension, until, when the anæsthetic was discontinued, he was breathing air, was awake, and had a pulse-rate of 104 and a blood-pressure of 106/70. His convalescence was then straightforward.

During the six and a half hour interval between this man's two appearances in the theatre I have no data about his condition. It happens too often that promising material like this is wasted from the research point of view owing to lack of accurate detailed observations.

Although this man displayed all the classical signs of shock, his rapid recovery was not like that of a truly shocked patient. Even if the same vicious circle eventually becomes established in both, I wonder whether a case of external hæmorrhage pursues the same course as a case of shock from other causes. There is no doubt that the effects of blood loss, if treated in time, can be largely removed by oxygen therapy and blood transfusion. Therefore, cases in which external hæmorrhage is the chief factor are comparatively easy to treat. In other cases, resuscitation has to overcome factors which are still militating against it. Some factors can be overcome in time, others cannot. Others, again, can only be eradicated by surgery. The benefit likely to be derived from operation, however, must be weighed against the risk of exposing the patient to further hæmorrhage, fresh trauma, and—unless anæsthesia is skilfully administered—also to the shock and asphyxia associated with a bad anæsthetic.

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Section of Physical Medicine

President—MAJOR G. D. KERSLEY, R.A.M.C.

[February 20, 1942]

Psychological Aspects of Rheumatoid Arthritis

By JAMES L. HALLIDAY, M.D.

THIS paper describes the results obtained by applying a psychological approach to a series of 20 unselected patients with rheumatoid arthritis—14 females and 6 males.

A. PSYCHOLOGICAL CHARACTERISTICS OF PERSONS WHO DEVELOP RHEUMATOID ARTHRITIS

(In collaboration with K. M. Abenheimer)

(i) On Looking at the Patient

The facies.—Textbooks describe in detail the appearance of the joints but are unanimously silent on the appearance of the patient as a person. Yet even a superficial observer on entering a waiting-room occupied solely by patients with rheumatoid arthritis, would probably receive an impression that they were on the whole, "a quiet, decent-looking lot". Possibly also, he might note that there seemed to be a dominant type of facial expression, but he would find it difficult to describe this in words. In the case of women patients my notes show a prevalence of such terms as "calm; detached; placid; pleasant, 'with little smiles'". In the male patients, the terms varied from "expressionless and blank" to "open and bright". Katz (1931) was so fascinated by the facies of rheumatoid patients that he suggested it could be compared to "a mask face" such as is found in chronic Parkinsonism—but this is doubtful and was not my impression. There is certainly a relative poverty of facial expression but this is probably not more evident than might be expected in the case of persons who habitually hide and repress many aspects of their inner emotional life. Irrespective of its explanation—and there may be a central nervous organic basis—the prevailing facies in rheumatoid arthritis with its calmness, detachment, and lack of exteriorized tension, is in definite contrast to that in many osteo-arthritis where the countenance is anxious, strained, irritable, dazed or depressed, i.e. shows evidence of an anxiety or depressive state. Indeed, one of my medical colleagues considers that the contrast is a point of value in differential diagnosis.

Behaviour and manner.—As regards behaviour, the patients sat calmly and quietly while being questioned and did not fidget or squirm or show obvious "nervous" movements. In the females the manner could often be described as "soft and refined" and in the males there was an unusual absence of truculence amounting almost to gentleness. Towards the examiner the general attitude of both sexes was one of quiet friendliness which, during succeeding interviews, did not vary—an observation made originally by Katz.

Comment.—These descriptions of the prevailing facies and manner in rheumatoid arthritis did not fit exactly in every case, but they were applicable to the majority and would seem to be fairly distinctive for a group of rheumatoids as contrasted with groups of other affections, e.g. peptic ulcer. A question arises here: Are the "typical" facies and behaviour in rheumatoid patients to be regarded as characteristics of the persons before the onset of the disorder, or as symptoms, or features, of a generalized morbid process? In other words, do the facies and behaviour belong to the Personality Type, or to the Disease? As the patients were seen only after the onset of the affection, a dogmatic answer is impossible, but in view of the psychological findings (which follow) it seems likely that the facies and manner are expressions of the personality and may therefore be regarded as characteristics which may, or may not, have become exaggerated during the progress of the rheumatoid illness. Compare the "typical facies" of the peptic ulcer patient which becomes more distinctive after the primary onset and deepens with the chronicity of the disorder.

(ii) On Talking to the Patient

Strength of character.—The patients did not strike me as being particularly "strong" characters—to use this term in its everyday significance. On the other hand, they were not exceptionally "weak characters".

B. THE PRECIPITATING EMOTION

In 9 of the 20 patients in the series, a definite upsetting event anteceded, and seemed to be connected with, the primary emergence of rheumatoid symptoms. External events which precipitated recurrences were noted in 7 cases.

The nature of the emotions associated with onset or recurrence was in no way specific. The emotional disturbances included shock following acute danger (e.g. air raid; assault by a handbag snatcher), anxiety over finance or the misbehaviour of relatives; fears of loss of a love-object or depression after its loss; paranoid resentment concerning superiors; frustration at being jilted, &c. However, irrespective of its nature, all the patients dealt with the precipitating emotion in a common way, namely, there was failure or incapacity to give it a liberating expression and "the feelings" were, so to speak, bottled up. This restriction of feeling and of emotional expression has already been noted as a typical characteristic of rheumatoid patients.

C. OTHER PSYCHOLOGICAL CONSIDERATIONS

(i) Gains from the Illness

There was no evidence that the illness served the purpose of the patient either consciously or unconsciously. After the onset there was no indication that any "secondary gain" (such as compelling others to give attention to the patient) was being aimed at. Also, so far as could be determined, there was, after the onset, no easing of pre-existing psychological tensions in the sense that the arthritis was being experienced as a punishment for, or as a relief from, feelings of guilt.

(ii) Symbolism

There was no evidence that rheumatoid arthritis could be regarded as symbolic in the strict sense of signifying some particular wish, impulse or emotion, and no connexion could be established between the precipitating emotion and the site or sites of the rheumatoid manifestation.

It is admittedly tempting to regard the locomotory limitations of rheumatoid arthritis as a bodily expression of the self-restricting psychological characteristics. Thus Jelliffe (1936) in discussing the role of aggressive impulses in somatic disease suggested that the criminal who expresses his aggressions outwardly is "locked up by others" whereas the arthritic, who expresses his aggressions inwardly, "locks himself up". This, however, is a fanciful conceit.

(iii) The Aetiology of Recovery

Rheumatoid arthritis is one of those affections such as peptic ulcer, mucous colitis, exophthalmic goitre, and asthma, which are characterized by phases of crudescence, subsidence and recurrence. The primary phase may be of varying duration and severity and may subside never to recur; or it may continue in severity and assume a progressive fulminating quality; or it may be followed by a period of quiescence of no standard length to be followed by a recrudescence of no constant duration. In other words, irrespective of any treatment applied, there exists the phenomenon of natural recovery brought about by forces within the patient. Nothing, however, seems to be known of the aetiology of such natural recovery apart from certain observations relating to subsidence during pregnancy or jaundice, yet further study of those patients who recover "on their own" should be highly profitable. In the short series under review, this aspect was unfortunately left largely uninvestigated from the psychological viewpoint and no comments can therefore be made on how far natural recovery is influenced by improvement in the life situation or in the providing of the patient with a new end in view.

A case may be quoted. A young man, deeply attached to a younger sister developed rheumatoid arthritis in February 1940 within a month after learning that his father had a tumour of the lung and within two weeks of a serious accident to his favourite sister. Phases of activity continued until March 1941 when he began attending the clinic for gold treatment and massage. He made a spectacular recovery in April. Yet this recovery coincided with the recovery of his younger sister who was successful in obtaining a situation and also with his own appointment to the position of foreman. It is evident that further research on the aetiology of recovery requires to take full account of the psychological factors involved.

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Moods.—Most of them described their prevailing moods before the onset of the illness as "cheerful, even-tempered and not the kind that worries", but four of the twenty patients had suffered from periodical depressions. After the onset of illness the majority remained "cheery" but a few admitted to having developed fits of depression when they looked at their joints and brooded on their deformity and incapacity. The relative absence of depression in these patients is in striking contrast to its frequent presence in patients with other affections, e.g. mucous colitis (White, Cobb and Jones 1939). The unusual quality of mood in rheumatoids was noted by Katz who depicts both their "euphoria" (as he calls it) and their depressions as "void of affect—without soul—which produces an effect of truly touching patience".

Social adjustment.—All the patients had been fairly well adjusted to social life in relation to career and occupation. None had held any outstanding position or showed outstanding capacities although one, a domineering hysterical woman, was the president of several women's associations in the small town where she lived. As workers, the majority were fairly active, were reliable and conscientious, and had held the same position for a long time. Even after onset they were little inclined to give in, and in spite of pain they carried on working as long as was humanly possible. This disregard for pain as well as their conscientiousness in work seems to be part-expression of a general tendency which may be described as one of self-limitation and restriction of feelings and emotions. These features are more strikingly illustrated in relation to other fields of life, thus:

(a) *Sex.*—Of the 14 female patients 8 professed to be either frigid or "not interested" in sex and their life-histories seem to corroborate this. Of the 6 males 3 were markedly fixed to the mother or sisters.

(b) *Childhood.*—In most cases the tendency of self-restriction had already developed in early childhood. The majority described at least one of their parents as strict or hard and they had to conform to his or her strict discipline. As children these patients were shy and retiring and at school were only moderately successful.

(c) *Later life.*—At the onset of illness 6 of the 14 women were living alone with old or invalid relatives whom they attended and for whom they made considerable sacrifices in money, work, comfort and freedom. They fulfilled these social duties as conscientiously as their business ones. The majority of the patients led a quiet life and were essentially "home birds". They had few friends and, being self-sufficient, they found it difficult to make friends but after establishing the relationship they were loyal to it.

(d) *Habits.*—The patients with perhaps one exception were markedly orderly, punctual, tidy or over-cleanly; that is to say they may be described as showing marked "obsessional trends" (this does not mean they showed full-blown obsessions), as is usual with people who control and restrict their emotions.

(iii) *The Rheumatoid Personality*

The psychological characteristics common to all persons in this series were marked emotional self-restriction (present in all cases and often suggested in the facies and manner) and marked obsessional trends (with perhaps one exception).

This self-limiting, emotion-inhibited, rather independent (but passively so) type of personality is not infrequent and may be found associated with various psychosomatic disorders. As thus crudely defined it cannot be regarded as specially related to rheumatoid arthritis in the way that the "allergic personality" and the "peptic ulcer personality" seem to be related to their respective affections. Indeed, from the common-sense point of view, most of the rheumatoid patients could be described as "normal" in that their self-restricting characteristics neither immediately unfitted them for life nor rendered them acutely unhappy. It remains to be seen whether further research will uncover a more specific rheumatoid personality type.

NOTE.—The patients did not conform to any "psychiatric disease type". Thus—some were markedly hysterical; some were over-scrupulous obsessionals with a tendency to worry; some suffered from phobias. Three of the patients were nail-biters; one had a history of enuresis; and one female patient who had frequent crying fits was on the verge of a melancholic breakdown owing to the absence of any reliable relationship to inner or outer love objects. The unsuitability of such classifications for studies in personality was noted by White, Cobb and Jones (1939) in their study of mucous colitis where they note that "classifications of persons in terms of these psychological reaction patterns fail to indicate personality". Another unsuitable classification is that of Kretschmer which was adopted by Ellman and Mitchell (1936) who examined a series of 50 patients with rheumatoid arthritis and reported that "Schizoid features with marked morbid anxiety predominated in more than half the cases before onset". Kretschmer's schizoid personality would seem to include the self-limiting emotion-inhibited type found in rheumatoid arthritis but the term is, *inter alia*, too vague and too wide to indicate in communicable form, the fairly well-defined and recognizable type of person who develops rheumatoid arthritis.

something to occupy his time. On the contrary, it is to-day generally accepted, that rehabilitation should commence as soon as possible after the patient has been admitted to hospital. In particular this is true of the accident case. The sooner a patient can be brought to realize that his injury, however severe it may be, does not mean the end of everything for him and that he is able, even while lying in bed to undertake some useful work, the more rapid and the more complete is his recovery likely to be. It is important, therefore, that the principle of occupation as soon as possible, should be practised in all hospitals and in particular those which deal with trauma.

Cost of Occupational Therapy

The cost of an occupational therapy department to a hospital, over and above the cost of providing suitable accommodation is not great. The heaviest annual cost is for salaries of the trained occupational therapist and of the assistants which she may require and I would here stress the necessity of putting the department under a really competent person. The average salary of the trained occupational therapist is in the region of £250, and it has been found that one person can handle quite a large number of cases particularly if they can find voluntary helpers. In normal times, the prime cost of the necessary equipment for a hospital of 400 beds, should not exceed £300.

As far as materials are concerned, the most satisfactory method is for the material to be sold to the patients at its cost price. When the patient has bought the material for the article he is making he is likely to put his best work into it if, when it is completed, he is to be the owner. I strongly deprecate any effort to get patients, even though it be part of their treatment, to make articles which are afterwards sold for the benefit of the department or the hospital concerned. Our experience has been, that not only is it easy to persuade the great majority of cases to undertake occupational therapy, but that once they have started, it is most difficult to keep them out of the workrooms.

One cannot, in dealing with occupational therapy ignore the question of vocational training with which it should be closely allied. Up to quite recently, it was nobody's business to be interested in the future of the injured person as far as his re-absorption into industry was concerned. The Ministry of Labour in conjunction with the Ministry of Health has now set up a scheme, whereby they are able to get in touch with the case before discharge from hospital. At the moment the scheme is only working in connexion with the orthopaedic and Fracture A hospitals. Each of these special hospitals is linked up with the Ministry of Labour office in its own area. Before leaving hospital a representative from the Labour Exchange interviews both the man and, if possible, the doctor in charge of the case. In the event of a man being unable to follow his former type of employment because of his residual disability, he is given the opportunity of going to a Ministry of Labour Training Centre where he will be trained for some other occupation more suited to his disability. The admission that it is possible to train many of the physically disabled in the same establishments as the normal individual, is a great step forward and it is to be hoped that its scope will be greatly extended in the post-war period, when the necessity of the greatest use being made of all available man-power to repair the ravages and wastages of war, will be of paramount importance.

Vocational training affects two quite different classes of individuals and there are a few institutions in the country which dealt with this type of training in the pre-war years. One dealt with the adolescent, whose residual disabilities were due to disease (polio-myelitis, spastic paraplegia, tubercle, &c.). In the majority of cases, the individual concerned had never worked, knew nothing of industrial conditions and was often firmly convinced that he, or she, was never likely to have any wage-earning capacity. Of this type of institution, the largest (The Derwen Cripples' Training College, Oswestry), with approximately 220 beds, was founded fourteen years ago, and experience has amply justified its existence. In this type of training school the course must necessarily be long and the period in practice is from three to four years.

The other type of institution, of which the best known is the Cripples' Training College at Leatherhead, deal chiefly with the industrial cripple, that is the cripple who has already been in industry but who, owing to accident, is unable because of this residual disability, to return to his previous employment. The system of training in this type of centre involves a much shorter period of instruction, usually not more than six to nine months, and it is run more on the lines of the Ministry of Labour training schemes. It is the latter type of training which will doubtless receive most attention in the post-war years.

For those cases left with a residual disability which makes their reabsorption into normal employment a difficult problem, the older idea was that every such person should be given a pension, thus leaving employment to the able-bodied. Why the physically disabled should be consigned to idleness and a compulsory early retirement for the benefit of the able-bodied when, given the opportunity he is not only willing but able to earn his own

[March 20, 1942]

DISCUSSION ON OCCUPATIONAL THERAPY

Mr. J. Rhaiadr Jones (Derwen Cripples' Training College, Oswestry), *Hon. Adviser in Rehabilitation to the Minister of Health*: Occupational therapy is one branch of the general scheme for the rehabilitation of the injured. Professor Jefferson and Dr. Riddoch in a memorandum on the subject say: "Rehabilitation is the planned attempt under skilled direction by the use of all available measures, to restore or improve the health, usefulness and happiness of those who have suffered an injury, or are recovering from a disease. Its further object is to return them to the service of the community in the shortest time."

Technically, the work which would be included within this definition of rehabilitation would be: (1) Active movements: (a) Without apparatus; (b) with apparatus (gymnasia and simple games). (2) Massage and electro-therapy: (3) (a) Physical training; (b) organized games—indoor and outdoor. (4) Occupational therapy: (a) Handicrafts at bedside; (b) work out of doors (such as gardening); (c) workshops where handicrafts such as carpentry, weaving, &c., can be practised. (5) Vocational training of those who, because of their residual disability are unable to return to their pre-accident or pre-disease form of employment.

Rehabilitation becomes of the utmost importance in war-time, when it is essential that the patient should be restored to full working capacity as soon as possible.

The main difficulty in the practice of occupational therapy in peace-time is an economic one, and this applies generally to most forms of rehabilitation. In peace-time it is impossible for the average hospital to allow one of its beds to be occupied by a patient until he is able to return direct to his normal occupation. The patient, in order to make room for a more urgent case, has to be discharged to his home, and convalescence becomes a tedious business—he is left to his own devices and the monotony is only broken by periodic visits to the Out-patient Department for general supervision, or for any further treatment. In the case of fractures, this is particularly unfortunate. Patients are put into plaster, kept a few days in hospital and then discharged to their homes, and the time which should be used in maintaining general physical fitness is spent in idleness and boredom.

The economic position of the individual is also of paramount importance in any scheme for rehabilitation. Service cases who are transferred to hospital for treatment suffer no financial loss, their personal allowances and their family allowances do not alter, so that successful treatment is not complicated by financial worry and anxiety. Very different is the position of the non-Service case. Physical misfortune is followed by serious diminution of weekly income, with consequent anxiety as to the well-being of dependants. Why the same reasons which call for the rapid return of the injured to employment in war-time should not apply with equal force to peace-time procedure, is a problem which one feels is bound up with the whole question of the incidence of unemployment, but there can be no justification for a system which eases the unemployment problem by prolonging the period of treatment of the injured.

The Ministry of Health in its attitude towards occupational therapy lays down the principle that it should be primarily remedial and not vocational. In many E.M.S. hospitals workshops have been established where different types of handicrafts can be practised and treatment commences as soon as possible after the injury.

The type of patient to be treated falls into one of two categories: (1) Remedial; (2) psychological and diversional. The remedial case is usually sent by a surgeon with the object of increasing the function of a limb by suitable occupation, and I would suggest that a particular exercise, if allied to production, is likely to prove more efficacious than an active movement where no effort on the part of the brain is involved.

On the stationary bicycle as used in massage departments a patient pedals until he has travelled a certain distance as shown on a clock fixed in front of the machine, or until he has attained to a given speed. In the occupational therapy department, precisely the same result can be obtained by the use of the bicycle fret-saw, on which the patient pedals and, at the same time works a fret-saw which gives both his brain and his hands employment. Many similar instances could be cited but in all, the general idea of production being allied to the necessary remedial endeavour, is the guiding principle.

Occupational therapy has also a definite value from the psychological point of view, and where the facilities are available every case, whether bed or ambulatory, should be given some occupation. Its usefulness in the treatment of all types of cases, in particular the long stay case, cannot be denied. Boredom is not a state of mind which helps either the physician or surgeon to obtain quick results. It is often said that occupational therapy is most useful after a patient is able to get out of bed and needs somewhere to go and

something to occupy his time. On the contrary, it is to-day generally accepted, that rehabilitation should commence as soon as possible after the patient has been admitted to hospital. In particular this is true of the accident case. The sooner a patient can be brought to realize that his injury, however severe it may be, does not mean the end of everything for him and that he is able, even while lying in bed to undertake some useful work, the more rapid and the more complete is his recovery likely to be. It is important, therefore, that the principle of occupation as soon as possible, should be practised in all hospitals and in particular those which deal with trauma.

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The cost of an occupational therapy department to a hospital, over and above the cost of providing suitable accommodation is not great. The heaviest annual cost is for salaries of the trained occupational therapist and of the assistants which she may require and I would here stress the necessity of putting the department under a really competent person. The average salary of the trained occupational therapist is in the region of £250, and it has been found that one person can handle quite a large number of cases particularly if they can find voluntary helpers. In normal times, the prime cost of the necessary equipment for a hospital of 400 beds, should not exceed £300.

As far as materials are concerned, the most satisfactory method is for the material to be sold to the patients at its cost price. When the patient has bought the material for the article he is making he is likely to put his best work into it if, when it is completed, he is to be the owner. I strongly deprecate any effort to get patients, even though it be part of their treatment, to make articles which are afterwards sold for the benefit of the department or the hospital concerned. Our experience has been, that not only is it easy to persuade the great majority of cases to undertake occupational therapy, but that once they have started, it is most difficult to keep them out of the workrooms.

One cannot, in dealing with occupational therapy ignore the question of vocational training with which it should be closely allied. Up to quite recently, it was nobody's business to be interested in the future of the injured person as far as his re-absorption into industry was concerned. The Ministry of Labour in conjunction with the Ministry of Health has now set up a scheme, whereby they are able to get in touch with the case before discharge from hospital. At the moment the scheme is only working in connexion with the orthopaedic and Fracture A hospitals. Each of these special hospitals is linked up with the Ministry of Labour office in its own area. Before leaving hospital a representative from the Labour Exchange interviews both the man and, if possible, the doctor in charge of the case. In the event of a man being unable to follow his former type of employment because of his residual disability, he is given the opportunity of going to a Ministry of Labour Training Centre where he will be trained for some other occupation more suited to his disability. The admission that it is possible to train many of the physically disabled in the same establishments as the normal individual, is a great step forward and it is to be hoped that its scope will be greatly extended in the post-war period, when the necessity of the greatest use being made of all available man-power to repair the ravages and wastages of war, will be of paramount importance.

Vocational training affects two quite different classes of individuals and there are a few institutions in the country which dealt with this type of training in the pre-war years. One dealt with the adolescent, whose residual disabilities were due to disease (polio-myelitis, spastic paraplegia, tubercle, &c.). In the majority of cases, the individual concerned had never worked, knew nothing of industrial conditions and was often firmly convinced that he, or she, was never likely to have any wage-earning capacity. Of this type of institution, the largest (The Derwen Cripples' Training College, Oswestry), with approximately 220 beds, was founded fourteen years ago, and experience has amply justified its existence. In this type of training school the course must necessarily be long and the period in practice is from three to four years.

The other type of institution, of which the best known is the Cripples' Training College at Leatherhead, deal chiefly with the industrial cripple, that is the cripple who has already been in industry but who, owing to accident, is unable because of this residual disability, to return to his previous employment. The system of training in this type of centre involves a much shorter period of instruction, usually not more than six to nine months, and it is run more on the lines of the Ministry of Labour training schemes. It is the latter type of training which will doubtless receive most attention in the post-war years.

For those cases left with a residual disability which makes their reabsorption into normal employment a difficult problem, the older idea was that every such person should be given a pension, thus leaving employment to the able-bodied. Why the physically disabled should be consigned to idleness and a compulsory early retirement for the benefit of the able-bodied when, given the opportunity he is not only willing but able to earn his own

livelihood, is one of those things which one finds it difficult to excuse in a more or less civilized community.

Another suggestion is that there should be reserved occupations, in which only the physically disabled should be employed, or to put it more exactly, trades in which the able-bodied should not be employed if there were a physically disabled person available who could do the work. This seems to be a more reasonable solution than the first, but there are many difficulties in its adoption, not the least of which would be to decide on the degree of physical disablement for entry into the suggested reserved employment. One feels that, if the physically disabled is to be brought to feel that he is not a burden to society, and if he is to lead a normal and happy life, he must be placed in a position where he can have equal opportunity of employment in those trades for which he is best suited.

There are, unfortunately, a relatively small number of residual cases, who could not benefit under this scheme for vocational training, those whose physical disabilities are such as preclude them from ever being able to re-enter normal industrial life. The Derwen Scheme has attempted to deal with this class by the foundation of a Settlement where such cases can remain living and working under sheltered circumstances.

It seems probable that post-war conditions will lead to a complete re-organization of our hospital system, when full consideration should be given to the question of rehabilitation.

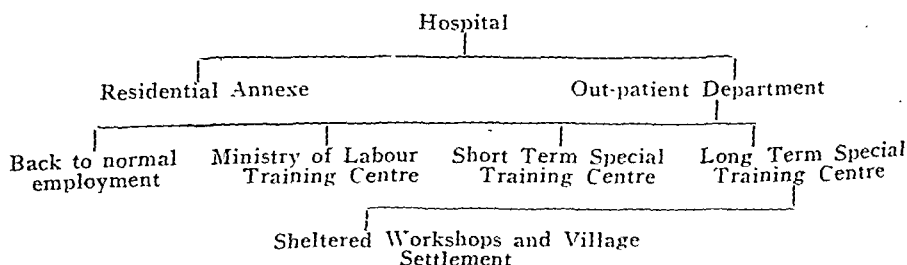
A complete scheme could be organized on the following lines:

Hospital to which the sick or injured is first admitted for the treatment necessary to arrest the disease, or to prevent as far as possible, permanent physical disabilities as a result of accident. The *Out-patient Department* under the control, if possible, of an expert in physical medicine who has at his disposal all those ancillary services including occupational therapy which may be considered necessary for the complete recovery of the patient. The Department should be closely linked with the local Labour Exchange, whose representative would be in a position to give guidance as to the best type of work for which a patient would be suited, in view of his possible residual disability. If the patient was not fit to re-enter his previous employment, he should be admitted from these departments to a Vocational Training Establishment.

Vocational Training Centres

These would be divided into three groups:

- (1) The normal Ministry of Labour Training Centre.
- (2) The Special Short-term Training Centre for those adults who have worked before, but who, because of their residual disability, need more specialized training than can be given in the Ministry of Labour centres.
- (3) (a) The Special Long-term Training Centre mainly for adolescents who have never previously been in employment or those who because of their acute physical disability are never likely to be able to take up employment under normal conditions.
 (b) *Sheltered Workshops*, where those last mentioned can be employed, and which would be associated with Village Settlements on the lines of Papworth, where they could live with their families and lead a more or less normal life.



This, admittedly, is an ideal scheme. It would involve radical amendment of the National Health Insurance Act, the Workmen's Compensation Act and the many other Acts concerned with the problem. Close liaison would be necessary between the various Government departments, the medical profession, the insurance companies, the employers, the trade unions and other bodies concerned, before the scheme could be even moderately successful.

To a large extent its ultimate success would depend on the problem of unemployment being adequately handled in the post-war period. The phrase "fit for light work"

which appears so often on medical certificates is unfortunate. Industry to-day has no room for the semi-fit. Their presence undoubtedly tends to slow down production and, if we are to compel employers to re-employ those injured in their factories and workshops, it seems essential that the injured should be returned in a fit condition to do a normal full day's work.

Miss E. M. Macdonald (Dorset House School of Occupational Therapy): Occupational therapy can be called the science of prescribed and supervised work and recreational activity, prescribed to hasten recovery from disease or injury. Occupational therapists work directly under medical supervision, and on prescription only—these being considered from four aspects: (a) The Physical; (b) the Psychological; (c) the Social; (d) the Economic.

The prescriptions are divided into two categories: (1) Special Occupational Therapy; (2) General Occupational Therapy.

The special application is in direct relation to the injury or disability, whether it be physical or psychological. General Occupational Treatment is also of therapeutic value but concerns, not a particular seat of injury, but the patient as a whole person. This general occupational work is for the cases whose recovery is fairly certain, but whose attitude to the disability, and to the course of recovery, is uncertain, and whose general physical fitness is poor. There is yet another use for it: as will be mentioned later when describing some recent cases, the general occupational treatment, given perhaps on the wards to patients in bed, may be the introduction to future possibilities in the workshop and so make interest in recovery and transition to special treatment, more easily carried through.

It is difficult to give anything but approximate figures at this early stage in a new experiment, but it has been suggested that there are not more than 10% to 15% of specially remedial cases in a hospital such as the Fracture "A" or special hospital under the Emergency Medical Service Scheme: 50% to 60% of the cases may be on prescription for general occupational therapy. In passing it may be interesting to note as a parallel, from data collected in America in 1938, that in a mental hospital with an excellent organization for occupational therapy the figures given for Special and General prescriptions correspond almost identically with these.

In the more particular application of the work to physical cases, occupational therapists are usually asked for treatment aimed at the restoration of function. Prescribing physicians and surgeons indicate that this is best achieved by the joint or muscle working with other joints and muscles in as normal a way as possible. Isolation of muscles is not encouraged unless essential. There is a need, therefore, for very careful choice of occupation and apparatus, and the working position of the patient, the particular stool or chair on which he will sit, as well as the work itself, must be considered. As essential as these points, are clear instructions to the occupational therapist, and ample time on the part of the latter to give supervision. Only if absolutely necessary are any artificial means of restriction or splinting used, and these for as brief a period as possible, their purpose being mainly to help the patient to think the right movement as well as to achieve it. To illustrate this point, there was one patient who had had a bad Colles's fracture. When given steady hammering to encourage flexion of this wrist, the patient worked merely by abducting and adducting his upper arm. For a brief period he was placed in a position with the forearm fixed and all movement had to be thought of and carried out at the wrist. The patient himself was impressed by the speed with which he overcame his efforts at compensatory movement and gained real benefit from the prescribed action.

In occupational therapy the psychological aspect of all cases comes under consideration. By this is not meant, however, any attempt on the part of the occupational therapist to treat the patient as a "mental case". Great emphasis is laid on the treatment of the case as a normal person—as a whole man. If, however, the patient is finding difficulty in adjusting to working conditions, a valuable contribution to the better and quicker recovery of the patient can be made by an occupational therapist, with a well-balanced outlook, based on a study of practical psychology as applied to patients, and with more time available for treatment than the medical officer in charge of the case.

This brings us to the social aspect. The interpretation of the term means the re-socializing of the a-social, thus counteracting the effects of long hospitalization and perhaps boredom. The busy atmosphere in an occupational therapy department, the expectation and acceptance of work, the comradeship and triumph of achievement can, almost without the direct agency of the occupational therapist, bring surprising results. Recreation is widely used, sometimes under the direction of the occupational therapist, sometimes as successfully, or more so, in the hands of other experts. Economically, occupational therapy would not be completing its work unless it made its contribution towards putting the patient one step further on the road to security. There is

some confusion as to where occupational therapy ends and vocational training begins.

The Ministry of Labour interim scheme for the training and rehabilitation of disabled persons offers valuable scope to occupational therapists for collaboration and experiment.

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Below is an outline scheme suggesting a possible grading of patients for rehabilitation in hospital, and the type of occupational therapy for each grade:

X. WARD CASES

A. Bed cases.—Given Special or General Occupational Therapy, e.g. Light Canework, Leatherwork, Weaving, Netting, Cord Knotting, Lettering, &c., or other suitable bed-work, such as making of requisites for hospital, &c.

B. Cases up for brief periods only.—Occupational therapy as above—or perhaps short visits to occupational therapy workshop for light work.

Y. AMBULANT CASES (Workshop and Gardens)

C. Those still on light work—with progressively extended work periods.—Light wood-work added to list of above occupations.

D. Stronger cases.—Doing some hospital duties, as general occupational therapy—and receiving other general or special occupational therapy in the workshop, probably doing woodwork, metal work or gardening.

E. Strongest cases.—Work as above, but for still longer periods and with greater resistance offered by graded tools and apparatus. Possibility of encouraging working speeds.

The minimum staff for the above would be a supervising occupational therapist, an assistant, a gardener-joiner, a part-time metal worker and voluntary helpers.

The training of an occupational therapist consists of medical (administrative and occupational) subjects, and the application of the latter to diseases and disabilities.

The qualifying examination is run by the Association of Occupational Therapists, the examiners being medical and occupational experts.

Two cases which received special treatment are quoted below:

(1) *Motor-cycle accident.*

Fracture of right femur and patella; right radius and ulna; and third metacarpal. Left Bennett's fracture.

After five weeks he was put on occupational therapy, in bed.

Tried weaving flat rug on frame. General movements too tiring. (Thumb plaster renewed, operation on arm.)

Leatherwork (successful) but fingers weak. Sent to workshop—four weeks later—to mobilize right knee. Hard work not contra-indicated. Used adapted weaving loom—the beating being achieved by strong extension of knee. Lifting foot on to high pedal produced flexion of knee.

Fingers of right hand specially mobilized by arrangement of design, and shuttlework. Right arm now in plaster—with elbow hinge made, at surgeon's direction, in occupational therapy department—and fixed into plaster at each end.

Treatment continues.

(2) *Air-raid casualty.*

Bilateral fractures of tibia and fibula. Lacerations and burns, legs, arms and hands. Occupational therapy prescribed. Knees and ankles stiff. Left leg in plaster. Right leg not weight bearing. Work given was turning of potter's wheel, rhythmically, in small range (providing foot power for another patient to work), and wedging clay with hands. Patient was transferred to light loom worked by roller skates on wooden track: sitting on special stool with flap support for knees.

Strength of resistance increased—work excellent. Patient showed considerable improvement on discharge.

[The views expressed in these papers are not necessarily the views of the Ministry of Health, but are a purely personal expression of opinion.]

Section of Anæsthetics

President—A. D. MARSTON, D.A.

[March 6, 1942]

Trichlorethylene as a General Analgesic and Anæsthetic

By C. LANGTON HEWER, M.B., B.S., D.A.

ABOUT two years ago, I was asked by Dr. Charles Hadfield, secretary to the combined Anæsthetics Committee of this Section and of the Medical Research Council, to investigate the possibilities of trichlorethylene as a general anæsthetic.

In June 1941 I made an interim report in the form of a short article in the *British Medical Journal*. The results obtained were so encouraging in certain directions that since that date I have continued to use the drug, and I now hope to put before you the conclusions I have reached.

Physical properties.—Trichlorethylene was first described in 1864; its chemical formula is $\text{CCl}_2:\text{CHCl}$. It is a heavy colourless liquid with a specific gravity of 1.47 at 15° C. and a boiling point of 87° C. The odour resembles that of chloroform without its pungency. In view of the similarity of the two fluids in respect of both weight and smell, I have suggested to the manufacturers that trichlorethylene prepared for anæsthesia should be coloured either blue or green. This would readily distinguish it from chloroform which is either colourless or tinted red by one maker.

Trichlorethylene is not inflammable in any circumstances nor will its vapour explode when mixed in any proportion with air, oxygen or nitrous oxide.

The pure drug tends to decompose in strong sunlight with acid formation, and should therefore be stored in stoppered amber bottles. The addition of 0.01% thymol retards decomposition, and this has been done in the case of the product known as "trilene". As an extra precaution, the manufacturers suggest that it should not be used for inhalation purposes after twelve months from the date of bottling.

The cost of purified trichlorethylene is very reasonable as if supplied in lots of 8 lb. to hospitals the price is 3s. 6d. per lb. It has been estimated that the average cost per administration works out at about 4½d.

Commercial uses.—Trichlorethylene is chiefly used for the dry-cleaning of clothes, the de-greasing of metals and the de-waxing of lubricating oils. For these purposes, the fluid is sold under a variety of trade names such as chlorylene, gemalgene, trethylene and triklone. These preparations may contain a variety of impurities and should not be used as anæsthetics.

It is of some interest to know that *addiction* to trichlorethylene is not very uncommon amongst industrial workers. The vapour used for de-greasing metals rises in a vat and is condensed by cold pipes. Some workmen find considerable pleasure in leaning over the sides of the vats and inhaling the vapour until they feel intoxicated.

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[The views expressed in these papers are not necessarily the views of the Ministry of Health, but are a purely personal expression of opinion.]

Details of present investigation.—The observations which follow are based upon about 400 administrations. Most of the commoner major and minor operations were performed under trichloroethylene anæsthesia or analgesia, their duration varying from five minutes to five hours and forty minutes: the latter operation being the removal of a cerebral tumour from a woman with bronchiectasis. The administration in this case was by my colleague Dr. B. Rait-Smith and I am glad to say that the patient survived. The patients' ages were between 14 months and 81 years and included Service casualties, air-raid casualties and ordinary hospital civilian patients.

Methods of Administration

It at once became evident that an open mask administration was impracticable owing to the low volatility of trichloroethylene. In America a special apparatus has been devised to vaporize the drug incorporating an air compressor driven by an electro-motor (Jackson, 1934). As we had neither the wish nor the facilities to develop complicated machinery, we began by putting trichloroethylene in the chloroform bottle of a continuous-flow apparatus and using it as an adjuvant to nitrous oxide and oxygen. When used with partial rebreathing this method was quite satisfactory. In most cases a face-piece was employed, but, when indicated, a nasal or oral endotracheal tube was passed. Muscular relaxation was usually ample for intubation to be performed without difficulty.

If the CO₂ absorption technique is used, it is essential to have the vaporizer in the patient's respiratory circuit. Insufficient concentration of trichloroethylene is obtained if the basal oxygen feed alone is used to vaporize the drug as in the single-phase Waters' system or in the standard two-phase apparatus as supplied to the E.M.S.

After a fairly full investigation of trichloroethylene as an adjuvant to nitrous oxide and oxygen, it was decided to find out whether the drug had any possibilities when given alone. After various trials and errors, a simple draw-over apparatus was adapted from a Walton ether bottle. In order to produce analgesia only, a small bottle without a rebreathing bag is sufficient, but to ensure that the third stage of anæsthesia is reached, a large bottle containing a wick is necessary and some rebreathing is desirable. By plugging in a similar ether bottle in series a compact apparatus results which is adaptable to practically any type of inhalation analgesia or anæsthesia including endotracheal work. In the latter case it is, of course, essential to have an air-tight fit, either by plugging or by the use of a ballooned tube. Dr. H. R. Marrett, Senior Resident Anæsthetist at Hill End Emergency Hospital is largely responsible for the design of this apparatus which (with one bottle) can be produced to sell retail at about £5 10s.

When used continuously for general anæsthesia, about 13 c.c. of trichloroethylene per hour are consumed.

Stages and Signs of Analgesia and Anæsthesia

In most respects trichloroethylene resembles chloroform rather than ether.

During the first stage, some degree of *general analgesia* always seems to be present. This comes on after a few breaths and varies in degree from some numbness to pain in resistant patients to that of absolute analgesia during which can be carried out such procedures as wedging plasters, insertion of bone pins, cystoscopies, painful dressings, &c. The sensation of general analgesia is one of curious detachment. One patient rather aptly described it by saying that he appeared to be watching himself being operated upon as if he was a disinterested third party. Two patients suffering from trigeminal neuralgia were given trichloroethylene and the pain was practically abolished in both cases, but at the same time sensitivity to pain was greatly diminished in all areas. I am convinced that the relief in this condition is due to general analgesia and not to any specific effect upon the trigeminal nerve, in spite of the statement made in the book just published by no less a pharmacological authority than Adriani. Trichloroethylene exerts the most potent and rapid analgesia of all the volatile drugs with which I am familiar, including nitrous oxide, and it appears to me that it might be of very great use, especially under war conditions where the absence of heavy and complicated apparatus, and hot water jackets, gas cylinders and inflammable vapours are all desirable.

The second stage of anæsthesia is not usually marked but occasionally violent excitement occurs.

The third stage is characterized by quiet automatic respiration and a pupil which is usually small. The eye reflexes are similar to those obtaining with chloroform.

Industrial poisoning.—On the Continent several writers have recorded cases of industrial poisoning in factory workers when crude trichlorethylene was used. Symptoms such as giddiness, vomiting, optic neuritis and various palsies have been described (Plessner, 1916; Gerbis, 1928). Some cases have proved fatal (Stiiber, 1931). It should be noted that all these patients had been exposed to the vapour of commercial trichlorethylene which, as already mentioned, may contain a great variety of impurities. Workers constantly exposed to trichlorethylene vapour in aeroplane works are said to be liable to dermatitis (Schwartz and Russell, 1941).

External application.—A few years ago, the use of trichlorethylene was recommended as a skin purifier and to clean up burns and dirty wounds (Trumper and others, 1936). For these purposes Imperial Chemical Industries Ltd. produced a specially purified and stabilized liquid under the name "trilene". With the exception of some pure trichlorethylene made and generously supplied by Mr. C. Chalmers who originally suggested the investigation, all cases have had trilene for inhalation.

Therapeutic inhalation.—The drug has been used for some time to relieve the pain of trigeminal neuralgia. For this purpose, the vapour is inhaled from broken capsules as in the case of amyl nitrite. The origin of this procedure is worth noting. Two separate observers reported that patients suffering from chronic trichlorethylene poisoning showed complete bilateral paralysis of all divisions of the trigeminal nerve (Plessner, 1916; Gerbis, 1928). It was supposed that the drug had a specific action on this nerve and its administration for the relief of trigeminal neuralgia was suggested (Oljenick, 1928) and shown to be effective (Glaser, 1931). It appears to me quite certain that the relief from pain is not due to any effect on the trigeminal nerve but to the state of general analgesia which is induced and to which I will refer later. This seems to be an example of faulty reasoning leading to the desired result.

Experimental work on animals.—During the past twenty-one years several workers have fully investigated the effects of trichlorethylene on animals.

In 1921 Joachimoglu found that the pure vapour was not irritating to the respiratory tract and that inhalation was not followed by hæmolysis or by fatty degeneration of the liver.

In 1934 Herzberg reported that specimens of spleen, liver, kidney, pancreas, adrenal, diaphragm, heart, lung and pectoral muscle taken from three dogs killed by overdosage of trichlorethylene after having been deeply anæsthetized for periods of two and a half to three and a half hours showed no gross or microscopic pathological changes.

In 1935 Krantz and others anæsthetized the same rats repeatedly (up to 30 times) with trichlorethylene. The animals were then killed and the various organs examined. Although some pathological changes were found, they were in the main slight and inconclusive. In rabbits slight hyperglycæmia was noted. The same workers found that anæsthesia could not be obtained by the rectal administration of the drug and that nerve conduction was not affected by the local application of trichlorethylene.

In 1939 Lande and others anæsthetized guinea-pigs, rats and mice with trichlorethylene daily for two and a half months, the duration of narcosis being from fifteen minutes to one and a quarter hours per day. After death it was hardly surprising that some inflammatory changes in the liver and kidneys were noted.

From these extremely severe tests it may reasonably be concluded that in the usual experimental animals, trichlorethylene is only slightly toxic—certainly much less so than chloroform.

Previous work in human anæsthesia.—The only published work which can be traced is that on a series of 300 patients anæsthetized with trichlorethylene by Striker and others in America in 1935. These were all short administrations for minor operations, and in some instances analgesia only was produced. Eight of these patients became violent in the second stage, and the third stage of anæsthesia could not be reached. One patient stopped breathing from an overdose but recovered after artificial respiration. After-effects were slight, and on the whole the drug was regarded as a satisfactory anæsthetic.

In the following year (1936) the Council of Pharmacy and Chemistry of the American Medical Association considered the evidence for the usefulness of trichlorethylene and concluded that "the case had not been completely made out".

Details of present investigation.—The observations which follow are based upon about 400 administrations. Most of the commoner major and minor operations were performed under trichlorethylene anæsthesia or analgesia, their duration varying from five minutes to five hours and forty minutes: the latter operation being the removal of a cerebral tumour from a woman with bronchiectasis. The administration in this case was by my colleague Dr. B. Rait-Smith and I am glad to say that the patient survived. The patients' ages were between 14 months and 81 years and included Service casualties, air-raïd casualties and ordinary hospital civilian patients.

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The third stage is characterized by quiet automatic respiration and a pupil which is usually small. The eye reflexes are similar to those obtaining with chloroform.

Effects on Respiratory System

The odour of trichlorethylene is not pungent nor does its vapour appear to have any irritant effect upon the respiratory passages. It follows that the vapour concentration can be increased rapidly leading to a short induction of anaesthesia. No excessive salivation or secretion of mucus was noticed even if premedication had been inadequate or badly timed. Several major thoracotomies were done under trichlorethylene anaesthesia as were many other operations upon patients suffering from bronchitis, bronchiectasis and active phthisis. In no case could any exacerbation of the pulmonary disease be attributed to the anaesthetic. The depth of respiration was not appreciably altered but in about 30% of patients receiving the drug as an adjuvant to nitrous oxide and oxygen, some increase in the rate occurred. This usually subsided after fifteen to twenty minutes but in a few cases it persisted. When present, this rapid breathing is a definite disadvantage in thoracic surgery. The tendency was not so marked when trichlorethylene-air was used. On one occasion an overdose was inadvertently given. Respiratory arrest occurred, but no difficulty was encountered in restoring natural breathing. The pulse remained steady throughout.

Effects on Cardiovascular System

The blood-pressures usually remained within normal limits except for some rise when the second stage of anaesthesia was marked by much excitement. Dilatation of the vessels in the skin and subcutaneous tissues such as occurs with ether and cyclopropane was not seen, and surgeons frequently commented upon the absence of capillary oozing. This was particularly noticeable in nasal and in ophthalmic surgery. The pulse-rate was generally raised slightly as in the case of most inhalation anaesthetics.

Particular attention was paid to the cardiac rhythm, as trichlorethylene contains three chlorine atoms in its molecule, and at least two other compounds with this characteristic (chloroform and trichlorethanol) can cause grave cardiac irregularities and even primary cardiac failure (Wood, 1938; Hewer and Belfrage, 1938). Clinically few cardiac irregularities could be detected apart from sinus arrhythmia in young patients which disappeared when full anaesthesia was reached. An irregular pulse was definitely less common than with cyclopropane, and several patients with extrasystoles before operation showed a regular rhythm after anaesthesia had been induced with trichlorethylene. This is, of course, common with inhalation anaesthesia generally. Two cases of partial thyroidectomy had auricular fibrillation which remained unchanged throughout the anaesthesia.

The possibility of primary cardiac failure still cannot be entirely excluded, but so far I have not heard of such an event although a large number of administrations have now been made. Since the publication of my original paper, 48 hospitals have been using trichlorethylene and it has been estimated that at least 10,000 patients have been anaesthetized with it.

Dr. K. D. Keele kindly arranged for electrocardiograms to be taken during the induction of anaesthesia in 33 cases. A Cossor-Robertson cardiograph and later a Cambridge instrument were installed in one of the anaesthetic rooms, and direct visual observations as well as photographic records were made before and during induction. In most of the cases so observed there was no alteration in the tracing apart from a slight decrease in the sinus tachycardia as the third stage of anaesthesia was reached. In one instance regular auricular extrasystoles alternating with normal systoles occurred over a period of some three minutes and then ceased. In two other cases occasional ventricular systoles were seen during the second stage, but disappeared as the third stage was reached. In no case were multiple ventricular extrasystoles seen. These were particularly looked for in view of their comparatively frequent occurrence during chloroform anaesthesia. This limited number of observations suggests that there is no evidence of any particular cardiac danger with trichlorethylene.

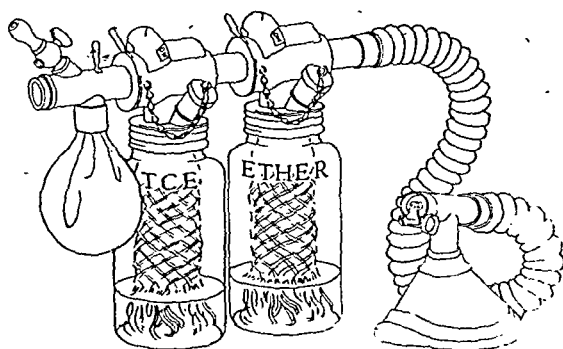
Muscular relaxation.—The degree of muscular relaxation obtainable with trichlorethylene is variable. As a general rule, operations upon the head, neck, thorax and limbs present no difficulty, but adequate relaxation for major abdominal surgery is not always readily available. It is possible that a really deep plane of narcosis may produce the desired result, but I have always played for safety and changed over to ether in preference to pushing trichlorethylene. One opportunity occurred for observing the effect of the drug upon the uterine musculature. An abdominal hysterotomy was performed

to terminate pregnancy on account of active phthisis. The surgeon remarked upon the exceptionally good retraction of the uterus following extraction of the foetus. After an average of twenty minutes' administration, it was found that the vapour concentration of trichlorethylene could be greatly reduced without prejudice to muscular relaxation.

Effects on sugar metabolism.—Dr. A. Jordan kindly made blood-sugar estimations before, during and after anæsthesia with trichlorethylene. In no case was there much disturbance, and taking an average, it could be said that there was no significant change. This contrasts with the rise constantly seen with chloroform and ether (Hewer, 1939).

One diabetic patient was included in the series. An abdominal operation lasting one hour fifteen minutes was performed under nitrous oxide - oxygen - trichlorethylene anæsthesia. The sugar tolerance was tested one week after operation and was found to be unchanged.

Effects on blood urea.—Blood-urea estimations were made by Dr. Jordan before, during and after anæsthesia. The changes were so slight as to be within the range of experimental error. Here again the contrast with chloroform and ether is marked.



"Draw-over" inhaler for producing either analgesia or anæsthesia with trichlorethylene and/or ether. It is suggested that this might prove useful both in civil practice and under Service conditions.

(Block kindly lent by Messrs. A. Charles King, Ltd.)

After-effects

If analgesia only has been produced, it is unusual for any after-effects to occur. Occasionally slight dizziness or headache may be present for a short time.

Of the first 127 patients anæsthetized for a great variety of operations, 61% had no nausea or vomiting, 26% had nausea or slight vomiting and 13% moderate or severe vomiting; 5% complained of headache. Those who had had previous experiences with ether nearly always commented favourably upon the absence of an unpleasant taste and smell.

In most cases the urine was tested as a routine on the day after operation. In one instance only was the presence of albumin found and acetone was always absent. This is in marked contrast with other anæsthetics. For example albuminuria has been found almost constantly after the prolonged administration of ether and in about 20% of patients anæsthetized with chloroform (Stephen, 1929). Again, acetonuria has been demonstrated in 67% of patients operated upon under all forms of general anæsthesia and in 85% under local analgesia (Schulze, 1924). No pulmonary complications were observed, and, as already mentioned, existing lesions did not appear to be affected.

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Section of Comparative Medicine

President—G. DUNLOP-MARTIN, M.R.C.V.S.

[December 17, 1941]

DISCUSSION ON THE CONTROL OF DISEASES OF CATTLE INIMICAL TO MAN

TUBERCULOSIS

[For first Discussion see PROCEEDINGS, December, 35, 115 (*Sect. Comp. Med.*, 1—8)]

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Section of Comparative Medicine

President—G. DUNLOP-MARTIN, M.R.C.V.S.

[December 17, 1941]

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other than bovine tubercle bacilli, the most important of which is the avian tubercle bacillus. While it has been recognized for some years that cattle become infected with this organism and develop an allergy to mammalian tuberculin, it is only within fairly recent times that the incidence of the infection has been realized. It has been shown conclusively that mammalian tuberculin of high potency demonstrate the allergy to avian tuberculous infection and so important has this state of affairs become that it has been necessary, in a number of herds, to use the specific avian tuberculin in differentiating bovine and avian tuberculous infections in cattle. Infection with avian tubercle bacilli has complicated the story of tuberculin testing. It is necessary in searching for the most satisfactory tuberculin to consider the potency and dose which will detect the largest number of animals infected with bovine tubercle bacilli and the least number infected with other organisms which give rise to an allergy detected by mammalian tuberculin.

A matter of much importance is the disposal of infected cattle. The regulations in this country provide for the slaughter of "open" cases of tuberculosis but no provision is yet made for the disposal of cattle which react to the tuberculin test. The result is that in clearing a herd from tuberculous infection by the use of the tuberculin test many infected animals may find their way to the open markets and in their turn may create new sources of infection or may perpetuate infections in herds throughout the country.

(3) The creating of resistance or immunity to tuberculosis in cattle has been the subject of study and many products have been used, varying from tuberculin to dead and living organisms. The use of each product has been followed by favourable reports, but few have stood up to carefully controlled experimentation. A brief reference will be made to B.C.G. and the vole acid-fast organism.

B.C.G. was originally a virulent strain of bovine tubercle bacillus which, by repeated subcultivation on a suitable medium was rendered innocuous to cattle though it still retained antigenic properties. The recorded results with B.C.G. vary considerably, but there is much evidence to support the original findings that, used in the manner prescribed, injections of this strain set up a high degree of resistance to infection with bovine tubercle bacilli. The experiments carried out in this country, notably those by Buxton, Glover and the late Stanley Griffith, demonstrate that B.C.G. is of considerable value for controlling bovine tuberculosis in cattle. The method devised by these workers is to create a resistance in the young, healthy bovine by the injection of B.C.G. and to reinforce the resistance by further injections at intervals. It is essential that the animals are free from infection at the time of the first injection and that they are protected from infection till the necessary degree of resistance has been established. The subsequent behaviour of the "protected" animals appears to be dependent largely on the degree of infection to which they are exposed. It is apparent that in spite of exposure to heavy infection the protected animals either resist the infection or become infected only to a mild degree, the lesions being much less marked, in general, than those of control, unprotected animals. More recent results of field experiments indicate that animals treated regularly with B.C.G. resist in a marked degree infection in herds in which "open" cases of tuberculosis are present. The results seem to be sufficiently encouraging to merit an extensive field trial in suitable herds. It must not be assumed, of course, that our knowledge of the use of B.C.G. is complete. Much information, now lacking, would be obtained from such a field trial.

Within quite recent times an addition has been made to the group of acid-fast organisms by the isolation by Wells from the vole of an organism with many features common to tubercle bacilli. This organism has been used in experiments for the creating of a resistance to infection with bovine tubercle bacilli. The conclusions of Wells and Brooke are that the vole bacillus "gives a degree of protection which apparently is far greater than has been recorded by other means". We have also worked with Wells' organism at Cambridge and in an article published by the late Stanley Griffith and myself, we recorded the results of its injection into guinea-pigs and calves. Although the number of calves used in the experiments was small we came to the conclusion that "the results obtained in calves with the vole strain of acid-fast bacillus were unexpectedly good and better than those which followed the use of B.C.G. as a vaccine". Griffith and I expressed the opinion that the immunizing power of the strain should be further tested in calves: work is now in progress at Cambridge and although the experiments have not progressed very far it is evident that the former results are being borne out. There is still much to be done concerning the use of the vole strain before we are ready for a large field trial. Such points as the duration of resistance to bovine tuberculosis following the use of a single dose of the organism and the degree of resistance set up must be the subject of further work. It would seem, however, that in the vole organism we have a product which might be found of value in the control of bovine tuberculosis.

A disadvantage of both B.C.G. and the vole organism is that their injection is followed by an allergy to mammalian tuberculin. There is some evidence that the allergy fades in time and though the use of these products may complicate eradication schemes in some measure, further work on the subject will show in how far the two methods of control (elimination of "reactors" and the creating of resistant animals) can be carried out at the same time.

THE PREVALENCE OF HUMAN TUBERCULOSIS OF BOVINE ORIGIN

Dr. S. Roodhouse Gloyne: Bacteriological statistics.—There is no reliable clinical test for differentiating between human and bovine type infections in man, whilst the belief that tuberculins prepared from the two strains do not produce diagnostically different skin reactions has been held fairly generally for many years. Calmette was very dogmatic that there was no difference whatever, and the question seems to be regarded as closed—perhaps a little prematurely. The detection of the bovine type has therefore to be arrived at by relatively slow and laborious bacteriological investigations which are usually considered to be of secondary importance in routine clinical pathology, and are, consequently, more often than not, omitted. If we had some quick and reliable method of demonstrating bovine infection, we could more readily bring the lesson home to everybody concerned. There is, however, no lack of statistical material on this aspect of the subject. The bacteriological investigations show that there is a higher percentage of bovine type infections in Scotland than in England. In 1937 Griffith summarized his findings which are shown in the rearranged table:

| Variety of tuberculosis | Percentage of cases infected with the bovine type of bacillus | | | | Variety of tuberculosis | Percentage of cases infected with the bovine type of bacillus | | | |
|-------------------------|---------------------------------------------------------------|---------------|------------|----------|-------------------------|---------------------------------------------------------------|---------------|------------|----------|
| | Number of cases | Under 5 years | 5-15 years | All ages | | Number of cases | Under 5 years | 5-15 years | All ages |
| ENGLAND | | | | | SCOTLAND | | | | |
| Cervical gland | 126 | 90.9 | 53.4 | 50.0 | Lupus | 13 | 100.0 | 71.4 | 69.2 |
| Lupus | 191 | 55.4 | 44.4 | 48.7 | Cervical gland | 43 | 65.0 | 62.3 | 51.6 |
| Scrofuloderma | 60 | 53.3 | 43.3 | 36.6 | Genito-urinary | 42 | — | — | 31.0 |
| Meningeal | 265 | 28.1 | 24.5 | 24.6 | Bone and joint | 218 | 46.2 | 28.9 | 20.8 |
| Bone and joint | 553 | 29.5 | 19.1 | 19.5 | Meningeal | 203 | 34.4 | 14.0 | 29.6 |
| Genito-urinary | 23 | — | — | 17.4 | | | | | |

One of the most extensive Scottish investigations is that of Blacklock, carried out on tuberculous children in Glasgow and the West of Scotland. Of 241 strains isolated, 152 were of the human and 89 of the bovine type. Pulmonary tuberculosis is not included in Griffith's table because up to 1922 only four cases of bovine bacillus infection in phthisis had been reported and the condition was regarded as a bacteriological curiosity. About this time Munro, and others discovered more cases in Scotland, and further investigation was stimulated, with the result that Griffith was able to report fifteen years later, no less than 163 instances in England and Scotland, and the number is still increasing.

Summarizing the figures, it may be stated that in England, taking all ages into consideration, about half the cases of cervical gland tuberculosis and lupus, about one-quarter of the meningeal, and rather more than one-sixth of the bone and joint and genito-urinary cases are due to the bovine bacillus. In practically all instances the figures for Scotland are higher. In pulmonary tuberculosis, this difference is especially marked. Griffith's last figures were 1% for Wales, 1.6% for England, and 7% for Scotland. Lastly, there is evidence to suggest that bovine type infections vary even in different parts of Scotland and that they are higher in the North of England than in the South.

Calculations from the Registrar-General's Statistics

Sir William Savage has estimated that 1% of respiratory and 23% of non-respiratory tuberculosis, or 5.5% of the total deaths from all forms of the disease at all ages are due to the bovine type of bacillus. Applying these factors to the Registrar-General's statistics he has estimated that in the year 1927 there were 310 deaths from respiratory and 1,635 from non-respiratory forms of tuberculosis, making the calculated total deaths from tuberculosis of bovine origin to be 1,945. If Savage's method of calculation is applied to the Registrar-General's figures for the last year's statistics available, viz. 1938, we obtain the following:

| | | |
|--------------------------------------|---|------------------------------------------------------|
| 21,930 respiratory disease deaths | = | 219 calculated deaths due to bovine bacillus |
| 4,246 non-respiratory disease deaths | = | 976 calculated deaths due to bovine bacillus |
| 26,176 total deaths | = | 1,195 calculated total deaths due to bovine bacillus |

Cobbett writing during the last war calculated from the Registrar-General's statistics that 33% of the mortality from all forms of tuberculosis in children under 5 could be attributed to the bovine type.

These figures based on Registrar-General's Reports take no account of the non-fatal cases. Stanley Griffith in one of his last papers stated that he regarded the bovine strain as at least as virulent as the human. If the calculations are based upon notifications instead of upon death statistics, the resulting figures for 1938 are:

| | | |
|----------------------------------------------|---|-------------------------------------------------------------|
| 37,879 respiratory disease notifications | = | 378 calculated notifications due to bovine bacillus |
| 12,810 non-respiratory disease notifications | = | 1,946 calculated notifications due to bovine bacillus |
| 50,689 total notifications | = | 2,324 calculated total notifications due to bovine bacillus |

Milk Sampling

The source of the bovine bacillus.—When Delepine began his pioneer work in Manchester, the percentage of positive milk samples was 17·2. That was in 1897. In his last year, 1907, the percentage had fallen to 5·9. In 1931 a Ministry of Health Memorandum gave percentage figures for 1922-27 for certain selected areas as ranging from 5 to 9·8. The results obtained in the laboratories of the London Chest Hospital are mostly from mixed samples collected in the Home Counties. Records of 1,560 samples examined during the years 1928-1931 yielded 9·1% of positives. Thereafter the figures improved, and during the period 1935-1939 the positives fell to 6·2%. Although our more recent records have been destroyed by enemy action, I have reason to believe that the percentage of positives had declined still further to about 4 at the outbreak of war. There was, however, a disturbing increase in our figures during 1940.

Special Categories of Milk

In dealing with special designations of milk the emphasis must be laid on the tuberculin test. Even with regularly conducted tuberculin tests in a dairy herd, positive milk samples are occasionally encountered, due presumably to infection arising between one test and the next. These positives probably number, however, less than 1%. Clinical examination of the udder unsupported by a tuberculin test does not in my experience of milk testing greatly reduce the percentage of positive samples, whilst in the case of pasteurized milk everything depends on the efficiency of the plant. An investigation in Scotland recorded in 1933 showed that the percentage of positive milk samples after the holding method of pasteurization varied from 4·9 in Dundee to 1 in Edinburgh with an average for the four large cities of Edinburgh, Dundee, Glasgow and Aberdeen, of 2·8%. (The figure for the raw bulked milks assembled in the containers averaging 600 gallons, was 37·5%.) Tests at the London Chest Hospital yielded 2·5% of positives in 276 samples. I presume none of us will be satisfied with these figures, and personally I am convinced that if pasteurization is to be generally adopted in this country, certain safeguards must be adopted: (1) The operation must be carried out at well organized central depots by experienced persons. (2) Only pasteurizing plants which have been tested and passed by the public health authorities should be used. (3) Bacteriological samples before and after pasteurization must be taken regularly in just the same way that bacteriological samples are taken of a well regulated water supply. (4) Regular inspection for the detection of technical engineering defects is needed.

Milk Products

The commercial products which are likely to contain bacilli are raw cream, dried and condensed milk, butter and cheese. When a milk sample containing tubercle bacilli is centrifuged, the organisms may be found in the cream layer as well as in the deposit. It is therefore reasonable to assume that raw cream separated from a bulked milk sample has about as good a chance of containing tubercle bacilli as the milk from which it is separated, but relatively few investigations have been made as compared with milk and in actual practice the amount of raw cream consumed is not great. Clotted cream and cream from pasteurized milk being heated come into quite a different category, whilst dried and condensed milk undergo special treatment which ought to destroy tubercle bacilli. Some years ago, I made an investigation of 30 different brands of condensed milk, machine skimmed and full cream, English and foreign purchased in small grocers' shops in the London area. Most of them showed recognizable cells in the centrifuged deposit, and one contained a few acid-fast bacilli, probably phagocytosed, but all the brands were negative to the guinea-pig test. There are only a few recorded series of tests with butter and cheese and the numbers are so small that they are not statistically significant. In peace-time about half our butter supply and a considerable amount of our cheese are imported from countries where bovine tuberculosis is not common, and pasteurization almost the rule. It is probably safe to assume for practical purposes, therefore, that, as Savage has remarked, the majority of the examples of human tuberculosis of bovine origin can be credited to the consumption of liquid milk containing living tubercle

bacilli, and I should doubt if the exigencies of the war are likely to alter this general statement. Liquid milk must always be the first object of our bacteriological control.

The Infecting Dose

Practically nothing is known as to the minimum dose of tuberculous milk which is necessary to cause infection in man, and not a great deal as to the dose in the guinea-pig.

In children, it is generally believed that the tubercle bacilli can pass through the intact mucosa of the intestine, but whilst this is so, there is evidence both in infants and in calves that primary lung infection can take place by the regurgitation of milk from the oesophagus. In the Lubeck disaster 251 infants were given by mouth B.C.G. vaccine to which a virulent human strain of tubercle bacillus had accidentally gained access in the laboratory. Of these children, 77 developed tuberculosis from 2 to 498 days afterwards. Naturally the majority had primary abdominal lesions, but a significant number had primary lung lesions.

In the guinea-pig I have, on two separate occasions, obtained positive results with the subcutaneous inoculation of 1 c.c. of uncentrifuged milk from a bulked sample from five cows.

This subject of dosage is therefore of more than academic interest. In man, it would throw light on the whole question of human tuberculosis of bovine origin to know how many of the healed mesenteric gland lesions met with post mortem, are due to bovine infection but unfortunately there are experimental difficulties in determining the answer. In the guinea-pig the variation in dose—and consequently in the length of time taken to develop tuberculosis—leads not only to delay in reporting samples, but to variations in the percentage of positives. It has been claimed, for example, that the percentage of positives can be increased by as much as 50%, by using two animals for each test, and allowing one of them to survive for eight weeks. It is false economy to be so parsimonious in our tests where so much is at stake.

CATTLE CONTACTS AND CARRIERS

This brief survey cannot be closed without some mention of the relationship which at first sight seems so anomalous. The investigations of Griffith, Munro and Cumming have brought new facts on the relationship between chronic pulmonary tuberculosis and bovine infection. A steadily increasing number of cases is being recorded in which the bovine bacillus has been recovered from the sputum of farm workers suffering from phthisis. One of the interesting side-lights on the subject is the association of cervical gland tuberculosis with the pulmonary lesion, an association which is so rare in the case of human type infection, that a previous generation of physicians taught that cervical gland disease protected against phthisis. Finally, strains of tubercle bacilli are sometimes obtained from milk samples which are of low virulence for the guinea-pig. It should be borne in mind that avian strains of the tubercle bacillus are occasionally met with in cattle, and a few such have been obtained by cultivation experiments with milk samples. Apart from these contingencies, we have to remember that bovine strains are not always of full virulence. In Canada, Watson has noted what he calls a carrier state in cows, in which he believes tubercle bacilli may live at the expense of the host without causing material damage or disease. He has further shown that these carriers may lose resistance and sooner or later, when physical and chemical factors and changing environment give favourable opportunities, develop typical tuberculosis.

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(1) It is estimated that 1% of deaths from respiratory, and 23% from non-respiratory tuberculosis at all ages are due to the bovine bacillus; whilst some 33% of children under 5 who die of tuberculosis in this country are victims of this type. Recent investigations have suggested that the estimate of 1% for the respiratory form of the disease errs on the conservative side. It is as high as 7% in some parts of Scotland. In 1932 Fishberg wrote that he believed tuberculosis of bovine origin to be proportionately "more common in the British Isles than in any country in the world".

(2) If we could assume that the ratio between human and bovine type infections remains at a constant figure, we should be entitled to assume also that tuberculosis due to the bovine type is sharing in the general decline in the tuberculosis death-rate. Unfortunately there is not sufficient bacteriological control of cases in each decade to be quite sure that this is so, though the improvement in milk sampling before the war points in this direction.

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Special Categories of Milk

In dealing with special designations of milk the emphasis must be laid on the tuberculin test. Even with regularly conducted tuberculin tests in a dairy herd, positive milk samples are occasionally encountered, due presumably to infection arising between one test and the next. These positives probably number, however, less than 1%. Clinical examination of the udder unsupported by a tuberculin test does not in my experience of milk testing greatly reduce the percentage of positive samples, whilst in the case of pasteurized milk everything depends on the efficiency of the plant. An investigation in Scotland recorded in 1933 showed that the percentage of positive milk samples after the holding method of pasteurization varied from 4.9 in Dundee to 1 in Edinburgh with an average for the four large cities of Edinburgh, Dundee, Glasgow and Aberdeen, of 2.8%. (The figure for the raw bulked milks assembled in the containers averaging 600 gallons, was 37.5%.) Tests at the London Chest Hospital yielded 2.5% of positives in 276 samples. I presume none of us will be satisfied with these figures, and personally I am convinced that if pasteurization is to be generally adopted in this country, certain safeguards must be adopted: (1) The operation must be carried out at well organized central depots by experienced persons. (2) Only pasteurizing plants which have been tested and passed by the public health authorities should be used. (3) Bacteriological samples before and after pasteurization must be taken regularly in just the same way that bacteriological samples are taken of a well regulated water supply. (4) Regular inspection for the detection of technical engineering defects is needed.

Milk Products

The commercial products which are likely to contain bacilli are raw cream, dried and condensed milk, butter and cheese. When a milk sample containing tubercle bacilli is centrifuged, the organisms may be found in the cream layer as well as in the deposit. It is therefore reasonable to assume that raw cream separated from a bulked milk sample has about as good a chance of containing tubercle bacilli as the milk from which it is separated, but relatively few investigations have been made as compared with milk and in actual practice the amount of raw cream consumed is not great. Clotted cream and cream from pasteurized milk being heated come into quite a different category, whilst dried and condensed milk undergo special treatment which ought to destroy tubercle bacilli. Some years ago, I made an investigation of 30 different brands of condensed milk, machine skimmed and full cream, English and foreign purchased in small grocers' shops in the London area. Most of them showed recognizable cells in the centrifuged deposit, and one contained a few acid-fast bacilli, probably phagocyted, but all the brands were negative to the guinea-pig test. There are only a few recorded series of tests with butter and cheese and the numbers are so small that they are not statistically significant. In peace-time about half our butter supply and a considerable amount of our cheese are imported from countries where bovine tuberculosis is not common, and pasteurization almost the rule. It is probably safe to assume for practical purposes, therefore, that, as Savage has remarked, the majority of the examples of human tuberculosis of bovine origin can be credited to the consumption of liquid milk containing living tubercle

bacilli, and I should doubt if the exigencies of the war are likely to alter this general statement. Liquid milk must always be the first object of our bacteriological control.

The Infecting Dose

Practically nothing is known as to the minimum dose of tuberculous milk which is necessary to cause infection in man, and not a great deal as to the dose in the guinea-pig.

In children, it is generally believed that the tubercle bacilli can pass through the intact mucosa of the intestine, but whilst this is so, there is evidence both in infants and in calves that primary lung infection can take place by the regurgitation of milk from the œsophagus. In the Lubeck disaster 251 infants were given by mouth B.C.G. vaccine to which a virulent human strain of tubercle bacillus had accidentally gained access in the laboratory. Of these children, 77 developed tuberculosis from 2 to 498 days afterwards. Naturally the majority had primary abdominal lesions, but a significant number had primary lung lesions.

In the guinea-pig I have, on two separate occasions, obtained positive results with the subcutaneous inoculation of 1 c.c. of uncentrifuged milk from a bulked sample from five cows.

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From the point of view of preventive medicine, the keypoint would appear to be efficient pasteurization with adequate bacteriological control.

Mr. R. E. Glover discussing the effective control of tuberculosis in cattle suggested a combination of the system of eradication by periodic tuberculin testing of infected herds and the elimination of reactors, with the system of vaccination with B.C.G. Where possible, the former method should be restricted to herds which were not extensively affected with tuberculosis, while vaccination should prove of great value in the densely populated dairying districts where infection was often heavy.

Under war conditions, and for some time afterwards, every effort would have to be made to conserve existing stocks. No great progress could be expected, therefore, in the eradication of the disease by the first mentioned method but there was some hope that vaccination would reduce the incidence of tuberculosis to such an extent that any infected animals which might still remain in the herds could be eliminated without difficulty at some future period.

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Dr. G. Gregory Kayne said that to those wholly engaged in clinical tuberculosis work, the bovine bacillus obtruded little in their consciousness. This was because its effects could not be distinguished clinically from those caused by the bacillus of the human type, so that one could not easily apportion the damage caused among human beings by the two types of bacillus.

In discussing the control of tuberculosis in cattle as a disease inimical to man, the problems involved must be solved on the basis of peace-time possibilities, and any essential modifications in view of the war then made. We were dealing with a medical and veterinary problem closely bound up with social and economic considerations. Vested interests should be forgotten, otherwise, there would be introduced an unconscious distortion of facts which would falsify any conclusions formed. We were dealing with a very important and very urgent problem in the public health—how important and how urgent the general public and the medical profession appeared to ignore. The late Stanley Griffith at a meeting of the Tuberculosis Association in 1937 said: "... the latest findings in regard to this intractable problem should be made widely known, since they prove beyond any doubt that bovine tuberculosis is a serious menace which must be fought with the utmost vigour. The apathy of public opinion in this respect is astonishing in face of the incontrovertible evidence which has been published during the last quarter of a century by the scientific journals and made available to the general public in reports by a Royal Commission, the Minister of Health, the People's League of Health and individual workers." This was plain enough, and the first question must therefore be—why this apathy of public opinion?

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What was the incidence of infected cattle in this country? The figure of 40% was constantly met in the literature; it seemed to be repeated from book to book, and paper to paper. He had traced it to Sir William Savage's Mitchell Lecture in 1933, where he stated: "... on the basis of the tuberculin test the percentage of cows infected with the tubercle bacillus is extremely high. The distribution is rather variable, and may vary from 10 to 80%, but for cows in England as a whole it cannot be put below 40%."

1938 to 2,073 in 1940, but I do not know how much of this rise, if any, is due to non-pulmonary tuberculosis which contains as we have seen, the largest proportion of bovine type infections. In Wales and Monmouth there has been a rise from 110 to 149 in the death-rate per million population. This rise—the first time since 1915 that the mortality rate of any year has been so definitely in excess of that of its predecessor—is mainly due to a disturbing increase in the number of deaths from tuberculous meningitis. Whether we can attribute the usual quota of one-quarter to bovine infections I do not know.

(4) Although pasteurization is valuable in reducing the amount of infection spread by milk, it cannot be regarded as a solution of our problem, and does not eradicate tuberculosis in cattle. It is not sufficiently recognized that pasteurization is a technical operation requiring careful routine bacteriological control and that without that control a false sense of security may be encouraged. We need more bacteriological records of our pasteurizing plants. The flash process may be used in war-time.

(5) Bacteriological statistics from various parts of the country indicate that the bovine infections are unevenly distributed. The percentage is highest in Scotland and lowest in the South of England, the North of England occupying an intermediate position. There are quite local differences also.

(6) Years ago, Delepine found that these local differences existed also between the farms of various areas sending milk into Manchester. Similar variations have been noted by numerous observers throughout the country since then. There appear to be areas where bovine tuberculosis is endemic.

(7) More investigations are needed to correlate the prevalence of tuberculosis in man and in dairy cattle in the same area, or in the areas from which the milk supply is drawn.

(8) Little is known about dosage, but it has been shown that 1 c.c. of tuberculous milk will infect a guinea-pig. The excretion of bacilli in the milk probably varies from day to day.

(9) Although primary infection with the bovine strain occurs most frequently by the intestinal tract, in infants and young children it may take place by the respiratory route by regurgitation from the œsophagus. This has been shown to occur also in calves (White and Minett).

(10) We are still in need of a rapid method of diagnosis if we are to bring home to all concerned the gravity of the situation. Although the guinea-pig test is slow, the results might be expedited and improved by duplicate or even triplicate inoculations, but expense is an overruling factor. More might be done by direct film examination from the udders of individual cows. In fact an improved bacteriological service would be a paying proposition from all points of view.

(11) The control of movements of positive tuberculin reacting cattle from farm to farm leaves much to be desired. Until this is done effectively there would seem to be little hope of dealing radically with the problem. Savage suggests segregation of positive reactors. This is based on the fact that positive reactors with no clinical signs are mostly in good health, giving normal milk and only occasionally excreting tubercle bacilli.

(12) More research is needed into the conditions of natural contagion in cattle. At present a clinically tuberculous cow must be slaughtered at once on diagnosis. A good deal might be learnt if greater possibilities existed under laboratory conditions for the study of the course of the disease, especially as regards the possibility of carriers, and the sources of infection generally—for instance, the presence of tubercle bacillus in nasal mucus and other discharges, urine and feces; its survival time on naturally infected grass land, in the environment of cow stalls, in railway trucks and sidings; and its dissemination by markets and agricultural shows have received but scanty attention in this country. Hitherto we have been chiefly concerned with the transmission of the bacillus from cow to man, and with the risks of pathogenic organisms other than the tubercle bacillus added during milking. It seems time we gave more attention to the passage of the tubercle bacillus from one animal to another. Shiel has recently shown in German herds, that cows may have tubercle bacilli in their bronchial mucosa before clinical signs are obtainable and that these cows act as "carriers", infecting other animals before they themselves are detected. There are records, too, of negative reactors becoming positive after a railway journey in infected trucks, and of negative reactors becoming positive after being sent to an agricultural show. The work of Maddock has shown that grass experimentally infected with bovine tubercle bacilli remains infective for nearly six months. If this can happen in an environment exposed to the sterilizing effects of sun, wind and rain, it requires little imagination to visualize what must happen in closed cattle sheds. The experience of Guernsey is instructive in this connexion. Bovine tuberculosis seems to have been unknown until 1902, and

for this reason, American cattle buyers purchased in the Guernsey market and came to exercise a great influence. In 1902 some cattle from Guernsey imported into the United States were found to be reactors and on inquiry in the Island it was found that in the previous year a local breeder had applied to the Royal Court of Guernsey for permission to bring back to the Island some cattle which he had sent to a show in England. Such a course was contrary to the Regulations then existing, but unfortunately permission was granted. These cattle were subsequently found to be suffering from tuberculosis and an outbreak of the disease occurred, which took some years to eradicate.

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An accurate estimate seemed important, because this 40% was constantly thrown up as a decisive reason for not applying the eminently successful American method—so successful that a case of tuberculosis in a child due to the bovine bacillus was now considered rare enough to deserve publication on that score alone. This method consisted in slaughtering all reactors and paying compensation to owners. It was claimed that the application of this method here would cause a very serious shortage of milk, and embarrass the farming industry. Which was considered the greater obstacle, the first or the second?

If this method were adopted, for some time to come safety could be obtained only by means of universal pasteurization. Why was it that the Bill that would have led to this was rejected before the war in spite of all the evidence on the effects of tuberculous milk in human beings? Ontario was the first large area in the British Empire to introduce compulsory pasteurization.

And lastly, Dr. Kayne added, why was there no propaganda that milk which had not been pasteurized should be boiled before it was given to children? Were the English less intelligent or did they care less for the welfare of their children than the Spaniards or the French? In a survey made some ten years ago in several rural districts in France, Hazemann found that out of 442 children not breast fed, only 21 did not have their milk boiled.

Mr. H. W. Steele-Bodger expressed the view that the method of control of tuberculosis whether by eradication or immunization, would depend upon the character of the herd and the incidence of the disease in the area under consideration.

He did not think that it was possible to compare the conditions prevailing in the U.S.A. with those which obtained here. The incident quoted of tubercle infection having been set afloat on the Island of Guernsey following exhibition at a show demonstrated the necessity of elaborating an efficient immunizing agent. He instanced one farm in his own practice on which, following an outbreak of foot-and-mouth disease, the farm had been restocked with an entire herd of tuberculin-tested pedigree Ayrshires, which, between the twelfth and eighteenth month after purchase, became infected with tuberculosis and at one test about forty of the fifty-six cows reacted and an approximately equal proportion of the young stock. It was unfair to submit the stockowner to these grave risks. He was encouraged to hear Professor Dalling and Mr. Glover plead for the use of an immunizing agent in heavily infected areas and he wished to associate himself with this.

Over a period of ten years Mr. Steele-Bodger said he had used B.C.G. vaccine on some thirty-three herds and he was satisfied that this vaccine conferred a high degree of resistance. He pressed for field trials using B.C.G. and the vole organism.

Pasteurization did nothing to remove the trouble at its source. Routine clinical examinations of herds was of great value. In the County of Stafford, the late Dr. Menton had reported that regular biological examination of milk from herds subjected to quarterly clinical examination showed an incidence of tubercle infection in the milk of 0.5% compared with an incidence of 5% in the milk of herds not so examined.

Lt.-Col. G. Rees-Mogg said that he had tried the Calmette-Guérin vaccine on his herd for about twelve years, but had given up using it two years ago, as he had come to the conclusion that it did not give any immunity. In Ottawa six years ago, he saw in Professor Watson's laboratory organs affected with tuberculosis, from animals which had been vaccinated with Calmette-Guérin vaccine.

The speaker also pointed out that once the animals had been vaccinated, they would not pass the tuberculin test, and so there was a great financial loss.

Dr. H. H. Green said that with reference to the comments of previous speakers on the difficulty of diagnosis of tuberculosis in dairy cows, and in particular of distinguishing between mammalian infection, avian infection, and other infections such as Johne's disease, by the recognized tuberculin test, it might be of interest to draw attention to certain recent work at the Veterinary Laboratory of the Ministry of Agriculture at Weybridge. The biochemical department had been conducting studies upon the changes in composition of synthetic media during the growth of various acid-fast organisms, notably human, bovine and avian strains of tubercle bacilli, B.C.G., Johne's bacillus and the non-pathogenic *M. phlei*. The tuberculin department had been subjecting various protein derivatives prepared in the course of this work to comparative biological tests with the ultimate object of utilizing them for differential diagnosis of natural infections.

Dr. A. Eden said the introduction of synthetic media of the asparagine-glycerol type in place of the chemically complex glycerinated broths as previously employed for the manufacture of tuberculins had opened up a ready means whereby tuberculins could be standardized. All available evidence showed that the active fraction of tuberculins is of

a protein nature, and this is readily capable of chemical standardization by precipitation of the protein with trichloroacetic acid and the determination of the nitrogen content of the precipitate. At the Veterinary Laboratory, Weybridge, chemical studies of the changes undergone by human type *M. tuberculosis* growing on synthetic media showed that by the end of eight weeks' growth practically all the glycerol, dextrose and amide nitrogen fractions disappeared from the media and that there was a huge production of ammonia and a gradual production of water-soluble, non-coagulable protein in the medium equivalent to as much as 0.15 g. protein per 100 ml. original medium. Similar studies carried out with avian type *M. tuberculosis* showed essentially similar chemical changes in the medium except that the production of the active, water-soluble protein was only one-third that of the human types.

Since tuberculins were prepared by heat concentration of the bacteria-free culture media at the end of growth, the potentialities of the two types, human and avian, for tuberculin production were very different, and assuming proportional losses in manufacture, such human type tuberculins would be three times as potent as the corresponding avian type, *apart from the question of specificity*.

The chemical method of standardization had been shown in repeated tests at Weybridge to run parallel with biological standardization and was obviously much more accurate. Further it was possible to isolate the respective proteins as dry powders (Purified Protein Derivatives or P.P.D.s) and by the simple method of solution in adequate buffer with suitable preservatives it was possible to prepare tuberculins for intradermal tests of any desired potency, and thus one variable in a number of unknown variables could be stabilized. Biological tests with such P.P.D.s reconstituted to the same protein concentration as any homologous tuberculin showed a strict parallelism. Thus chemical analysis of tuberculins was an important factor in assessing the potency of a particular batch.

Mr. J. Reid (Weybridge) said the work he had done with the P.P.D. preparations was undertaken in order to study whether by using the graded Mantoux intradermal test on suitably sensitized guinea-pigs it was possible to assess the degree of relationship in specificity between the various purified protein derivatives of the members of the acid-fast group of organisms. In other words they set out to study how many units of the heterologous product were required to stimulate a dermal reaction of intensity equal to that produced by one unit of the homologous protein, the homologous product being derived from the type of bacilli used to sensitize the test animals.

| | |
|--------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Group of guinea-pigs sensitized with human type tubercle bacillus; approx. test values obtained: | |
| One unit of human P.P.D. was equal to | <ul style="list-style-type: none"> 10 units avian P.P.D. 1 unit bovine P.P.D. 2 units B.C.G. P.P.D. 40 units johnin P.P.D. 100-200 units phlei P.P.D. |

| | |
|--------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Group of guinea-pigs sensitized with avian type tubercle bacillus; approx. test values obtained: | |
| One unit of avian P.P.D. was equal to | <ul style="list-style-type: none"> 20 units human P.P.D. 20-40 units bovine P.P.D. 20 units B.C.G. P.P.D. 5 units johnin P.P.D. 100-200 units phlei P.P.D. |

The result of these tests to date indicated that it might be possible, by applying the graded intradermal test with selected doses, to group infections into: (1) Mammalian, consisting of human, bovine, and variants of these two types; (2) avian which would include Johnes'; (3) the non-pathogenic group of acid-fast organisms.

Dr. C. L. Oakley said that the expectation that pasteurization could be abandoned when the incidence of tuberculosis in cattle was greatly reduced appeared to him illusory, as it would still be difficult to prevent the infection of milk during and after collection. He hoped that pasteurization would always be retained.

Mr. H. R. Tinney referred to remarks by Professor Dalling and other speakers concerning certain failures which occur in tuberculin testing.

He drew attention to an article by Dr. L. B. Bull in the *Australian Veterinary Journal* for April 1941 based on investigations carried out by Legg and Maunder in Queensland.

In one series of experiments 865 cattle were tuberculin tested, the results showing approximately 11% to be affected. These cattle were examined post mortem. 1.5% of non-reactors showed small lesions post mortem and 1.8% showed moderate to advanced or generalized lesions. Those animals showing lesions were divided into three categories: (a) Generalized cases; (b) those showing moderate to advanced lesions; (c) those with small lesions. Reactors in the above categories were (a) 68%, (b) 86.5%, and (c) 50%.

The speaker commented that no indication was given as to the character of the lesions and, therefore, the apparent poor results obtained in respect of (c) may possibly have been due to the inclusion of small, calcified abscesses. The results were alarming in so far as the most serious cases gave poor results on testing and further investigation was

undertaken at the instance of the Tuberculin Committee of the Australian Veterinary Association. Sera from 13 cattle with tuberculosis, but which had failed to react, were submitted to A. D. Campbell of the Animal Health Division, C.S. and I.R. 11 were from advanced cases and 2 from cases with early lesions. Campbell obtained a positive complement-fixation reaction from all the advanced cases. Of the other two, one gave a weak or doubtful reaction and the other was negative. Five other sera which were tested all gave positive reactions though three of these showed early lesions only.

Dr. Chalmers Watson asked if Professor Dalling had any grounds for thinking that any of the National Foods, now of necessity used for the feeding of dairy stock, might be proving definitely injurious by infecting the healthy stock in the Attested Herds of the country? He asked this very important question because it had come recently and acutely under the notice of the Scottish Association of Certified and T.T. Milk Producers.

A general observation that the speaker wanted to make had reference to the lack of confidence that was widespread in the industry at the present time in relation to the present tuberculin test used in the attested herd scheme, and to the urgent need for adequate steps being taken to restore speedily the confidence of the industry in the tuberculin test. It was unfortunate that the new test introduced in May 1940 had, with good reason, been the subject of so much serious criticism, largely as the result of its being prematurely introduced, without conforming to use and wont, by securing the interest and co-operation of herd owners and of the veterinary profession in relation to new legislative and administrative measures. It was, however, satisfactory to learn that before long a pronouncement from the Agricultural Research Council, with the co-operation of the Joint Tuberculosis Committee, would be forthcoming, this being essential for restoring the confidence of the industry in the value of the tuberculin test employed.

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DISCUSSION ON THE CONTROL OF TUBERCULOSIS IN CATTLE— (1) BY IMMUNIZATION; (2) BY ERADICATION OF INFECTED ANIMALS (Continued from December 17).

Mr. H. T. Matthews: There have been many attempts to create an immunity or to minimize the effects of bovine tuberculosis by artificial means. In general, vaccination with killed organisms has been found unsuccessful but claims are made for living vaccines modified in various ways. Von Behring used a human type, B.C.G. has received notice and attention is now being directed to the vole bacillus. Although reports conflict, there seems to be general agreement that resistance to infection can be enhanced and also that, even if infection should occur, its spread in the body can be impeded or arrested.

The oldest organized attempt to eradicate tuberculosis from infected herds is probably that known as Bang's method. It has been practised mainly in Denmark, Austria and Hungary and relies on the infrequency of congenital tuberculosis. It involves the segregation of calves from birth, so forming a new herd to replace the original within a few years. Its practicability has been demonstrated, even when only one set of premises is available and the adult herd heavily infected. Ostertag's system has been applied principally in Germany, Switzerland and Sweden and comprises elimination by slaughter of cases which are detected by clinical examination reinforced by laboratory test of specimens. It is reported that the application of the system has failed to bring about any reduction of bovine tuberculosis in Germany. The radical systems, based on the use of tuberculin with the elimination of reactors, have been employed extensively in America and Canada. In the United States, twenty-five years of intensive attack have resulted in a reduction of incidence from an average of 5% to under 0.5%. The Canadian plan is similar but rests more on voluntary than compulsory application. Incidence was rising from 3% in 1910 to over 7% in 1925. The scheme in its present form was initiated in 1922 and the incidence is now under 2%, although only about one-third of the total cattle are included in official measures yet. In Great Britain the Attested Herds Scheme is on a voluntary basis and deals only with individual herds.

There are three distinct methods of direct attack which could be applied singly or in combination: artificial immunization, separation of young stock from infective surroundings and the weeding out of infection by the use of tuberculin. Examination of conditions in Great Britain shows them to approximate in most respects to those prevalent in Europe rather than on the American continent and the immediate problem is how to reduce the disease in preparation for the more distant objective of total eradication. The stimulus to activity in the past has been the danger to human health, and much

good work has resulted, but we cannot be certain whether tuberculosis in cattle is greater or less than it was twenty-five years ago. The primary motive of the future will lie in agricultural economics, even though tuberculosis is not the most expensive disease to stockowners.

It is argued that, on the factor of comparative incidence alone, methods followed in America are not applicable here. The figure 40% has been quoted as representative of incidence in dairy cattle. There are districts in Great Britain where the incidence approximates to 100% and others where it is correspondingly low. The density of the cattle population ranges from under one hundred to over four hundred and fifty per thousand acres of cultivated land: it is highest in Cheshire and lowest in the Cambridge arable country. The proportion of dairy to total cattle ranges from under 20 to over 95%. The ratio of young stock to adults, dividing at 2 years old, varies from 20 to 60%. There is reason to believe that the distributional incidence of tuberculosis is not less variable. Using tuberculin as a diagnostic agent, Montgomerie and Rowlands found 11.8% reactors in a North Wales district, Thomas 2.6% in South Wales, Rabagliati 7.6% in a part of Yorkshire. The conception of a uniform 40% incidence as a total bar to the application of American methods is therefore misleading.

Discussion might be directed to considering the practicability and probable effects of applying three distinct methods of control simultaneously: in districts where the incidence is low, extension of the Attested Herds Scheme to areas on American lines, based on the use of tuberculin; in districts with medium incidence, the promotion of calf nurseries, either co-operative or State-owned, with the intention of replacing whole herds by new ones from the calves reared free from disease; in districts where the incidence is high, the use of living vaccines in an attempt to reduce the effects of disease and the number of grossly infective cases.

Dr. R. F. Montgomerie: The medical man who sees the tragic effects of bovine tuberculosis infection on the child population of this country must, naturally, tend to demand the eradication of tuberculosis from the cattle population. The destruction of all infected cows is an impossible programme. To have the desired effect slaughter would have to be completed within a relatively short period, otherwise the remaining infected animals would soon spread infection to hitherto clean cattle and the slaughter within a short space of time of any proportion of infected cows appropriate to progress in national eradication would affect the fertility of our land to a disastrous extent and reduce the amount of milk available for human and animal consumption to an impossibly low level. As the position is I do not see any hope of a policy of slaughter of tuberculin positive reactors being practicable in this country until, by other means, we have greatly reduced the incidence of the disease.

We can, however, segregate our infected animals from our clean and this policy has been in operation for some few years and is in my view the correct policy for this country.

The greatest hindrance to the establishment of tuberculosis-free herds is due to the apathy of the public in not demanding milk from tuberculosis-free herds and in not being prepared to pay a fair price for it. There are other lines of attack and I agree that vaccination holds promise and may become a potent weapon. I would, however, be interested to hear discussed what we expect of vaccination in tuberculosis. Until we know more about vaccinal reaction to the tuberculin test and can distinguish the vaccinated "clean" animal from the vaccinated "infected", I doubt very much the progress which will be made by vaccination if we have to accept that it will not prevent infection. One can also say that much more active and extensive work is still to be done before veterinary tuberculin and the test itself is in a satisfactory position. Despite its deficiencies and past imperfections, it has aided the eradication of tuberculosis from many herds in this country.

Dr. H. H. Green: In contributing to the adjourned discussion from the previous meeting it may be of interest to refer to the chemical nature of tuberculin and in particular to the purified protein derivatives (P.P.D.) which are now being advocated in place of the earlier heat-concentrated bacterial filtrates. "P.P.D." and "P.P.D. tuberculins" of sufficient purity for all diagnostic requirements are little more trouble to make than "heat concentrated tuberculins".

When *Mycobacterium tuberculosis* is grown in any suitable liquid medium protein derivatives find their way into solution along with other products of bacterial metabolism, while the medium itself undergoes various chemical changes. Koch himself held that the active principle of his "old tuberculin" was protein in nature although the difficulty of separating protein of bacterial origin from protein derivatives in the original broth rendered further investigation difficult. With, however, the introduction of protein-free synthetic media of the glycerine asparagin type any protein found after completion of

undertaken at the instance of the Tuberculin Committee of the Australian Veterinary Association. Sera from 13 cattle with tuberculosis, but which had failed to react, were submitted to A. D. Campbell of the Animal Health Division, C.S. and I.R. 11 were from advanced cases and 2 from cases with early lesions. Campbell obtained a positive complement-fixation reaction from all the advanced cases. Of the other two, one gave a weak or doubtful reaction and the other was negative. Five other sera which were tested all gave positive reactions though three of these showed early lesions only.

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In the series of specificity tests at Weybridge the guinea-pig has been used as the experimental animal but observations on the bovine subject have also been made and will be extended further.

As a broad preliminary generalization it may be stated that the "specificity factor" as between protein derivatives from strains of *M. tuberculosis* of mammalian origin is comparatively small—about 2. Thus with guinea-pigs sensitized to a human strain 2 units of P.P.D. from B.C.G. were sufficient to elicit the same intensity of skin reaction as 1 unit of P.P.D. from a mixture of three human strains. Human strains amongst themselves seem to show little or no specific difference. On the other hand the specific difference between any of the mammalian P.P.D.s and an avian P.P.D. or a johnin P.P.D. is comparatively wide, the factor being about 20 to 40; while the specific difference between either mammalian or avian and phleïn is much wider still, the factor being about 100 to 200. The specific difference between avian P.P.D. and johnin P.P.D. is again small, factor about 5.

The practical result of these observations is that comparative intracutaneous tests with specific purified protein derivatives readily differentiate between infections with mammalian and avian strains of tubercle in the guinea-pig and, in so far as present observations have gone, also in cattle. The specificity factor as between avian P.P.D. and johnin P.P.D. has not yet been determined on cattle but unless it is wider than the figure 5 shown in guinea-pig tests it may not prove easy to distinguish between avian tuberculosis and John's disease.

In actual practice the most important differentiation to make is that between bovine and avian infection of dairy cows. In many cases simultaneous comparison of two reaction sites with selected "average doses" of P.P.D. may be sufficient but the degree of certainty would be very much enhanced by comparing four standardized injections, i.e. two of each P.P.D. at widely divergent concentrations, so as to allow for the very wide variations in "allergic status" commonly encountered in individual members of a herd or in the same individual at different times.

Dr. H. J. Parish said he did not agree with Dr. Montgomerie when he blamed the apathy of the general public for the unsatisfactory state of our milk supply. If the dangers of tuberculous milk were fully appreciated throughout the country, the public would be prepared to pay for improved methods of control. If milk containing dangerous organisms had been sold to the public surely it was not the public but the Government departments concerned which were responsible. At the previous discussion the point made by the medical contributors was that the outstanding practical measure in the control of our infected milk supply was efficient pasteurization. The sale of infected milk surely ought to be a criminal offence, but unfortunately it was not so regarded.

Mr. H. W. Steele-Bodger said that for a number of years he had persuaded his clients to create tubercle-free herds; there had been no difficulty in getting the herds free but it had been found impracticable and uneconomic to keep them free; there were so many limiting factors outside the control of the farmer. All animals imported into these tubercle-free herds had come from free areas but invariably they were the first to succumb to infection and became reactors to the tuberculin test, usually after an interval of six months. There were too many heavily infected areas in this country to be restocked from the free areas. He urged that methods of immunization should be more fully investigated and if found to be sufficiently reliable, a policy of immunization should be adopted, at least in the heavily infected areas.

In 1927 he commenced vaccinating with B.C.G. in some 30 herds, but the number had, for various reasons, dwindled. The total number of vaccinated calves he had done was 1,538 with 3,170 re-vaccinations—a total of 4,708. He had made only 30 post-mortem examinations himself; of these 22 were entirely negative, three showed calcified lesions in the glands and five were advanced. These figures might appear far from satisfactory but so were the conditions under which these field trials were conducted. Only the subcutaneous route was utilized in immunizing the calves. No effort was made to keep them in isolation either before or after they were injected. It had proved very difficult to follow many of the vaccinated animals to the slaughterhouse though he had received, but treated with reserve, a great deal of information from sanitary inspectors, butchers and others as to the freedom from tubercle infection of vaccinated stock.

Lieut.-Colonel H. A. Reid said that he could not agree with the statement of a previous speaker that failure to lead public opinion had accounted for many of the present difficulties. It was not the fault of the medical or veterinary professions who with the help of the press had done all that was possible to educate the public on the subject of tuberculosis. The belief in the hereditary nature of tuberculosis was still widely held. Because milk looked clean and wholesome people did not hesitate to consume it in the

bacterial growth must necessarily be of bacterial origin and Seibert showed that several soluble proteins were present in the filtrate from the bacterial debris, all of which had tuberculin activity, while any non-protein constituents were not specifically active although they might show certain non-specific toxic properties.

The proteins of highest molecular weight were coagulated by heat and were antigenic, while those not coagulated by heat were of lower molecular weight and so feebly antigenic as to cause no complications in the routine intradermal test. Because of the method of heat concentration these simpler protein derivatives were the "active principle" of the older "tuberculins".

Knowing these facts the preparation of P.P.D. tuberculins of sufficient purity for all practical purposes becomes simple. After growth of any desired strain in any suitable synthetic medium is completed, the culture flasks are heated to kill the organisms and throw down the unwanted coagulable protein. This coagulated fraction remains with the bacterial bodies on filtering, so that the filtrate contains only the "heat-denatured non-coagulable non-antigenic soluble protein derivatives". If to the clear bacterial filtrate any good protein precipitant is added, this "P.D." is thrown out. Trichloroacetic acid is used at a concentration of about 5%, partly because it effects complete precipitation and partly because it is very easy to get rid of in subsequent purification to "P.P.D." Most of the polysaccharide associated with the protein is split off by the trichloroacetic acid although, at the pH concerned, some of the nucleic acid in the medium comes down as "protein nucleate". If the precipitate is washed a few times with dilute trichloroacetic acid the other non-specific constituents of the medium are removed and the trichloroacetic acid itself can then be washed out with water, either plain or buffered around the isoelectric point of the protein. This leaves a wet P.P.D. which can be redissolved in dilute soda, brought to approximate neutrality with phosphoric acid, so providing a "phosphate buffer", standardized to any desired strength by Kjeldahl N. determination, fortified with glycerine as stabilizer and phenol as preservative, and issued as "P.P.D. tuberculin". If so desired, allowance can be made for the nitrogen present as nucleic acid by calculation from the phosphorus content. If the P.P.D. is wanted in the dry state the trichloroacetic-washed precipitate is triturated with successive portions of anhydrous ether, which removes both trichloroacetic acid and "wetting" water, and finally dried in a vacuum desiccator. The dry product so obtained usually contains about 90% of true tuberculo-protein derivative, the remainder consisting of nucleic acid, polysaccharide, and residual moisture. If a higher degree of purification is required the "P.P.D.", while still at the wet stage, can be redissolved in dilute alkali, neutralized, and precipitated with ammonium sulphate. Very little nucleic acid is precipitated around the neutral point and this component, along with most of the residual polysaccharide, is so got rid of. If desired this precipitation can be repeated several times, the ammonium sulphate dialysed away and the product dried. In her latest technique Seibert reprecipitates seven times but even this leaves traces of nucleic acid and polysaccharide. For commercial biological purposes this more extensive purification is unnecessary, although of course desirable for subsequent studies on the chemistry of the proteins concerned.

No mention has been made of the "ultrafiltration washing and concentration" of Seibert. Such a technique is useful for concentration purposes but is not an essential step in the preparation of a sufficiently pure "reconstituted P.P.D. tuberculin". It saves trichloroacetic acid but is more troublesome, and in large-scale production for veterinary requirements would be used or omitted according to the economics of the matter.

"P.P.D.s" of various acid-fast organisms have been prepared in the biochemical department at Weybridge and tested for specificity in the tuberculin department. The series included products from human, bovine, and avian strains of *M. tuberculosis*, from B.C.G., from Johne's bacillus and from *M. phlei*. Since these keep well in the dry state they also serve as provisional standards for potency tests on any other products subsequently prepared.

In testing any given "tuberculin" it is obviously necessary to distinguish between "potency" and "specificity" and in doing so the following definition of terms has been adopted:

Potency factor.—The number of units of the unknown preparation, in prescribed volume of test dose, required to elicit the same skin reaction as one unit of a selected reference standard (P.P.D.) in specified experimental animals sensitized to a bacterial strain homologous to that from which the standard was prepared.

Specificity factor.—The number of units of heterologous protein derivative required to give the same intensity of skin reaction as one unit of homologous protein derivative, i.e. homologous to the species of acid-fast used to sensitize the experimental animal.

In the case of "purified protein derivatives" which can be weighed out in the dry state comparison is of course made direct. In the case of solutions the protein derivative can be determined analytically and the figure used for calculation on a protein basis.

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raw state and give it to children. His attempts some years ago in New Zealand to supply clean, tubercle-free milk produced under modern hygienic conditions had not been encouraging and had resulted in financial loss.

Dr. J. T. Edwards (Pirbright) reverted to the experiences related by Colonel Rees-Mogg at the close of the preceding meeting from which the conclusions were drawn that B.C.G. vaccination proved unavailing to diminish the incidence and intensity of tuberculosis in a herd of Friesian cattle. Experiences of that kind needed careful examination. McFadyean and his associates (*J. Comp. Path. & Therap.*, 1901, 14, 136; *ibid.*, 1913, 26, 327) repeated von Behring's work and compared it with vaccination in a similar way with tubercle bacilli of the avian type, inasmuch as the excretion of live human bacilli was anticipated to be fraught with obvious danger to the human consumer of milk from the vaccinated cattle. The conclusions drawn that "by the intravenous inoculation of avian bacilli it is possible to confer on healthy calves a markedly increased power of resistance to infection with bacilli of the bovine type" seemed to have settled in principle the reality of vaccination. Nevertheless (unpublished results) the later findings that intravenous injection with some strains of avian tubercle bacilli was not always safe for cattle, together with other obvious objections to the method (such as infection of poultry with the excreted bacilli) did not afterwards commend it for adoption in practice. The researches of Wells and Brooke on guinea-pigs and the preliminary experiments of Dalling and Griffith on cattle, with the vole bacillus, renders the situation still more hopeful. In comparison, despite its almost complete innocuousness for cattle and man, B.C.G. would seem on the whole to constitute a rather weak antigen. Reviewing the published evidence, and taking into account his personal experiences the following conditions appeared to be essential for the success of vaccination of cattle against the bovine tubercle bacillus: (1) Vaccination must be adopted only on animals that are free from the natural infection. In naturally heavily infected herds, this would mean that vaccination should be undertaken only on newly born calves. (2) A period, which may prove to extend to three months (unpublished personal observations) must elapse after vaccination before the calves are allowed to become exposed to natural infection. (3) The immunity appears to wane quickly after the vaccinating bacilli are excreted from the animal's system, and so vaccination needs to be repeated at intervals not exceeding about nine months. (4) The immunity is not a very powerful one, and so the animals need to be excluded throughout life from risks of massive infection.

While expressing much hope for the chances of vaccination in selected circumstances, well-thought-out trials on a field scale should first be carried out before the method was adopted in general practice.

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Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

[March 27, 1942]

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Historical note.—Naegeli's classical post-mortem investigations (1900) in Switzerland showed that nearly all adults had been infected with tuberculosis. The Pirquet test appeared to confirm these findings as 95% of Viennese children were positive to the test at the age of 13. It was admitted that in country districts the incidence of infection might be lower, but it was generally considered that by adolescence nearly everyone had been primarily infected. Pulmonary tuberculosis in adults, spreading by the bronchi (and therefore suitably termed bronchogenic tuberculosis), was believed to have its origin in renewed activity of tubercle bacilli in the primary lesions or in those that immediately followed the primary infection in children. Later it was added that this bronchogenic tuberculosis might arise also as a new exogenous infection which remained confined to the lung owing to the allergy that followed primary infection. The greater incidence of tuberculosis among adult home-contacts than in the general population was adduced as evidence in support of this view.

The tuberculosis seen during the first world war in soldiers brought to Europe from communities among whom tuberculosis was not endemic, showed that primary infection did occur in adults, and that it could assume a generalized and rapid form. This was explained as due to the fact that, unlike Europeans, these natives had had no opportunity of being primarily infected in childhood, and therefore of developing the immunity that followed such infection. Actually, as was shown in connexion with South Africans on the Rand, this was not the true explanation, but in any case the above phenomenon was not related to European adolescents, though as early as 1919 Ghon and Pototschnig reported finding recent primary infection in adults post mortem. This finding was considered exceptional. And it was not until 1927, when Heimbeck in Oslo published his work showing that a large proportion of student nurses were tuberculin-negative, that primary tuberculosis in adults began to attract attention. Accurate pathological evidence was provided by Ragnotti (1930) who, among 4,000 consecutive post-mortem examinations at all ages in Berlin, found 36 cases of recent primary complex, in adolescents and adults, both pulmonary and intestinal, the largest number occurring in the age-group 20-25. During the last twenty years publications have multiplied enormously, although apart from the excellent monograph by Frimann-Dahl and Waaler of Oslo (1936), little has been added on the pathological aspect.

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France, where unfortunately the Pirquet test is still extensively relied on, and from South American countries. The best investigation is undoubtedly that of Malmros and Hedvall (1938) among students and nurses in Lund, Sweden.

Perusal of the literature shows that in some countries a large proportion of the population reach adolescence without having been infected with tuberculosis, and that primary infection occurs during adolescence and adult life following contact. Granting this, several questions at once present themselves: (1) What is the incidence of tuberculous infection when adolescence is reached? At what rate does primary infection occur later? On what factors does this rate depend? (2) Are the morbid anatomical lesions of primary infection the same in adolescents and adults as in children? Is the subsequent morbid anatomical evolution the same as in children? (3) What are the clinical, including radiological, manifestations of primary tuberculosis in adolescents and adults? Is the subsequent clinical course the same as in children? (4) What are the epidemiological implications? How far will the mortality in different age-groups be affected by the fact that more and more people receive their first infection in adolescence? I shall attempt to answer these questions very briefly on the basis of published work in other countries (since unfortunately none is available here), and I have collected representative samples in the following table, ignoring those investigations in which the Pirquet test alone was used:

Incidence of Tuberculous Infection in Adolescents

TABLE I.

| Author | District | Material: character and number | Tuberculin test | Percentage negative |
|----------------------------------|---------------------------|--------------------------------------------------|-----------------------------|-----------------------------------------------------------------------------------|
| Arborelius (1930) | Sweden | 2,230 recruits, mainly aged 19-21 | Mantoux 1 mg. | 34.2% from country; 4.8% from Stockholm |
| Holm (1940) | Denmark | 1,500 medical students | Mantoux P.P.D. 2nd strength | 33% |
| Soper and Wilson (1932) | Yale University U.S.A. | 3,000 undergraduates | Mantoux 1 mg. | 45.5% |
| Long and Seibert (1937) | U.S.A. | College entrants in various parts of the country | Mantoux P.P.D. 2nd strength | From 20% to 80%: high negative rate in Central States, low rate in Eastern States |
| Stiehm (1939) | Univ. of Wisconsin U.S.A. | Students: (1) aged 16 (2) aged 20 | Mantoux P.P.D. 2nd strength | 81% 73% |
| J. A. Myers <i>et al.</i> (1940) | Minneapolis U.S.A. | Student nurses and education students (entrants) | Mantoux 1 mg. | 70% to 85% according to school |
| Israel <i>et al.</i> (1941) | Philadelphia | 643 nursing students, aged 17-21 | Mantoux P.P.D. 2nd strength | 43% |
| Sayago and Casco (1939) | Argentina | 279 students, aged 19-22 | Mantoux | 21% |
| Vaja (1939) | Roumania | College students, aged 18 | Mantoux | 46% |

From these and other published studies it is evident that while in some urban localities the percentage of infected people may be as high as 80% at the age of 20, in many it is no higher than 40%, and may be as low as 20%. These figures will clearly be lower at the age of 16, the onset of adolescence. The incidence of infection at this age varies directly with density of population and height of tuberculosis mortality. Repeated tuberculin testing in the same district after an interval of a few years, for instance, in Norway (Ustvedt, 1932), in Holland (Heynsius van den Berg, 1934), and in America (Tenth Annual Report of the Tuberculosis Committee, American Student Health Association, 1939-40, *Journal-Lancet*, 1941, 61, 115), suggests that the incidence of infection has fallen in recent years with the fall in tuberculosis mortality. Thus data obtained from 166 colleges in America show that the percentage of students tuberculin positive has more or less gradually fallen from 35% in 1932-33 to 25% in 1939-40. A fall in rate of infection during the past twenty-five years has also been clearly demonstrated in the morbid anatomical material of Frimann-Dahl and Waaler (1936) in Oslo, compared with that of Harbitz in 1904; and in the post-mortem investigation of Uehlinger and Blangey (1937) at Zurich, compared with those of Naegeli in 1900. It may therefore be concluded that at least half the people in civilized communities first become infected with tuberculosis after adolescence has been reached. The rate at which infection occurs from then on appears to depend on opportunities for contact with the disease. Published investigations suggest that most of the late primary infections occur between 16 and 30. While among the general population the increase in incidence of infection is more or less gradual, among certain groups exposed to special risks the increase may be very rapid. Thus medical students and nurses previously tuberculin-negative are usually all infected before qualification, and half of them may become tuberculin-positive within a few months when working in tuberculosis wards. The investigations of Stewart *et al.* (1939) have shown a very striking difference between the rate of increase in infection among the above groups and that among other college students. Thus, allowing for length of exposure, the attack rate was among medical students attending a two weeks' tuberculosis

service, twenty-eight times that among students of a college of education. Similarly, the attack rate among nurses attending a six weeks' tuberculosis service was twelve times that among those not attending such a course. Hedvall (1940) gives similar findings among student nurses at Lund, Sweden, and shows that the greatest conversion rate of tuberculin reaction among medical students occurs during the course in morbid anatomy.

It would be inadvisable to apply too closely the findings from one country to another. One must therefore regret that so far no related investigations have been published in this country. There are indeed only three investigations on the incidence of infection among non-tuberculous people, and these deal with relatively small numbers, and only with hospital patients; moreover they were carried out eight to thirteen years ago (Hart, 1929; Dow and Lloyd, 1931; Kayne, 1934). They do suggest however that ten years ago an appreciable proportion of adolescents had not been primarily infected.

Quite recently figures have been published, though again concerning small numbers, by Crowe (1942) from Eire. The tests were carried out in the County of Laoighis, where no less than 56% were found negative to a complete tuberculin test among the age-group 15-20, and even 33% among the age-group of 20 and over

Morbid Anatomical Note

In an adolescent or adult the primary lesions are similar to those that develop in children. But there are differences: unlike in children, caseation in the corresponding hilar glands may be less extensive than in the pulmonary focus; and presumably because of this, epituberculous lesions, so common in childhood, and due either to collapse or to perifocal inflammation, are seldom observed. The evolution of the disease differs but little from that seen in childhood, from its most favourable outcome—calcification of the primary complex—to the unfavourable disseminations, both acute and chronic. There is one difference. In adolescents and adults, within a comparatively short time of the apparent healing of the primary complex, whether insignificant or extensive, lesions typical of bronchogenic tuberculosis may develop. This has been demonstrated in all relevant morbid anatomical material available. This fact cannot be overstressed, since it is evident that when the primary lesions are insignificant and not demonstrated clinically, the onset of lesions of the bronchogenic type within some months of the change in tuberculin reaction, might be regarded by the clinician as a form of the primary lesion itself. This indeed has recently been assumed by some American physicians (*see below*).

Clinical Manifestations

In order to study these with accuracy, much published work is of little value. Only such investigations should be considered in which certain criteria have been adopted. These are: (1) a complete tuberculin test, if negative, should be repeated *at least once* a year (preferably every six months); (2) a full-sized radiograph should be taken when the tuberculin reaction becomes positive, and repeated at short intervals; and (3) a film, taken before the tuberculin reaction became positive, should be available for comparison. The need for a *complete* tuberculin test should be obvious; and stress must be laid on the criteria adopted in regarding a reaction as positive—these must not be fixed too low. Repetition at short intervals is essential; otherwise difficulty will arise in dating clinical manifestations. Screening is not reliable enough for the detection of very small lesions; and a film taken when the tuberculin test was negative, for comparison, is necessary in order to appreciate slight enlargement of the hilar shadows. The investigation that fulfils these requirements best is that of Malmros and Hedvall from Lund, Sweden, and I shall, therefore, briefly summarize their findings among students and nurses.

Among those who became tuberculin-positive during the period of observation—a total of 151—only 47, representing 31%, showed clinical manifestations. These were all aged between 20 and 25; and the incidence of manifestations was much higher among medical students and nurses than among students of other faculties. The 47 clinical manifestations included:

TABLE II.

| | | | | | | | | | |
|-------------------------------------------|-----|-----|-------|----|--------------------------------------------------|-----|-----|----|----|
| Erythema nodosum alone | ... | ... | in | 3 | Pleural effusion alone | ... | ... | in | 6 |
| Erythema nodosum and primary complex* | ... | ... | in | 6 | Glands in neck or abdomen alone | ... | ... | in | 2 |
| Phlyctenular conjunc. and primary complex | ... | ... | in | 1 | Miliary tuberculosis alone | ... | ... | in | 1 |
| Primary complex alone | ... | ... | in | 14 | Pulmonary foci alone ("subprimary initial foci") | ... | ... | in | 14 |
| | | | Total | 47 | | | | | |

* "Primary complex" implies either (radiological) enlargement of hilar shadows alone or with a pulmonary focus corresponding to the hilar enlargement.

Thus a primary complex was noted in only 21 of the 47 students and nurses, but it may be assumed that it was also present in the remainder though not big enough or in such a position as to show in a radiograph—as indeed must have been the case in the 104 students and nurses who became tuberculin-positive without associated symptoms or signs.

All the patients with erythema nodosum were women; later one developed a pleural effusion and 3 bronchogenic tuberculosis, of whom one was given an artificial pneumothorax and another developed peritonitis and died.

Of the 21 students and nurses with a primary complex, 13 showed typical pulmonary and hilar components. In 7, only hilar enlargement was seen. Among the 21 patients with a primary complex, 5 later developed a pleural effusion and 4 bronchogenic tuberculosis; the average interval between the appearance of the primary complex and the pleural effusion was six months, and the average interval between the primary complex and bronchogenic tuberculosis was twelve months. The bronchogenic tuberculosis developed as a fresh focus or foci in the apex or upper zone, and not from the foci of the primary complex.

The average time between primary complex and pleural effusion was six months; between the finding of a negative tuberculin test and a pleural effusion it was ten months; one may therefore justifiably assume that even in the students and nurses with a pleural effusion *ab initio* a primary complex had previously been present, and that the pleural effusion did not represent the first tuberculous lesion.

Of special interest are the 14 young people in whom foci were seen in the upper part of the lung without a preceding primary complex. These foci were always seen in the apex or upper zone and were uni- or bilateral. On an average they were noted fourteen months after the last finding of a negative reaction, and on an average twelve and a half months after a normal radiogram. Note that in addition such foci were observed in 5 people after erythema nodosum or a primary complex, the average interval being eleven and a half months. It may therefore again be assumed that these foci did not represent the primary lesion, and that they arose a short time after the development of a primary complex which had remained unrecognized. Their origin is most likely hæmatogenous. That these foci represent the initial lesion of a bronchogenic tuberculosis may be seen from the follow-up of the 19 students and nurses with such foci. One died of the disease, another 6 later required pneumothorax treatment (bilateral in 2 cases), in another progression occurred followed by quiescence. Malmros and Hedvall called these foci "subprimary initial foci", but there seems no reason for separating them from bronchogenic tuberculosis, and indeed similar foci developed among students and nurses who when first seen were tuberculin-positive.

The symptoms associated with the radiological findings in the 47 people were: In those exhibiting erythema nodosum with or without primary complex, fever, cough, and pain in the side; the sedimentation rate was nearly always raised. When a primary complex alone was seen, there was generally fever, cough, fatigue, pain in the chest, catarrhal symptoms, and a raised sedimentation rate; nevertheless, 4 of them remained quite well and showed a normal sedimentation rate although in 2 there was much hilar enlargement. On the other hand, the onset of the subprimary initial foci were usually associated with no symptoms and a normal sedimentation rate. When symptoms did occur they were "flu", cough, fatigue, fever and hæmoptyses. In most of the students and nurses the lesions were detected only as the result of systematic radiograms.

Similar clinical pictures are obtained in this country. Before, however, presenting my own cases I should like to mention American workers who claim different findings. Some of these studies are not as reliable as that of Malmros and Hedvall because often the tuberculin tests were carried out at too long intervals, often only screening was the routine radiological procedure, or no film was taken before the tuberculin test became positive. But it must be admitted that even allowing for these facts, the clinical manifestations in America might differ from those in other countries. Israel, Hetherington and Ord (1941) from Philadelphia, in their follow-up of 1,643 student nurses found that among those tuberculin-negative, 48% became positive at the end of four months, and 86% at the end of the year: they report that there were only slight differences between those originally tuberculin-negative and those tuberculin-positive, in regard to incidence, anatomical character and distribution of the lesions, as determined by fluoroscopy; the subsequent clinical course, too, showed no difference. This may well be because many of the recently primarily infected nurses had developed a bronchogenic lesion within a relatively short period of the primary infection. Israel and his co-workers state that no serious illness and no deaths occurred among the nurses, but this good outcome is put down at least in part to prompt treatment. They note that the development of a positive tuberculin reaction was associated with a high incidence of non-specific gastro-intestinal, febrile, and vague toxic illness, and with an especially high frequency of abdominal symptoms simulating appendicitis. The findings of Israel and his co-workers contrast sharply with those in Denmark, where Holm (1940) found that of 1,500 medical students who were followed up for three years, none broke down with tuberculosis during the three

years among the 1,000 positive reactors, while among the 500 negative reactors, of whom 81 became tuberculin-positive, 19 developed active pulmonary tuberculosis and 3 died.

In contrast to Israel and his co-workers, Myers and his colleagues (1937) classify manifestations following a change in tuberculin reaction amongst a large number of students and nurses into four groups: (1) Erythema nodosum occurred in 2 male medical students; (2) 21 students and nurses developed a "primary" focus in the lung. Myers and his co-workers admit that in some of these it was impossible to determine with certainty that the shadow represented only the first infection type of tuberculosis because the tuberculin test was not always done with sufficient frequency; (3) 18 students and nurses developed a pleural effusion; a "primary parenchymal lesion" was previously demonstrated by X-ray in some but not in others; (4) in 25 students and nurses a change of tuberculin reaction was followed by clinical pulmonary tuberculosis later, with or without a demonstrable primary focus or pleural effusion in the interval. The authors state that in the majority of these patients sufficient time had elapsed between the finding of a positive reaction and the development of a lesion to assume with a reasonable degree of certainty that the lesion was of the reinfection type.

The findings of Myers and his co-workers do not really differ greatly from those of Malmros and Hedvall, and hardly fit the suggestion put forward by Israel and Long (1941) that while in Scandinavian countries people being less naturally resistant react to a primary infection like children, North Americans being more resistant react not by forming a primary complex but by developing a lesion corresponding to the first focus of bronchogenic tuberculosis. I feel that these authors have tended to disregard morbid anatomical evidence, and have been influenced by the experimental work of Lurie (1941) on resistant and susceptible rabbit families obtained by inter-breeding to a degree quite unknown among civilized races.

Here is a summary of my own cases. I am not able to give the crucial proof of primary tuberculosis since in dispensary practice it is rarely possible to carry out periodic tuberculin testing of adolescents and adults, but in the light of published work and taking into account all the circumstances, I have felt justified in applying in each case the diagnosis of primary tuberculosis.

TABLE III.—FEMALES.

| Name | Age | Contact | Symptoms | X-ray | S.R. | Course |
|--------------|-----|---------|-------------------------------------|----------------------------------------------------------------------------------------------|------|------------------------------------------------------------|
| * (1) E.B. | 16 | No | Cough, lost weight | Left hilum + | 27 | Lt. pleural effusion 10 weeks later, then to sanatorium |
| (2) M.H. | 16 | No | Eryth. nodosum | Not known, but 1 year later, calcified primary complex on rt. | ? | Rt. pleural effusion 2 months later, then to sanatorium |
| * (3) J.I.N. | 16 | Yes | None | Lower pole of rt. hilum +, and focus in field near it (radiograph neg. 3 months before) | 4 | To sanatorium |
| * (4) E.C. | 17 | No | Eryth. nodosum, 6 weeks later cough | Lt. hilum +; 1 yr. later, calcification in lateral radiograph, where previously dense shadow | 12 | To sanatorium |
| * (5) E.L.C. | 17 | No | Eryth. nodosum, pyrexia | Rt. hilum +, focus in rt. lower zone | ? | 6 months later, right pleural effusion, then to sanatorium |
| (6) D.R. | 17 | No | Eryth. nodosum | N.a.d. (Patch tuberculin test neg., but + when tested 1 mth. later) | 2 | Under observation |
| * (7) J.E. | 18 | Yes | Sore throat | Rt. hilum +, focus in lower zone (5 months later, not visible) | 8 | To sanatorium |
| (8) D.W. | 18 | Yes | None | Upper pole of rt. hilum + and mottling near it; 6 months later, calcification in this area | 2 | To sanatorium |
| * (9) D.C. | 19 | No | "Flu," sputum staining | Focus below rt. clavicle; 9 months later, calcification | 18 | To sanatorium |
| * (10) M.H. | 21 | No | Nausea, weakness, cough | Both hila + (Gastric contents + for tubercle bacilli) | 14 | Refused sanatorium |
| * (11) M.W. | 21 | Yes | Fatigue, later hæmoptysis, pyrexia | Rt. hilum +, focus 4th inter-space | 22 | To sanatorium |
| * (12) C.H. | 24 | No | Eryth. nodosum, abdom. pain | Both hila + | ? | To sanatorium |

TABLE IV.—MALES.

| Name | Age | Contact | Symptoms | X-ray | S.R. | Course |
|------------|-----|---------|----------------------------------------|------------------------------------------------------------------|------|-----------------------------------------------------------------------------|
| (1) M.D. | 16 | No | Pain in chest, giddiness; later, cough | Rt. hilum + 9 weeks after onset; apparently not + 5 weeks before | ? | 1 year after onset, mottling both upper zones with cavity left apex; T.B. + |
| (2) I.G. | 16 | No | Fatigue | Rt. hilum +, focus right lower zone | ? | Kept under observation |
| * (3) P.L. | 16 | Yes | "Flu" | Lt. hilum +, mottling in left mid-zone | 18 | Sanat. : at bronchoscopy, tub. vegetations in left main bronchus |
| * (4) D.M. | 16 | No | "Flu" | Rt. hilum +, mottling in rt. lower zone | 8 | Sanat. : at bronchoscopy, tub. vegetations in right main bronchus |

TABLE IV.—MALES (contd.).

| Name | Age | Contact | Symptoms | X-ray | S.R. | Course |
|--------------|-----|---------|------------------|-------------------------------------------------------------------------------------------------|------|-----------------------------------------------------------------------------|
| * (5) E.P. | 16 | No | Malaise, pyrexia | Rt. tracheobronchial glands +, and lt. hilum + | ? | Lt. pleural effusion 6 weeks later; bilateral apical cavity 17 months later |
| * (6) H.C.H. | 17 | No | Eryth. nodosum | Both hila +, mottling near rt. lower pole | ? | Refused to attend again |
| * (7) D.H. | 23 | Yes | "Cold" | Both hila +; normal 2 months later; calcification appeared upper pole of rt. hilum 2 yrs. later | 2 | Sanat.: at bronchoscopy, anterior wall of lt. bronchus showed pressure |
| (8) F.P. | 24 | No | Eryth. nodosum | ? | ? | 3 months later, rt. pleural effusion |

[Radiograms (slides) of the cases marked with an asterisk (*) in Tables III and IV were then demonstrated.]

TABLE V.—SUMMARY OF 20 CASES (TABLES III AND IV).

Contact established in 6 only.

Main initial symptom: erythema nodosum in 7. Fatigue in 3. "Flu" in 3. Cough in 1. Sore throat in 1. "Cold" in 1. Malaise and pyrexia in 1. Pain in chest and giddiness in 1. None in 2 (contacts).

Other symptoms: staining or hemoptysis in 2. Abdominal pain in 1.

Sedimentation rate raised in 8 (out of 12 only).

X-ray: enlarged hilar shadow alone in 7. Enlarged hilar shadow and parenchymatous focus in 9. Parenchymatous focus alone in 1. No changes in 1. Not known in 2.

EPIDEMIOLOGICAL IMPLICATIONS

We must agree that a large proportion of children do not become infected with tuberculosis, a proportion that is, or at all events, before the war was, increasing; and that therefore more and more people are first infected as adolescents or adults. Primary infection occurring at any age weeds out the most susceptible people who die from the primary infection or its sequelæ. In times when primary infection occurs in childhood, adolescents and adults who have overcome this infection represent a more resistant group, and may develop bronchogenic tuberculosis following fresh contact or endogenous exacerbation when resistance is low. But when primary infection increasingly occurs in adolescence and adult life, more and more susceptibles are first infected at this age, and we may therefore expect more and more adults to die of disseminated forms. Those more resistant, on the other hand, are in the same position as adults surviving primary infection in childhood, since bronchogenic tuberculosis may follow soon after primary infection. To what extent do these facts account for the comparatively low tuberculosis mortality of older children? Contact in the home may play a predominant role in early childhood, whereas contact outside the home is not likely to be of great importance until adolescence is reached following the beginning of employment and just at the time when great demands are made on bodily resistance. These facts might also account at least in part for the retardation in the drop of mortality among young women noted since 1900. For the fall in the general tuberculosis mortality and morbidity would have the effect of reducing infection and mortality in childhood at the expense of exposing an increasing proportion of susceptible young adults to primary infection. This effect is likely to be more marked in young women because firstly, the incidence of infection among females is generally lower than among males of the same age, presumably because they are kept more indoors, and secondly, because since 1900 young women have increasingly entered industrial employment. The recent appreciable increase in deaths from tuberculous meningitis among adolescents and young adults supports this view.

Discussions on endogenous and exogenous reinfections are largely academic. For when there are many uninfected adolescents, and when bronchogenic tuberculosis may follow soon after the primary infection, exogenous infection as a cause of bronchogenic tuberculosis plays an essential role. Thus both contact and lowered resistance are important in the control of the disease. We must, therefore, continue to stress the importance of reducing the human tubercle bacilli reservoir not only in homes but in crowded communities by the detection of symptomless disease through mass radiography, of adequate and immediate facilities for treatment, and of the provision of institutional accommodation for bad and good chronic cases.

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Dr. W. Pagel: Tubercle bacilli infecting the human body for the first time produce characteristic changes at the site of entry, the primary focus. This is soon followed by a similar lesion in the corresponding lymph glands. The primary focus at the portal of entry together with the glandular changes is called the "primary complex". It is commonly observed in the lungs or in the digestive tract of children dying of generalized tuberculosis. The common type of adult pulmonary tuberculosis (to be referred to in future as "bronchogenic tuberculosis") is a *post-primary* condition, distinct from the primary complex and its early sequelæ. In bronchogenic tuberculosis the hilar glands are devoid of caseous changes, and the primary complex is usually found in an inactive calcified or ossified state.

An inactive primary complex is found in a large proportion of healthy individuals under 18. Of late, however, the incidence of tuberculous infection in the general population has undergone a remarkable change: only 20% (as against 60% before) show evidence of primary infection up to the age of 18, while high figures up to 100% are only reached in the highest age-groups (Uehlinger and Blangey, 1937). It is therefore to be expected that a larger proportion of adults than before should show evidence of fresh primary infection.

It has been said that the primary complex is essentially characteristic of the first infection in childhood and that when such an infection occurs in adult life only the primary focus develops, the lymph glands being unaffected. This was explained by the assumption that the lymphatic glands of adults possess a high resistance to infection. When, as rarely happened, a fully developed primary complex was seen in adults, it was supposed that a first infection had occurred in individuals with little or no natural resistance. In other words formation of the primary complex is said to depend on age and natural resistance and not upon the fact of first infection alone. This theory is based on experiments made by Lurie (1938) who succeeded in breeding strains of rabbits with high inborn resistance to tuberculosis. Primary infection by inhalation was followed, in these rabbits, by the formation of a primary lesion in the lung but not by a primary complex. The primary lung focus developed at once into a cavity with subsequent bronchogenic spread restricted to the lungs. In other words, bronchogenic tuberculosis was reproduced by a primary infection occurring in animals with high natural resistance to tuberculosis.

In considering primary tuberculosis in adults from the pathological side, the following questions arise:

- (1) Can pathological evidence be adduced for the occurrence of primary tuberculosis in the adult? (2) What are its characteristic features? (3) How often does it occur? (4) Are its characteristics due to the fact that it is the first manifestation of tuberculosis in the individual or is there evidence of an influence of age or natural resistance?

(1) Occurrence of Primary Infection in Adults. Personal Observations in Necropsies (1938-1942)

Case I (Hounslow Chest Clinic).—Boy, aged 16, exposed to home contact. Developed a transient infiltration in the right upper lobe, followed by enlargement of the hilar glands. Death from toxæmia. *Necropsy*: Large caseous infiltration near the base of the right lower lobe. Extensive caseation and partial hæmorrhagic liquefaction of the lymph glands at the right hilum.

Case II (figs. 1, 2, 3). (Central Middlesex County Hospital, P.M. 42/27).—Male, aged 17. No history of contact. Admitted for typical tuberculous meningitis. Skiagram of chest showed enlargement of the left hilum and a small doubtful area in the left infracalicular region. *Post-mortem*: Shallow, longitudinal ulcer 3×2 cm. at the posterior wall of the *bulbus duodeni*. Gross enlargement and complete caseation of the peri-

portal, peri-pancreatic and retro-duodenal lymph glands. Other parts of the intestine and mesenteric lymph glands free. Lungs: 2 cm. below the apex of the left lung a subpleural quadrangular caseous and partly liquefied focus, 1.5 cm. in diameter. Hilar lymph glands enlarged on the left side but containing small caseous specks on both sides, these being very marked in the lower intrapulmonary posterior mediastinal glands on the right side. Right lung free of tuberculous changes. Smears from retroduodenal lymph glands show large numbers of *short acid-fast bacilli*. Typing of the bacillary strain by animal inoculation is still in progress. Exudative tuberculous meningitis.

The compact caseation of the duodenal (portal) lymph glands suggests the duodenum as the entrance site of the real primary complex. Caseation of the mediastinal lymph glands was less intense than that of the upper abdominal glands and could be interpreted as an extension of the abdominal glandular changes, the focus in the left upper lobe being due to an independent blood-borne spread. This interpretation would be certain if we could succeed in recovering a bovine strain of tubercle bacilli. The shape of the bacilli seen makes bovine infection likely. The possibility of a double primary infection—pulmonary and intestinal—cannot however be excluded.

Case III (C.M.C.H., P.M. 40/313).—Female, aged 18. No family history of tuberculosis. Admitted with typical tuberculous meningitis. Lungs: Round caseous focus 1 cm. in diameter, about 1 cm. below the pleura, in the infraclavicular parts of the right upper lobe. Similar focus, 1 cm. in diameter, in one right upper intrapulmonary gland. Exudative tuberculous basal meningitis.

Case IV.—Male, aged 18. Clinical picture was that of a classical *tabes mesenterica*. Post-mortem: Extensive ulcerative tuberculosis of the ileocaecal region with compact caseation of the mesenteric lymph glands. A bovine strain of *Mycobact. tubert.* recovered from the mesenteric glands.

Case V.—Male, aged 19. Necropsy: Fresh genito-urinary and basal meningeal tuberculosis. Soft caseous focus in the right upper lobe subpleural and infraclavicular. Right tracheobronchial lymph glands with extensive soft caseation.

Case VI (Clare Hall).—Female, aged 19. Bronchogenic-tuberculosis. Death from intestinal tuberculosis. Plum-sized cavity with caseous wall in the mid-zone of the left upper lobe. Extensive lobular caseous spread (bronchogenic) in the right lower lobe. Encapsulated caseous focus the size of a large pea in the lower parts of the right upper lobe near the interlobar fissure. In right upper intrapulmonary glands two caseous foci, the size of a small lentil, one pin-point. Other hilar glands free.

Case VII (fig. 4) (C. M. C. H. P. M. 41/201).—Male, aged 20. Death from ulcerative colitis. (No evidence of tuberculous aetiology). In the left upper lobe 2 cm. below the apex a subpleural encapsulated focus 3×2 cm. with pleural adhesion. Hilar glands small and free with the exception of an intrapulmonary gland regional to the focus. This gland contains a small caseous nodule 0.5 cm. in diameter.

Case VIII (fig. 5) (C. M. C. H. P. M. 42/38).—Female, aged 20. For seven years diabetes. Now ill for some months with tuberculosis. Post-mortem: Large, thin-walled cavity, ill-defined, in the left mid-zone. Extensive caseous spread throughout both lungs (bronchogenic aspiration). In a few intrapulmonary lymph glands near the large cavity in the left mid-zone, a few caseous deposits, each about 0.3 cm. in diameter. Other hilar glands free.

Case IX (fig. 6, fig. 7, A and B) (Clare Hall).—Male, aged 24. A younger brother suffered from psoas abscess five years ago. Death from tuberculous meningitis. Encapsulated, round caseous focus near the left base, 1 cm. in diameter. Extensive caseation of the hilar glands, especially on the left side and regional to the left base. Miliary tuberculosis.

Case X (fig. 8) (Clare Hall).—Labourer. No known contact. Left pleural and pericardial effusion and ascites. Animal inoculation with pericardial fluid positive. Death from tuberculous meningitis. Post-mortem: Large wedge-shaped, caseous focus at the right base, 4×3 cm. Caseous deposit 0.5 cm. in diameter in a lateral hilar gland of the right lower lobe. Tuberculous meningitis and pericarditis.

Case XI (Clare Hall).—Female, aged 30. No evidence of contact. Following abortion, miliary tuberculosis and meningitis. Caseous salpingitis. A pea-sized fresh caseous focus about 1.0 cm. in diameter, with thin capsule in the marginal parts of the left lower lobe about 1 cm. below the pleura. Caseous deposit about 1 cm. in diameter in the left bifurcation gland. Miliary tuberculosis, especially in the lungs. Tuberculous meningitis.

Case XII.—Male, aged 33. Three months before death: abdominal pain, fever, dyspnoea. Diarrhoea. Death from tuberculous meningitis. Post-mortem: Single ulcer of ileo-caecal region with large caseous ileo-caecal lymph gland. Urogenital tuberculosis. Meningitis.

Case XIII (C.M.C.H., P. M. 41/154).—Female, aged 40. Rheumatic fever twenty-two years ago. Obliteration of pericardial sac. Death from congestive cardiac failure. In the left lower lobe, a well-defined quadrangular subpleural caseous focus, 2×1 cm. Confluent caseation of the right intrapulmonary and bifurcation glands. Scattered tuberculous nodules in spleen, liver and lungs.

(2) *Anatomical Diagnosis of Adult Primary Tuberculosis. Differentiation from Reinfection*

All these observations have one feature in common: in the lungs or in the intestinal canal there is a caseous (or ulcerative) focus with similar changes in the corresponding lymph glands; in other words, a fresh primary complex. In all cases this is clearly marked out from other tuberculous lesions (if present). Careful dissection and screening of the lungs have shown that no traces of infection older than the primary complex described above were present.

True reinfection, i.e. the establishment of a lesion with all anatomical characters of the primary complex, in an instance in which the calcified or ossified residues of a previous primary infection were found, has been recorded. I have seen this in my own material in two instances only, and I think that it is not frequent enough to discredit the current anatomical view of primary infection, i.e. that this occurs as a rule only once in life. It does justify, however, a careful search for traces of an older, i.e. the true primary lesion, especially when a fresh primary complex is found in adults.

(3) *Anatomical Features of Adult Primary Tuberculosis in Detail*

In the present examples no calcification was seen and thereby the fact that in each case we were dealing with a recent, primary infection in adult life was established. Out of 13 cases, 3 were intestinal and 10 pulmonary infections, which is approximately the same distribution as in children. In anatomical detail, the changes in most cases resembled those observed in childhood. The primary focus was found in the lower parts of the lungs, usually near the base and not far from the pleura.

In one respect a difference was found. In children, the primary focus is, as a rule, smaller than the changes in the lymph glands. In the present series of adults this ratio was reversed in four out of eight cases of pulmonary infection, and in one instance primary and glandular foci were about equal in size and very small. It may be assumed, therefore, that in adults an enlargement of the hilar glands need not be conspicuous either in the anatomical specimen or in the skiagram. It must be emphasized, however, that *the lymph gland corresponding to the primary focus does show changes which are identical in quality with the latter*. In spite of the comparatively small size of the glandular changes, *the classical picture of the primary complex does appear in adults just as in children*. In many adults the glandular changes were extensive and in no way less than those observed in children.

Age.—The present series includes persons up to the age of 40. Anders observed three instances in persons over 40, one was 79. Out of 16 cases reported by Terplan, 11 were over 40, one was 80. In all cases typical primary complexes were observed.

(4) *Frequency and Influence of Age and Resistance*

Up to 1931, cases of primary infection in adults were described as pathological curiosities. Then 36 observations made by Anders were described by Ragnotti. More recently Terplan (1940) has given an account of 23 cases, i.e. about 7% of 286 adults. This figure is much higher than those given ten and fifteen years ago, when about 0.1-0.5% were reached (Schürmann, 1926; Ragnotti, 1931). The 13 cases of my own material were

THIRTEEN PERSONAL OBSERVATIONS OF PRIMARY TUBERCULOSIS IN ADULTS AND ADOLESCENTS.

| No. | Age | Site of Primary Complex | | Size of Gland relative to prim. focus | Post-primary tuberculosis | Cause of death |
|-----|-----|-------------------------|-----------------|---------------------------------------|----------------------------------------------------------|----------------------------|
| | | Pulmonary | Intestinal | | | |
| 1 | 15 | R. lower | — | Larger | — | Toxaemia |
| 2 | 17 | — | Duodenum | Larger | Round focus in lt. upper lobe. | Tub. mening. |
| 3 | 18 | R. upper | — | Equal | Meningitis | Tub. mening. |
| 4 | 18 | — | — | — | Meningitis | Tabes mes. |
| 5 | 19 | R. upper | Small intestine | Larger | Urogenital tub. Meningitis | Tub. mening. |
| 6 | 19 | R. upper | — | Larger | Bronchogenic tub. | Bronchogenic tub. |
| 7 | 20 | L. upper | — | Smaller | — | Ulcerat. colitis |
| 8 | 20 | L. mid-zone | — | Smaller | Bronchogenic tub. | Bronchogenic tub. diabetes |
| 9 | 24 | L. base | — | Larger | Miliary tub. Mening. | Tub. mening. |
| 10 | 29 | R. base | — | Smaller | Tub. polyserositis. | Tub. mening. |
| 11 | 30 | L. lower | — | Equal | Genital tub. Mening. | Tub. mening. |
| 12 | 33 | — | Heccæcal | Larger | Urogenital tub. Discrete miliary spread in lung. Mening. | Tub. mening. |
| 13 | 40 | L. lower | — | Larger | Negligible spread | Cardiac failure |

observed during the last four years. Previously I had no such pure instances, only cases occurring in puberty in which a large primary infection was quickly followed by bronchogenic tuberculosis. I can, therefore, confirm the impression that *primary tuberculosis in adults is nowadays more common than before*.

The characteristic feature of primary tuberculous infection, namely, the involvement of the corresponding glands, appears in children as well as in adults with such regularity that it is obviously due to the absence of previous infection and not to age and resistance. Influence of age on the extent of the lesion, however, is not unlikely, in that the glandular part of the primary complex may be smaller in adults than in children. It is difficult to give an anatomical reason for this. I have examined histologically hilar lymph glands from 30 non-tuberculous individuals between the ages of 3 and 50. I have found no essential structural differences (such as an increase in fibrous tissue due to collection of dust or residues of inflammatory changes) which could account for an age-resistance of the lymph glands to tuberculous infection. Primary tuberculous changes in the hilar glands of adults may involve areas much larger than those in many children. Finally, in adult consumptives in which the hilar glands are, as a rule, devoid of caseation, tubercle bacilli may be found in considerable numbers in the sinuses without more than trivial changes. This militates against the theory that the lymph glands of adults do not allow of as easy a penetration of the bacilli as do the glands of children.

It has been inferred that generalization of tuberculosis subsequent to primary infection indicates low natural resistance of the individual to tuberculosis whereas this is higher in bronchogenic tuberculosis, since this is restricted to one organ, the lungs. The highest degree of natural resistance, however, would appear to be in those in whom the primary complex remains the only manifestation of tuberculosis. Judged by these standards the influence of natural resistance on the occurrence and anatomical features of primary tuberculosis in adults is negligible. Among Anders' 36 instances were no less than 14, among the 23 cases observed by Terplan no less than 16 accidental findings, in other words, they were found in persons with the highest degree of natural resistance. In the present series 3 out of 13 belonged to this group and 2 were cases of bronchogenic tuberculosis. In the latter, bronchogenic tuberculosis seemed to follow quickly on primary infection in adult life. This is different from the long interval between primary infection and bronchogenic tuberculosis which we deduce from the observation of a calcified or ossified primary lesion in about 70-92% of the necropsies in bronchogenic tuberculosis (Pagel, 1930). The unusually short interval between primary infection and bronchogenic tuberculosis may be a feature associated with late primary infection. It might explain the frequency of contact found in early cases of bronchogenic tuberculosis in young adults. Evidence of contact has been taken as proof of "exogenous reinfection" (Opie and McPhedran, 1935) leading to bronchogenic tuberculosis. Contact, however, could have caused, in these cases, a late primary complex with quickly ensuing bronchogenic tuberculosis due to "endogenous" spread.

Tuberculosis, therefore, obeys the laws of primary infection, irrespective of age and natural resistance. Primary and glandular foci develop in children as well as in adults, in persons with low, as well as with high, natural resistance. Clinical inferences against the occurrence of the primary complex in certain adults are based on radiological evidence only and find no support in the anatomical observations.

SUMMARY AND CONCLUSIONS

(1) The anatomical basis of primary tuberculosis in adults has been illustrated by thirteen personal observations in persons aged 15-40 years. (2) In all cases, infection obeyed the law of the primary complex. In some instances the changes in the corresponding lymph glands were smaller than the primary focus in the lung. (3) Anatomical evidence suggests a greater frequency of primary infection in adult life in recent years. (4) A fresh tuberculous primary complex occurs in adult persons associated with post-primary (disseminated and bronchogenic) as well as without post-primary tuberculosis. The primary complex develops, therefore, in adult life independent of age and natural resistance.

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FIG. 1 (Case II).—? Primary tuberculous ulcer in the duodenum.

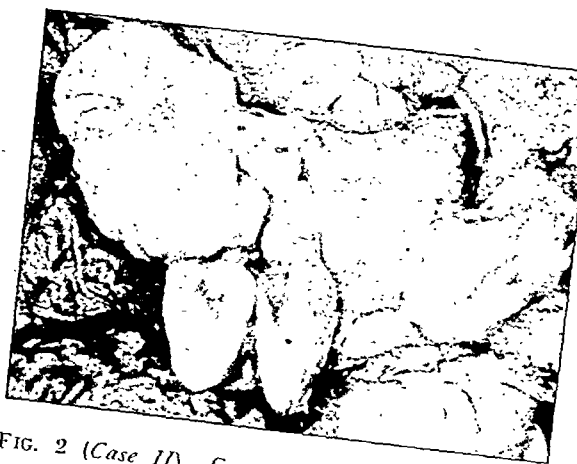


FIG. 2 (Case II).—Compact caseation of enlarged retro-duodenal lymph glands.



FIG. 3 (Case II).—Infra-clavicular caseous focus with enlarged and partly caseous hilar lymph glands.



FIG. 4 (Case VII).—Left upper lobe with primary caseous focus below the apex (arrow) and smaller caseous deposit in lymph gland (arrow).



FIG. 5 (Case VIII).—Extensive caseous destruction of both lungs (bronchogenic tuberculosis). Primary cavity left middle zone (not shown). Small caseous deposits in three intrapulmonary lymph glands.

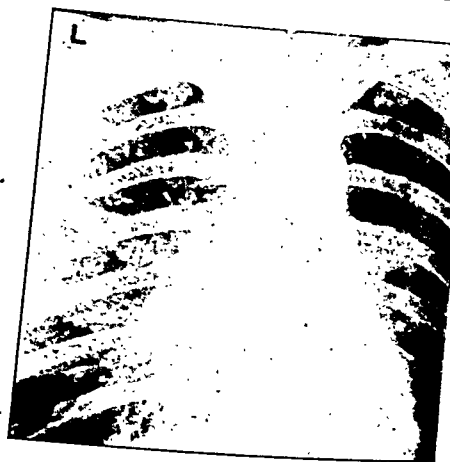


FIG. 6 (Case IX).—Skiagram of chest. Enlargement of left hilum.

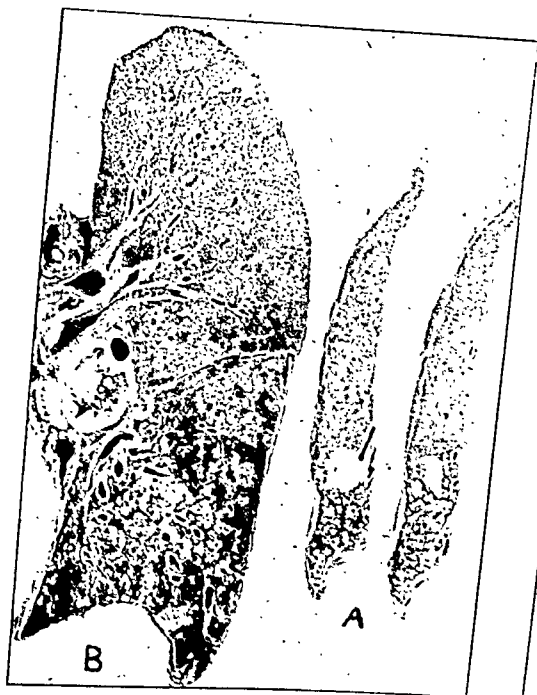


FIG. 7 (Case IX).—Left lung. A. Small primary focus in the dorsal parts near the left base. B. Larger caseous hilar lymph gland and survey of ventral parts of the left lung.



FIG. 8 (Case IX).—Wedge-shaped, primary focus near right base (arrow). Small caseous deposit in lymph gland (arrow).

Section of Medicine

President—GEOFFREY MARSHALL, O.B.E., M.D.

[May 5, 1942]

DISCUSSION ON THE WORKMEN'S COMPENSATION ACT AS A FACTOR IN PROLONGING INVALIDISM

Dr. Bernard Hart : In the B.M.A. "Report of Committee on Fractures" (1935) the statement is made that: "The Workmen's Compensation Acts, although designed to protect the workman, have in many cases played a prominent part in prolonging his disability, in delaying his return to work, and, on occasion, in converting him into a permanent invalid". It is our task at this Discussion to consider how far such a statement is justified. I shall define "invalidism" as a condition due to psychological factors which has become superimposed, either on a pre-existing organic disability, or on a pre-existing neurosis. I am not concerned with prolongation of a disability by physical factors, e.g. deficient diet, though such factors are of undoubted importance and may be closely connected with the administration of the Acts.

In so far as the Workmen's Compensation Acts are responsible for invalidism, such effect must be due in the main to the intervention of psychological factors. Russell Brain (1942), who has recently investigated two groups of head-injury cases, one due to road accidents and the other to industrial accidents, found that the incidence of neurosis in the second group was much higher than in the former. This clearly indicates that other factors than the injury itself are concerned, and it may be inferred that these must be factors of a psychological order. No doubt psychological factors are operative in both groups of cases, but the explanation of their differential action in the two groups must be sought in a comparison of the procedures of the Workmen's Compensation Acts with those affecting road accident compensation.

We shall begin, therefore, by considering the various psychogenic processes which may play a part in the production of invalidism, examining in each case the extent to which it may be called into action by the circumstances of the Workmen's Compensation Acts. The first of these underlies the notion of "compensation neurosis", but is in fact much wider in its significance than a mere relation to monetary compensation. It is perhaps best described as "purpose" or "motive", and purpose, whether conscious, semi-conscious, or unconscious, plays a very important part in the production of neurosis. It would be unreasonable to ask for what object a patient suffering from scarlet fever has acquired that illness, but it is not unreasonable to ask it in many cases of neurosis. A glimmering of the notion of "purpose" as a factor in neurosis arose in the days of "railway spine", but the clearest evidence of its functioning is to be found in the psychoneuroses of the last war, absurdly misnamed "shell-shock".

A simple example from everyday life will serve to indicate the nature of the mechanism involved. Suppose that I am standing in the middle of the road and a motor bus bears down on me. The self-preservation instinct, which is allowed free-play here, will ensure that I step out of the way on to the pavement. Suppose, however, that I am standing on the pavement and a small child is playing in the road, right in the path of the oncoming bus, two factors will then come into action, the self-preservation instinct which would keep me out of danger on the pavement, and what we will call "duty" which will drive me on to the road to rescue the child. Here there is a conflict between two diametrically

opposed forces. Now consider the soldier in the trenches, and it will be seen that he is subjected constantly to a conflict of this kind. Self-preservation instinct, if allowed free play, would cause a prompt removal from the post of danger; "duty" constrains him to remain where he is. The training of the soldier is designed to deal with this conflict by so enhancing the strength of the duty-discipline factors that the self-preservation instinct has no chance against them. So long as this relation holds, all is well, but if for any reason the duty-discipline factors become weakened, or the self-preservation factors enhanced, then the conflict becomes acute. Now an acute conflict cannot be borne indefinitely; some solution has to occur, and it must be understood that "solution" means satisfying both the opposing factors, not surrendering to one of them. There are three possible solutions. The first is the advent of a serious wound. This provides a perfect solution of the conflict, because both self-preservation and duty are satisfied, the man going down the line preserving both himself and his self-respect. The second solution is being taken prisoner, which similarly satisfies both the opponents. But there is a third solution, the development of a psychoneurosis, let us say a hysterical paralysis. This equally satisfies both the opponents, the man being removed to safety, but preserving his self-respect because, as he believes, he has a disability similar to that of his comrade who has been shot through the spine. There is a fundamental difference, however, between this third solution and the other two. Those two arose from external causes, the third is brought about by internal causes, causes of a psychological order. The fact that wound or capture will solve the conflict is not a cause of the wound or capture, but only a happy coincidence. But the fact that a psychoneurosis will solve the conflict is an actual cause of the production of the psychoneurosis. Here, therefore, "purpose" appears as a fundamental factor in the causation of disorder. Its existence explains the familiar observation that it is rare for a seriously wounded man to show psychoneurotic symptoms. If a man is seriously wounded, it may be said from this point of view that there is no need for him to have a neurosis. The conflict, if it existed, has been solved very effectually without such aid. The existence or absence of "purpose" also provides at any rate one explanation of the remarkable fact that in the last war there were an immense number of psychoneurotic casualties in the Army, whereas in the present war similar casualties amongst civilians subjected to severe blitzes are rare.

The action of "purpose" in these cases was not "conscious" in the full sense of that word. It has been maintained that, as the man was perfectly aware of the fact that he would like to be out of the fight, the part played by this desire would necessarily be fully conscious. This is a misunderstanding of the actual situation. The man is aware of the desire, but he is not aware of the machinery by which it produces the neurosis. If he were so aware the machinery set in action would be that of malingering and not of neurosis. "Purpose" in this connexion may therefore properly be called "unconscious motive", and that term is in fact the one generally employed.

Later in the history of a psychoneurotic patient of the last war "purpose" often shifted from self-preservation to pension, but by that time many other complicating factors had come into action. The failure to realize this, and the assumption that the condition was then simply a purposive compensation-neurosis, led to many mistakes in handling. For example the payment of a lump-sum compensation instead of a continued pension in the hope that the removal of the end to which the purpose was thought to be directed would produce prompt recovery. The experience of the Ministry of Pensions decisively contradicted this view, a fact to be borne in mind when we are considering the application of similar methods in dealing with Workmen's Compensation cases. Indeed the payment of lump-sum compensation as a weapon of recovery is largely discredited by those in it is absolutely prohibited (Wilson and Levy, 1939).

With a Compensation Act in force, "purpose" as a factor working in the direction of compensation is likely to play a part, and its action in prolonging disability certainly cannot be neglected, but we must not assign to it too large a part. In the war cases the neurosis arose as a compromise between self-preservation in a situation of extreme danger and the factors we have termed "Duty and Discipline". These are forces of great potency, but where can we find their equivalent in the neurosis of the injured workman? Surely not in a drive to obtain a weekly payment of less than half his former income, at the price of indefinite unemployment and idleness, a price which no workman unless he is in other respects pathological, is prepared to pay. It is clear, therefore, that other factors must be at least co-operating. When those other factors are examined it will be found, indeed, that "purpose" remains a predominating cause in the invalidism associated with the Workmen's Compensation Acts, but it is not a mere drive to monetary compensation.

The first of these other factors is "Preoccupation". It may be defined as consisting in fixation of attention upon a function of the body or mind plus apprehension or anxiety. Fixation of attention in itself is not, of course, preoccupation. It only becomes so if the element of apprehension or anxiety is added. Preoccupation is a familiar factor in neurotic conditions, for example the functional dyspepsias, effort syndrome, and a host of similar disorders. There is, indeed, no function of the body or mind which may not be subjected to a preoccupation process, and hence become the focus of a neurosis.

It will be obvious that a mechanism of this kind is easily capable of being set up in an injured workman, and there can be no doubt that it plays a part in a large number of the cases which travel along the neurotic road. The mere existence of an injury which in his ignorance is fraught with unknown and sinister possibilities, sets the stage for preoccupation to come into action. But another factor exerts its influence here in precipitating and maintaining the machinery of preoccupation, the factor of "Suggestion". Preoccupation and suggestion are closely allied, but it may be said for our present purpose, that preoccupation is a process which takes place wholly within, while suggestion is applied from an external source. Such suggestion is, however, a very potent engine in initiating and maintaining an internal preoccupation.

The atmosphere surrounding a workman who has recently sustained an injury is one which is capable of starting or accentuating preoccupation. Thus the ministrations of his own doctor, innocent in intent but often none the less malignant in result, may play an important part here. Visits to his lawyer or trade union official, with the constant accent on compensation and the need for obtaining security, add to the pathological suggestive factors. Insurance companies, with their legal and medical advisers, necessarily regarded as hostile agents whose aim is to whittle down that security, do not, as is sometimes fondly hoped, exert an opposing suggestive effect, but merely serve to strengthen the suggestion proceeding from what may be called his own side. Relatives and friends help to carry on the bad work, and it is small wonder that, in the words of Sir John Collie, "by the time the trial is reached, he is commonly the victim, not so much of the accident, as of the numerous influences which have been brought to bear on him since it occurred" (Knocker, 1910).

Though all these sinister possibilities hover round the injured workman, yet only a small minority develop neurosis, while the great majority make an uninterrupted recovery. In the first place the influences I have depicted in somewhat highly coloured terms are not in fact applied to any notable extent in a large number of cases. Particularly is this true in minor injuries capable of rapid settlement and recovery, the incidence of neurosis naturally being higher in long-drawn-out cases, in which suggestive factors have a greater opportunity of influencing progress. In the second place, however, much depends on the man's psychological constitution and temperament, and the presence or absence of prior neurotic trends.

These are of great importance not only in conditioning the onset of a neurosis after an accident in which no organic injury was sustained, but also in influencing the growth of a neurotic superstructure upon a primary organic disability. The accident may, indeed, merely serve as a peg to which pre-existing neurosis, conflicts, and semiconscious or unconscious trends attach themselves. But as it cannot be denied that the accident does play some part in fashioning the final picture, attributability in the legal sense has to be accepted, and with it the benevolent or malignant results which attend the operations of the Workmen's Compensation Acts. Such cases, which lead to much unedifying wrangling amongst medical witnesses, have a complex causation which cannot be put into the simple and definite terms demanded by the legal mind, and they constitute an insoluble problem, so long as attention is concentrated solely on "attributability" and "compensation". They provide strong support for the contention that reform of the Acts must move in the direction of taking away the accent on compensation, and must concentrate on the essential problem of how best to get back to normal activity and work a man who is temporarily disabled. Incidentally such reform would do away with the futile disputes between medical witnesses about a causation which is inevitably obscure and largely beyond our present knowledge accurately to assess.

The lawyers have invented a term "brooding", to indicate a mental state which debars a workman from a successful result to an application under the Workmen's Compensation Acts (Brend, 1938). I am unable to discover any medical meaning in this term, however satisfactory its legal definition may be. Unless it is assumed that a man "broods" deliberately, and can stop doing so whenever he likes, a patent absurdity in neurosis cases, brooding is practically equivalent to preoccupation. In that sense it is a frequent and integral factor in accident-neuroses, but it is not a volitional process,

and it is obviously an effect of the accident, however many other factors may co-operate in its production.

I have mentioned the part which the injured man's anxiety about his position and future may play in setting the stage for the onset of preoccupation. But this does not exhaust the action of anxiety as a psychological factor in neurosis. Indirectly it may add to neurosis such undermining influences as insomnia, and directly it is a substantial factor in producing a purposive reaction in that much wider sense of "purpose" than mere drive to monetary compensation. A hint of this wider sense was mentioned before, and an attempt must now be made to explain more fully what is meant.

Man may be regarded as a psycho-physical organism whose function it is to adapt himself to the environment in which he lives, such adaptation being effected by a due co-ordination of internal drives and stresses with external conditions. From this point of view neurosis in general, which it must always be remembered is a disorder of the whole individual and not of a single system or organ, is an attempt at such adaptation. It is of course a bad and inadequate one, only to be obtained at the price of illness, but it does in fact provide some sort of adaptation to the internal and external stresses to which the patient is subjected. This adaptation aspect is very clear in the case of the soldier who breaks down in the trenches with a hysterical paraplegia, but it is to be found in a much wider field of neurosis than hysteria. Now the fact that an illness may provide an adaptation is only a fortuitous circumstance when the illness is determined by organic and external factors, but it is of prime importance when the illness is one determined by psychogenic factors, because the circumstance that it does provide an adaptation takes its place among the causal psychogenic factors. This is that wider sense of "purpose", far wider than mere compensation, which must be taken into account over almost the whole field of neurosis. Efficient therapy must aim at blocking the channels whereby "purpose" leads to that morbid adaptation which is neurosis, and at directing it into the channels which lead to efficient and adequate adaptation.

There are certain prophylactic factors which should come into operation from the date of injury, and exert their effect throughout the whole course of treatment. These are the factors which subserve the maintenance of bodily and mental efficiency, physical and mental exercise, the preservation of self-respect, and the constant nourishment of the "will to recover". As Jefferson, in a recent communication, has said: "From the first the patient must be made to realize that he is an active collaborator in his own treatment and that he must accept some responsibility in his progress towards a successful result" (Jefferson, 1942). Measures of this kind may well make all the difference between rapid recovery and needlessly prolonged invalidism.

If we review all these various psychological processes which may come into psychogenic action in the injured workman it will be obvious that equating accident-neurosis with compensation-neurosis is a gross misinterpretation of the actual situation. Compensation of course plays a part, and must always be reckoned with, but it is probably a very small part in early cases, although it may dominate the picture later on, when all those other factors I have mentioned come into operation. Owing to the structure and procedure of the Acts, indeed, it easily becomes the focus upon which all these co-operating factors finally converge. And it does so because the Acts are focused upon that one point of monetary compensation, and neglect altogether the really essential points of recovery and rehabilitation. The Acts have no provision for treatment, and for this the workman is thrown on his own resources with such aid as can be obtained from hospitals and the National Health Insurance. It is no wonder, therefore, that anxiety about family and future, constantly fed by the factors of suggestion and preoccupation described, ultimately converge into a drive for security, which is far more often the "purpose" or "unconscious motive" behind these neuroses than compensation in the narrower sense. Naturally, as monetary compensation is the only satisfaction of the desire for security which the Acts provide, this becomes the focus of the psychological factors. But it has become a symbol rather than an end in itself.

These considerations relate particularly to those cases which commence with an organic disability and progress to invalidism. They apply also to those cases of primary neurosis in which the initial injury is slight or altogether negligible, but in which this fact is not clearly and promptly understood by the patient. To him the injury may be of unknown magnitude, and a sinister threat to future security, and this fear, accentuated by the medical and legal procedures associated with the Acts, is a potent inducement to neurosis and invalidism. A practical lesson to be drawn here is the grave responsibility which falls in these early cases upon the patient's doctor. If he explains clearly to the patient the precise nature of the injury, vigorously reassures him about his baseless fear, and instils in him the conviction of speedy recovery, all should be well. If he fails

to do these things, or implants instead, however inadvertently, contrary suggestions and fears, then he has ranged himself among the factors responsible for neurosis.

Many cases of neurosis following accidents in which physical injury is negligible or absent, are not primary neurosis, but cases of pre-existing neurosis in which the accident has simply provided a favourable opportunity for manifestation. In such cases the anxiety, preoccupation, and suggestions, associated with the accident of course help to colour the picture or to provide a superstructure to the original neurosis, and they require appropriate treatment. But they are not accident-neurosis in the proper sense, although they come equally under the Workmen's Compensation Acts, because from the legal standpoint the employer's liability is the same, whether the man before the accident was normal or neurotic, if the accident can be held to have made more manifest or worsened a pre-existing neurosis.

Having considered the foregoing arguments, we are now in a position to assess more accurately the common notion that the best method of preventing and curing accident neurosis is prompt settlement of the claim by a lump-sum payment.

The remedy for invalidism in accident cases is not to be found in any such simple panacea as this, but must be sought by a systematic attempt to combat all the factors which have been described. The action of preoccupation, suggestion, and anxiety, must all be taken into account, and above all the potent and ramifying effects of the drive for security, a much wider thing than monetary compensation, must be fully considered. It is immediately obvious that these factors cannot be satisfactorily combated, so long as the Acts remain as they are at present. The structure of the Acts may, indeed, not unfairly be described as carefully calculated to enhance each and every one of those factors. This country stands almost alone in its failure to incorporate in its accident legislation any provision for treatment and rehabilitation, obviously the most essential weapons for attacking the psychogenic factors underlying invalidism, and until this failure is remedied the problem of invalidism in these cases will remain with us.

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- The Legal Position

His Honour Tom Eastham, K.C.: Compensation under the Workmen's Compensation Acts is payable where total or partial incapacity for work results from injury by accident arising out of and in the course of the workman's employment. The words in the Act are "incapacity for work". These words are not the same as "incapacity to work". They mean the loss or diminution of wage-earning capacity and they include inability to get work if that be the result of the accidental injury. Whether there is incapacity for work or not is a question of fact which has to be determined by the County Court Judge. He has to decide whether the incapacity has resulted or continues from the injury and the amount of such incapacity. These are questions of fact, and the burden of proof is upon the workman.

It is not necessary for the workman to show that the incapacity was the natural or probable consequence of the injury so long as it results from the injury. Upon such questions of fact the County Court Judge's findings one way or the other cannot be disturbed (apart from misdirection in law) if there be evidence upon which a reasonable man might come to the conclusion arrived at by the County Court Judge. In other words, the Court of Appeal has no power to interfere with a finding of fact of a County Court Judge if there is evidence to support it and the House of Lords has said: "It is of first importance that the finality of an arbitrator's finding of fact under the Workmen's Compensation Act should be jealously maintained." It is very important to remember that there is no appeal upon fact from a County Court Judge's decision in a Workmen's Compensation case that has been taken to the Court of Appeal or to the House of Lords. The Appeal Court is bound by the findings of fact; it cannot vary or reverse them if there was some

evidence upon which they could be based, and the Appeal Court has to deal with the case on the basis that the findings of fact are conclusive. Even if the judges of the Appeal Court would have arrived at different findings of fact upon the evidence, they cannot reverse the findings of facts of the County Court Judge if there was any evidence upon which he could so find.

Having made these preliminary observations I shall now deal with incapacity for work from nervous effects or loss of will power. The best way of doing so is to give a summary of a number of these cases which have been before the Courts.¹

Even when a workman has recovered from the physical or muscular mischief caused by the accident, he may still be incapacitated owing to a nervous or mental condition, and such incapacity may be the result of the physical injury. For instance, a condition known as "traumatic neurasthenia" frequently results from an injury: the workman may have entirely regained his former physical state and really be competent to work, but he genuinely, though mistakenly and unreasonably, believes that he is incapable of working. Such were the circumstances in *EAVES v. BLAENCLYDACH COLLIERY CO., LTD.* (1909), where a workman sustained a muscular injury to his leg through an accident. He had entirely recovered from the muscular consequence of the injury, but suffered from traumatic neurasthenia and anaesthesia of the leg as a consequence of the accident. It was held that his right to compensation did not cease when the muscular mischief was ended, but continued so long as the nervous effects remained and caused total or partial incapacity for work.

EAVES' case (*supra*) was followed in *CHARLES WALL LTD. v. STEEL* (1915), where a builders' labourer in March 1913 suffered an injury to his head, but the wound was completely healed by July 1913. In September 1914, the employers applied to diminish the compensation which they were paying him, on the ground that he was fit for light work, which they had offered him, but which he had refused. The medical evidence was conflicting, but the medical assessor reported that, although he could do light work on the level, he was not a malingerer and genuinely believed he was unable to work. The County Court Judge found that the workman honestly believed he was incapable of work, and that his condition was due to neurasthenia resulting from the accident, but he strongly advised the man to try and get work. He dismissed the application, and it was held by the Court of Appeal (Phillimore, L. J., dissenting) that there was evidence to justify him doing so.

But to entitle the workman to a continuance of the compensation, the neurasthenia must be genuine and there must be no suspicion of malingering. When the nervousness resulting from the accident is such that a reasonable man could overcome by making a genuine effort to work, the decision in *EAVES v. BLAENCLYDACH COLLIERY CO., LTD.* (*supra*) does not apply. For instance, in *TURNER v. BROOKS AND DOXEY LTD.* (1909) the County Court Judge found that the refusal of a workman to continue work was due to nervousness which an average reasonable man would overcome and, although the nervousness was due to the accident, he declined to award compensation. The Court of Appeal agreed with his decision, Lord Cozens-Hardy, M.R., saying that he had no doubt that the learned Judge meant to find that the man was perfectly able to work and that the result of payment of compensation would take away all stimulus to do so. A somewhat similar decision was given in *HOLT v. YATES AND THOM* (1909) where it was held that the Arbitrator was right in finding that a man, who was not suffering from any incapacity for work as a result of the injury, but where inability to work was caused by brooding over the effects of the accident, was not incapacitated within the meaning of the Act.

In *HIGGS AND HILLS v. UNICUME* (1913) the County Court Judge terminated compensation on the grounds that the workman had unreasonably refused an offer of light work; that an average reasonable man suffering as he did would long ago have gone back to work; that, acting on unwise medical advice, and under the domination of his wife, he had behaved in an unreasonable way, but was not a malingerer; that he was suffering from weakness of will and a fixed, but erroneous, idea that he was a chronic invalid; and that a continuance of compensation was likely to keep up his present condition. It was held that, on these findings, the workman's condition was not the result of the accident. But in *STRIDE v. SOUTHAMPTON GAS LIGHT AND COKE CO., LTD.* (1916) where a workman's legs were paralysed owing to loss of will power as the result of an accident, the Court of Appeal held that there being no evidence that light work which the applicant could perform was available, and no evidence of unreasonable conduct or malingering on his part, the Arbitrator was wrong in reducing the compensation to 1d. per week and that the award should have been on the basis of total incapacity.

¹ Elliott's Workmen's Compensation Acts, 9th Ed., and Willis's Workmen's Compensation, 34th Ed.

"The learned County Court Judge," said Lord Cozens-Hardy, M.R., "seems to me to have overlooked the fact that the loss of will power is just as much a result of the accident as any objective symptoms would be."

In *YATES v. SOUTH KIRKBY & Co., COLLIERIES LTD.* (1910) the doctrine established in *EAVES v. BLAENCLYDACH COLLIERY CO., LTD.* was extended to a remarkable degree. Here there was no traumatic neurasthenia, because the workman had not suffered any specific injury by the accident; he merely sustained a shock to his nervous system through the excitement and alarm caused by seeing the effects of an accident to a fellow-workman. Owing to the shock, neurasthenia supervened and rendered him unable to follow his usual occupation, and it was held that the incapacity resulted from "personal injury by accident".

In that case the collier suffered no specific injury but it was held that a nervous shock causing incapacity to work was as much a "personal injury by accident" as a broken limb or other physical injury.

The Courts have also considered cases where the incapacity for work has been caused through *loafing*. In one case the Court decided that if a workman's incapacity (for example the soft condition of his muscles), is brought about by loafing the County Court Judge may justly terminate or reduce the compensation [*DAVID v. WINDSOR COAL* (1911)]; but in another case the Court declined to do so if the workman's physical incapacity is due to his not working and *he has tried* but failed to obtain employment suited to his condition [*BONSALL v. MIDLAND COLLIERY COMP.* (1914)].

The reasonableness or unreasonableness of the workman's conduct and its effects are questions of fact.

Since these cases were decided the law has been amended to the advantage of the workman by a Workmen's Compensation Act 1931. The material part is as follows:

If a workman who has so far recovered from the injury as to be fit for employment of a certain kind has failed to obtain employment and it appears to the County Court Judge either—

- (1) that, having regard to all the circumstances, it is probable that the workman would, but for the continuing effects of the injury, be able to obtain work in the same grade in the same class of employment as before the accident; or
- (2) that his failure to obtain employment is a consequence, wholly or mainly, of the injury,

the judge shall order that the workman's incapacity shall be treated as total incapacity resulting from the injury for such period, and subject to such conditions, as may be provided by the order, without prejudice, however, to the right of review conferred by this Act:

Provided that—

- (1) no order shall be made under this subsection if it appears to the judge that the workman has not taken all reasonable steps to obtain employment; and
- (2) every such order shall be made subject to the condition that it shall cease to be in force if the workman receives unemployment benefit.

This Act only applies if there is partial incapacity for work. It has no application if there has been complete recovery so that all incapacity is negated. In cases of partial incapacity the employer who seeks to diminish the payments of compensation should prove that suitable employment is available for the workman, and that he is capable of doing such work. The medical witness should know what the work is and be able to say that the workman can do it. That being so, before going into the witness box the medical witness should if possible see the work that the employers offer to the workman so that he can say that he knows what the work is, and then he can say whether or not such work is within the capacity of the workman.

I have tried to state the law as it is to-day. If you are not satisfied with it as it is then your Society should consider in what way the law should be amended. It may be your view that in the workman's interest and in the public interest more power should be given to the Court to suspend the payments of compensation in a case where incapacity results from mere nervous effects or loss of will power or by brooding over the effects of an accident that a reasonable workman in his own interest should overcome.

evidence upon which they could be based, and the Appeal Court has to deal with the case on the basis that the findings of fact are conclusive. Even if the judges of the Appeal Court would have arrived at different findings of fact upon the evidence, they cannot reverse the findings of facts of the County Court Judge if there was any evidence upon which he could so find.

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¹ Elliott's Workmen's Compensation Acts, 9th Ed., and Willis's Workmen's Compensation, 34th Ed.

(3) *The Curse of Under-Nourishment*

In the Interdepartmental Committee Report on Rehabilitation, which was published in 1939, in a passage referring to industrial injuries, the statement is made that: "The out-patient departments of our hospitals are crowded with patients suffering from the combined disabilities of injury and under-nourishment."

This is a reference to an investigation of mine into a very large number of industrial injuries, which showed that in the out-patient departments of our hospitals in London the average workman in receipt of Workmen's Compensation showed evidence of malnutrition within six weeks of his attendance as an out-patient. The reason is clear. In pre-war days the maximum weekly sum paid under the Workmen's Compensation Act was 30/-, and in big cities this is quite insufficient to pay for rent and food for the average-sized workman's family. Undernourishment amongst the industrially injured city dwellers is one of the prime factors in the greater prolongation of invalidism in the town than in the country.

In Russia the law recognizes the fact that nourishment plays an important part in recovery from injury and disease, and it stipulates that the workman shall have his full pay, and in special circumstances 25% more pay, when incapacitated by injury.

(4) *The Plague of Light Work*

Under the provisions of the Light Work Clauses in the Workmen's Compensation Acts both before and after 1931, the employer may reduce the amount of the weekly payment of compensation to the workman if he can show that the workman is fit to undertake light work and if light work is available. When the Light Work Clause first became law it was thought, or perhaps hoped, that light work would help to rehabilitate the patient. The evidence given by the British Medical Association before the Royal Commission on Workmen's Compensation has abundantly proved the opposite, and the reasons are not far to seek.

In the majority of trades in which accidents occur there is no real light work and jobs are made by the employer for the workman at the instigation of the insurers. In the building trade, for instance, a skilled artisan has to become a tea-boy or a sweeper-up, and an inferiority complex, perhaps not present before, is immediately born and with it a sense of bitter resentment against his employers and against fate. In another case a skilled workman, perhaps a mechanic, is given specially selected light jobs in his trade. It is true that this occasionally helps him to get better, but more often, because he has to work in pain even at the light tasks which he is doing, pain and work become linked in his mind and his sense of incapacity is driven home with increasing force each time he tries to turn a spanner.

A year or two ago a man who was making a rapid progress towards recovery after a fracture insisted on leaving my gymnasium to do a light job found by his employer, both parties being satisfied that he would be fit in a matter of days. He came to the hospital six months later not as fit as when he left it. When I asked him what he was doing and if he was still working, he replied: "Oh, yes. I'm with the other cripples." This man's light work had consisted in sail repairing with three other men who were permanently incapacitated.

(5) *The Scourge of Over-treatment*

There is one disease which I believe to be definitely attributable to the Workmen's Compensation Acts, which I will call "Recurrent Painful Nerve Endings". It is a condition which I have only seen arise in patients in receipt of Workmen's Compensation or in expectation or hope of a lump sum settlement. I have never seen it in patients in receipt of National Health Insurance, where, of course, there is no right, real or fancied, to lump sum payment.

We are all familiar with the painful stump resulting from partial amputation of the finger, in some cases with and in some without a definite amputation neuroma. The condition is quite easily dealt with surgically in uncomplicated cases, but where Workmen's Compensation is a complication, it is my experience that any form of treatment is invariably followed by recurrence of the pain in a more intensified form.

As soon as the claim is settled radical treatment, by whatever method, is invariably successful.

The folly of operating on these cases whilst there is a claim pending cannot be too strongly condemned: in my view it amounts to malpractice.

A milkman who lost the tip of the little finger of the left hand developed a painful scar. Although he was fit for most of his work I advised the Insurance Company to settle

In other words you may consider that more power to suspend payments of compensation should be given to the Court in cases where the payment of compensation is the main factor in prolonging invalidism; if that is so, your Society should formulate its proposed amendments with evidence and reasons to support them and submit them to the Home Office for consideration.

His Honour then referred to the reports of the important and recent legal decisions upon this subject and read a number of extracts from the judgments in support of his views.

Mr. H. E. Griffiths: The part which the Workmen's Compensation Act takes in prolonging invalidism after an accident may be summarized as follows: (1) The Toxins of Man's Imagined Rights. (2) The Pest of the Lump-Sum Settlement. (3) The Curse of Under-Nourishment. (4) The Plague of Light Work. (5) The Scourge of Over-Treatment.

(1) *The Toxins of Man's Imagined Rights*

In the majority of cases which I have seen in which I felt that prolongation of disability after an accident was due indirectly to the Workmen's Compensation Acts, a predominant factor was the patient's mistaken idea of his rights to a lump sum payment. Actually, in the Workmen's Compensation Act, contrary to the belief of all patients and most doctors, there is no right to lump sum payment, except in those cases in which the accident proved fatal. The workman's sole "right" is to the payment of weekly compensation during incapacity from earning—and here again the Acts are misunderstood, because there is a vast difference between disability and incapacity from earning.

There is, however, provision in the Acts for the lump sum settlement of claims by agreement between the parties, subject to the settlement being passed by the Registrar or Judge of the County Court. Since this settlement is not a "right" but only an "agreement", it leads to endless and often bitter wrangling.

Throughout all this the workman's determination to defend his imagined rights becomes more and more fixed, and the ground for the development of all the mind disorders which Dr. Bernard Hart has dealt with becomes more intensely fertilized.

(2) *The Pest of the Lump Sum Settlement*

Now that the turmoil of the fight for "right" is over, the workman has been paid his lump sum. He has repaid out of it money advanced by the Public Assistance Board, and has settled his personal and often pitiful debts and perhaps been able to redeem some of his household possessions. Perhaps there is still a little money left over.

How far does this affect the period of invalidism?

A great deal of evidence on this subject was offered by the Stewart Committee, the Delevigne Committee, and before the Royal Commission on Workmen's Compensation. Before the Stewart Committee and the Delevigne Committee the evidence was almost unanimous in stating that the settlement of the claim by a lump sum payment operated to the physical and mental benefit of the patient and indirectly to his material welfare. The only contradictory evidence was that given by the representative of the Ministry of Pensions before the Delevigne Committee. Curiously all the other evidence was based upon opinion: the only evidence of fact was that given by the Ministry of Pensions.

But the evidence given before the Royal Commission on Workmen's Compensation was almost unanimous in condemning lump sum payment as a factor in rehabilitating the injured workman.

There can be no doubt that in the case of minor injuries rapid settlement of the claim by lump sum payment shortens the period of invalidism, but in the case of major disability with permanent incapacity, lump sum payment is an unmitigated evil.

An investigation was undertaken for the New York State Education Department by Dr. Carl Narcross. He investigated 321 cases three years after their settlement by payment of a sum of upwards of 1,000 dollars, with an average payment of nearly 3,000 dollars.

In the 67 neuroses that occurred amongst the 321, one case had made a complete recovery. The remaining 66 divided into three equal groups. The first 22 had improved and were back at work but their average weekly earnings were down by over 40%. The second 22 had not improved but they were back at work with their average weekly earnings down by over 60%. The third 22 had deteriorated: none was at work, 2 were dead, 17 were "on the rates". Of the remainder of the 321 several had developed neuroses since the settlement of their claims.

JOINT DISCUSSION No. 4

Sections of Neurology and Orthopædics

Chairman—GEORGE RIDDOCH, M.D.

(President of the Section of Neurology.)

[January 17, 1942]

DISCUSSION ON SCIATIC PAIN

Dr. Purdon Martin: "Sciatic" means referring to the socket of the hip-joint. The Romans called this socket "the vinegar cup" (acetabulum) and perhaps for this name there were more reasons than meet the eye of the anatomist.

By sciatic pain, I mean pain referred to the course and distribution of the great sciatic nerve and I do not apply the term to any other pain in the lower limb. The nerve is situated very deeply in the buttock, and runs down the middle of the back of the thigh, under cover of the long head of the biceps femoris. Its cutaneous sensory distribution is limited to areas on the foot and lower two-thirds of the leg, but this is not the distribution of sciatic pain. The nerve gives motor branches, and evidently also, branches conveying deep pain sensation, to all the muscles on the back of the thigh, and to the muscles on the back and outer side of the leg. While there may be hyperæsthesia in the cutaneous distribution, sciatic pain is typically a deep pain, and is felt in the muscles. The pain may be severe, but even when it is not severe, there is a sensation of discomfort in the muscles and the patient moves them frequently to try and get relief. In many cases there is also pain in the buttock. Among the associated phenomena are tenderness along the course of the sciatic nerve and pain excited when the extended leg is flexed at the hip. The former is usually attributed to tenderness of the nerve trunk itself, but it is doubtful whether this is always justifiable. I have observed that the muscles on the outer side of the leg may be tender when there is no tenderness of the common peroneal nerve at the neck of the fibula. Possibly the tenderness attributed to the sciatic nerve is often due to hyperalgesia of the muscles, and if so, the long head of the biceps femoris and the calf muscles must be particularly sensitive. As regards the so-called stretching of the nerve, it is doubtful whether the nerve is actually stretched by the usual manoeuvre, and it seems more likely that the pain which is excited is due to the stretching of the hyperalgesic muscles. There is a degree of spasm of these muscles. The pain has been attributed to stretching of the inflamed nerve and even to adhesions between the nerve and surrounding tissues, but in cases of referred sciatic pain this phenomenon is present but it can be abolished at once by anæsthetization of the exciting focus of the pain, so that it cannot in those cases be due to inflammation or adhesions.

Causes of sciatic pain.—We recognize two groups of causes of sciatic pain, viz. disturbances affecting the nerve itself, and pain referred into the distribution of the sciatic nerve. As the latter is the more common I will refer to it first.

Mackenzie's conception that referred pain was due to what he called "an irritable focus in the spinal cord" has stood the test of time, and it received strong support from Sherrington's demonstration of a "central excitatory state" affecting the motor cells in the spinal cord. The cells implicated in referred pain are those situated in the posterior horn of the grey matter, and these form in the cord a continuous column which is not segmented. While referred pain produced by a moderate stimulus is more or less segmental, it may, with a more intense exciting cause, affect several segments, or, on the other hand, only a portion of one segment, or adjacent portions of adjoining segments. It is consequently spinal, but not strictly segmental, and we should not expect to find it so. Referred pain causes a hypersensitive state in all the structures within a certain territory and consequently the muscles, as well as the skin are hyperalgesic. It may cause some exaltation of function, but it cannot cause any diminution of function or any impairment or abolition of reflexes. The chief sources from which pain is referred into the sciatic distribution are: arthritis of the hip, and perhaps also arthritis of the lumbar spine, disturbance of the spinal ligaments and local painful areas in the lumbar and gluteal muscles. In certain cases of malignant disease of the lumbar vertebræ pain may be present in the sciatic distribution at a time when no abnormality can be detected in

the claim by the payment of the £10 which he wanted. Other counsels prevailed; the man had his nerve injected with alcohol; he next had the finger amputated at the distal interphalangeal joint and then at the proximal interphalangeal joint; at a later stage at the knuckle. The next operation removed the 5th metacarpal bone, but still intense pain persisted. The last operation was for amputation of the ring finger and of the 4th metacarpal bone—all of the hand supplied by the ulnar nerve. The case was finally settled by a payment of £600 and the man, I am informed, lost his pain. Let it be clearly understood that this man was *not* malingering. He *did* have genuine return of pain with increasing severity. He *did* develop bulbous nerve endings which were easily seen beneath the skin.

Finally, the ignorance of the doctor who treats the patient or supplies the weekly certificate is as much to blame as any other factor in the Compensation law in keeping the man away from work. Because the man has lost a digit, or has a little shortening of the leg, or has not got full movement of a joint, he is advised by the doctor that he is not fit to do the work of a greasy birlor or of some other occupation of which he has not the slightest idea what are or are not its anatomical or physiological requirements.

The tragedy of the Workmen's Compensation law is that it was born out of season, before National Health Insurance.

Prof. Hermann Levy (co-author of "Workmen's Compensation", Vols. I and II, Oxford) said that the Workmen's Compensation Acts in Britain not only lacked any provisions to accelerate the recovery of the injured worker, but actually contained many provisions which tended to prolong sickness. Lump sum settlements were only one instance. They were not the result of any recommendations made by the medical profession, but mainly provided for the benefit of the insurance companies which liked to clear their accounts of liabilities and which used such agreements as a means of bargaining. Further, the will of the injured to resume work was constantly hampered by the insecurity of his future position. Would he get "light work" and what kind of light work? What would happen to him if by resuming some work there might come an aggravation or recrudescence of the injury? Insurance companies were eager to reduce the compensation payment from that for full incapacity to that for partial incapacity, but the worker's doctor was frequently disposed to reject offers of "light work" and even of operations in the interest of the injured. There was in Britain no compensation fund for second injuries, and this led to a reluctance on the part of employers to employ partially disabled workers. All such factors coupled sometimes with an endless litigation led to a feeling of insecurity on the part of the injured worker which again reacted unfavourably on his will for recovery and created neurotic fear. The creation of a Workmen's Compensation Board with a Board of Medical Referees, both being representative of employers' and employees' interests, would in conjunction with measures for rehabilitation and the re-settlement of partially disabled workers bring the necessary improvement of the present position. Moreover, groups of industry might form *collective* bodies dealing with accident prevention, compensation and rehabilitation under the guidance and control of the National or Regional Workmen's Compensation Boards.

indications that the inflammation is more widespread in the lumbosacral plexus. For instance, I have noticed that there is very frequently a tender point below the outer part of the crest of the ilium, about the termination of the iliac branch of the ilio-hypogastric nerve, which is derived from the 1st lumbar root. The course of these cases, too, is different from that of the cases of ruptured intervertebral discs, and does not show the remissions which characterize the pain of the latter.

Mr. V. H. Ellis: Sciatic pain may be due to causes (1) arising within the nerve or its central connexions; (2) producing irritation of the nerve or its roots from without; (3) stimulating nerve endings of similar segmental distribution in other areas of the body from which pain is referred to the sciatic area.

(1) In the first class we have the classical cause of sciatic pain, namely, neuritis. Sciatic pain is associated with tabes, with diabetes and presumably, therefore, with other toxins. Causes arising in the first class are by far the most infrequent cause of sciatic pain.

(2) Irritation of the nerve or its roots from without, is now attracting attention owing to its proved frequency, its more definite pathology and its successful treatment. It must be remembered that not only lesions of the intervertebral disc come into this class but also intraspinal tumours, subarachnoid adhesions and possibly also irritation of the roots by spinal osteophytes and tumours in the pelvis. For instance it has been found that 8% of cases of tuberculous caries of the sacro-iliac joints present sciatic symptoms. It is often said that sciatic pain can be due to the pressure of a tumour on a nerve root. But pressure on a nerve most usually produces paralysis or anaesthesia. Movement plus pressure is required to elicit pain. This factor may account for some of the successes of empirical treatment which will be referred to later.

(3) Before discussing in more detail the pathology and treatment of this class of sciatic pain, the third class must be considered. It is probable that the majority of cases of sciatic pain are of the referred type. It is true that most of them are of the more transient and less severe kind. A lesion of any type of tissue could be responsible for this stimulus though I think skin and the viscera are unlikely, but fascia, muscle, ligament and periosteum may provide the focus. Whatever we may believe about the pathology and causation of fibrositis this is undoubtedly a common cause of sciatic pain.

Injuries and the irritation of nerve endings in ligaments have been proved experimentally to cause sciatic pain, anaesthetization of such a ligament has relieved it. Ligaments are most commonly injured by stretching, either sudden or gradual, and the interspinous ligaments are obviously liable to both postural and traumatic strains.

Let us consider the causes of sciatica which have recently sprung into prominence. Rupture of an intervertebral disc suggests a severe traumatic strain of an intervertebral joint (if we exclude clumsy lumbar puncture). Hypertrophy of the ligamentum subflavum suggests a similar aetiology. With interspinous ligament strain we have here three real causes of sciatic pain which may often be associated.

However, I submit that movement is required to elicit pain in each and all of these cases for the following reasons: (1) Sciatica can nearly always be relieved by complete immobilization of the lumbar spine. Even the relative immobilization of plaster of Paris is often effective. (2) Fusion of the lumbar spine relieves symptoms in disc and other lesions (Farrell, B. P., and MacCracken, W. R., *J. Bone & Joint Surg.*, 1941, 23, 457). (3) That prolapse of the disc is not the only operative factor in many cases is suggested by (a) J. S. Barr and W. J. Mixter (*J. Bone & Joint Surg.*, 1941, 23, 444) who found that fusion after laminectomy gave better results (see Table below); and they say that 30%

| | | | Results with fusion | | | Results without fusion |
|---------------------|-----|-----|------------------------|-----|-----|---------------------------|
| Persistent sciatica | ... | ... | 9% | ... | ... | 31% |
| Low back pain | ... | ... | 27% | ... | ... | 48% |

of cases should be fused. (b) Watson Jones recommends posterior root division after disc removal to avoid recurrences (Watson Jones, R., "Fractures", Edinburgh, 1939, p. 252).

With these considerations we begin to see a much wider cause of sciatica—what may be called intervertebral strain of which the ruptured disc may be merely a complication.

Laminectomy for sciatica where no prolapsed disc has been found fortunately often cures the symptom as well as allowing removal of lipiodol. Does the removal of a strained ligament cure it? If, as I suppose, ligamentous strain is a common cause of sciatica, Kellgren's method of novocain injection will often be of value in this as I have often proved it to be in the so-called fibrositic type. It also explains some of the occasional successes and aggravating failures of manipulation.

X-ray films of the vertebral column; there is no loss of the ankle-jerk, or other sign of impairment of the function of the sciatic nerve, and I believe the pain in these cases at this stage is a referred pain arising from the diseased bone. The treatment of referred pain is the treatment of the primary source, usually the hip-joint. Local foci of exciting pain can sometimes be treated by injection. Wilfrid Harris was using this method twenty years ago, employing quinine urca, and later, very small injections of alcohol, and in the last few years Kellgren has used novocain preparations with considerable success.

The disturbances which may affect the nerve itself or its roots, are many, but I will refer only to protrusion of the nucleus of an intervertebral disc, and to interstitial neuritis. It is now three years since Love came over here and gave us a paper on protrusion of the nucleus of the intervertebral disc (*Proceedings*, 1939, 32, 1697), but we are still somewhat weak in the diagnosis of this condition. The symptoms, if the lesion is in the lumbar spine are: sciatic pain, pain in the back, stiffness of the spine and some deformity of the spine, such as reduction of the normal curve, or scoliosis; the pain is intensified by coughing or sneezing, and typically it is subject to remissions. The protruded nucleus irritates an emerging spinal root and so causes pain referred to the distribution by that root. This is undoubtedly the same condition as the *sciaticque radriculaire*—a sciatica affecting a single spinal root—described by Déjerine in the early years of this century, and the exponents of protruded intervertebral discs have not yet added anything to the account which Déjerine gave of the symptomatology of "radicular sciatica". In these cases in which usually a single spinal root is affected the problem of diagnosis resolves itself, as in other neurological conditions, into the two questions: Where is the lesion? What is the lesion? The nervous signs and symptoms enable us to answer the first question; for the answer to the second we must turn to the general examination of the patient, special investigations and the history. The site of the lesion causing irritation of a single root is determined by the distribution of the pain and by signs of interference with root function, such as sensory impairment, or loss of the ankle-jerk (usually weakened with the 5th lumbar and lost with 1st sacral lesions). Dandy, in his most recent paper, quotes Love for the statement that 96% of all disc lesions affect either the 4th or 5th lumbar intervertebral disc, and consequently the roots involved are the 5th lumbar or 1st sacral. To recognize with confidence the nature of the lesion we must determine the syndrome associated with injury of the disc itself and so far it is not very well defined. The pain in the back is probably a referred pain arising either from the damaged disc or from ligaments under stress. The spinal deformity is either due to the injury to the disc, or, more probably, is protective. I do not think the possibilities of diagnosis by clinical observation are yet by any means exhausted. A history of injury is suggestive but can be misleading. As regards special investigations, the simple X-ray appearances are not as a rule very helpful in the differential diagnosis, and the C.S.F. findings are by no means typical. We are consequently dependent on the use of X-ray examination with contrast media. At first we depended on lipiodol and, more recently, air has been used. Lipiodol is not to be used lightly, and it should not be injected unless the X-ray examination can be carried out under the most favourable conditions. I believe as we become more familiar with this condition, and recognize the clinical syndrome arising from rupture of the disc at whatever level, we shall be able to make the diagnosis without the use of contrast media. In fact, I believe that we shall do better than with contrast media, because there must be some cases in which the protruded nucleus does not cause any constriction of the theca. and in such cases contrast media may mislead us.

In dealing with the cases in which sciatic pain is a consequence of the rupture of an intervertebral disc, we have, perhaps, not given sufficient weight to the fact that there are two more or less separate conditions to be treated. The irritation of one of the roots of the sciatic nerve by the extruded nucleus of the disc is, in fact, only a complication, and when the irritating element has been removed by operation, we have still only treated the complication. It is true that there are many fortunate cases in which the primary condition—the injury of the vertebral column—gives rise to few symptoms, but we cannot reasonably expect that the patient will be relieved of all his symptoms unless the primary condition is treated, as well as the complication.

Finally, there is such a thing as sciatic interstitial neuritis. A similar clinical condition may be observed in many other spinal nerves where the irritation is certainly not due to protruded discs and it is very often associated with arthritis. Recent pathological confirmation for the sciatic nerve is admittedly lacking, but Ballance and Duel found in the facial nerve thickening of the interstitial tissue, and thickening and scarring of the sheath. In the sciatic nerve the outstanding symptom is pain, but there are signs also of interference with nerve function; the ankle-jerk is likely to be lost and after a time there is slight wasting or flattening of the calf muscles. The signs and symptoms in these cases of neuritis are not localized to the distribution of a single spinal root; there are usually

about is the relative importance of one cause as against another. Some authors find arthritis of the hip the commonest cause. Others claim priority for low back strain. A third group accepts herniation of the nucleus pulposus as the most important factor, and so on. I suppose that to some extent these differences of opinion are due to the selected nature of the material which the specialist is called upon to see. I have collected a small series of unselected cases which were studied in order to view the sciatic problem as a whole. Of 115 cases seen, the pain was of true sciatic distribution in only 84. Pain limited to the posterior cutaneous nerve of the thigh was not regarded as sciatica. The findings are tabulated hereunder:

| DIAGNOSIS | | | | | | | | No. |
|----------------------------------------------|-----|-----|-----|-----|-----|-----|-----|-----|
| Fibrositis | ... | ... | ... | ... | ... | ... | ... | 16 |
| Neuritis | ... | ... | ... | ... | ... | ... | ... | 3 |
| Radiculitis | ... | ... | ... | ... | ... | ... | ... | 2 |
| Osteo-arthritis of L. 4-5 | ... | ... | ... | ... | ... | ... | ... | 4 |
| Osteo-arthritis of hip | ... | ... | ... | ... | ... | ... | ... | 8 |
| Sacro-iliac arthritis | ... | ... | ... | ... | ... | ... | ... | 10 |
| Tuberculous(2) | | | | | | | | |
| Subacute, origin obscure (5) | | | | | | | | |
| With homolateral sacralization (3) | | | | | | | | |
| Pott's disease of L. 4-5 | ... | ... | ... | ... | ... | ... | ... | 3 |
| Malignant secondaries of L. 4-5 | ... | ... | ... | ... | ... | ... | ... | 7 |
| Tumour of cauda equina | ... | ... | ... | ... | ... | ... | ... | 3 |
| Protrusion of nucleus pulposus (lumbosacral) | ... | ... | ... | ... | ... | ... | ... | 2 |
| Chondroma of intervertebral disc (L. 2) | ... | ... | ... | ... | ... | ... | ... | 1 |
| Malingering | ... | ... | ... | ... | ... | ... | ... | 1 |
| | | | | | | | | 60 |
| Diagnosis unproved | ... | ... | ... | ... | ... | ... | ... | 24 |
| Total | | | | | | | | 84 |

The cases of fibrositis were clear cut, with evidence of muscular rheumatism in other parts of the body. Where the diagnosis rested on the novocain injection method introduced by Steindler, the case was regarded as unproved owing to my experience that this test is occasionally unreliable. Neuritis, with its severe pain, hyperæsthesia, absent ankle-jerk, and prolonged course, so unlike any other cause of sciatic pain, was seen thrice. There were two young men with a high sciatica whose cerebrospinal fluid showed a protein of 110 mg. and 90 mg. respectively; laminectomy after equivocal lipiodol examination, was completely negative, and both cases made a slow recovery. I think these conform to the early descriptions of radiculitis. In four cases there was reason to suspect that sciatic and low back pain was associated with severe osteo-arthritic changes in the lower lumbar vertebræ. Professor Putti drew our attention to the possible importance of such a lumbar arthritis in the causation of sciatica. The difficulty is to demonstrate that the arthritis seen radiologically is the cause of the symptoms. My reason for tracing such a connexion in these four cases is that the injection of novocain into the intervertebral canals (i.e. the site of osteo-arthritic inflammation and compression) of the lower two lumbar roots gave complete though temporary relief from pain. In three cases the 5th root only was affected. In the fourth case both the 4th and the 5th foramina had to be injected. Osteo-arthritis of the hip appeared to be the source of the pain in eight cases, for the sciatic pain could be relieved by filling the joint space with isotonic 2% novocain. A supra-trochanteric approach was used, and the procedure was without ill-effects. The reason for doing it at all was the difficulty in knowing whether the sciatic pain was due to the arthritis itself, or whether it originated in an associated fibrositis or in the lumbar spondylitis which was so frequently present. In two cases, listed as undiagnosed, cocaineization of the hip failed to relieve the pain, whereas a deep injection into the region of the transverse process of the 5th lumbar vertebra had an immediate and permanent effect. The fact that tuberculous and pyogenic disease of the hip do not give rise to sciatic pain is a further warning against assuming a direct connexion between the symptoms complained of and the arthritis which may be present. The diagnosis in ten cases of sacro-iliac arthritis rested on the radiological evidence of extensive and progressive destruction of one sacro-iliac joint as against a normal on the sound side. The clinical tests for disease in this joint were consistently unreliable in these cases; pain antedated both localizing signs and X-ray evidence by many months in some instances, leading to the use of lipiodol in one case. No cases of sacro-iliac strain were encountered. Two women presented the classical picture of sacro-iliac subluxation, but in both cases the pain was confined to the back of the thigh and they were therefore excluded from the series. I mention this to indicate to our orthopædic colleagues that the absence of these

Sceptics of the teachings of Leriche do not understand how novocain cures sprained ligaments. But the treatment is effective, and I believe a large number of cases of sciatica would be amenable to this form of treatment if the affected ligaments could be found, and injected, which may be technically very difficult. There seems no reason why anterior as well as posterior ligaments in the lumbosacral or even sacro-iliac region may not be at fault.

Before considering the treatment of sciatic pain the differential diagnosis of the three types must be determined.

The first presents the greatest difficulties unless other nerves are affected in the same manner. The absence of a history of trauma or precursory low back pain with the evidence of some general disease or toxæmia, would be helpful. Tenderness of the nerve itself is difficult to differentiate from tenderness of the structures overlying the nerve which may be present in any case of sciatic pain. Relief of pain following anaesthesia of the sciatic nerve trunk would be very suggestive though I have never yet been successful in such an attempt (possibly because I have never met a case of sciatic neuritis) and even this procedure would not relieve a radiculitis.

Differentiation between the second and third classes should be easier, but does not always prove so in practice. Possibly this is because, as already pointed out, both classes may be present in the same case. With definite pressure upon and irritation of a nerve root, signs as well as symptoms might be expected and in fact they are seldom absent in well marked cases. Loss of ankle-jerk is not uncommon, though possibly only when the 1st sacral root is involved. It is difficult to see how loss of ankle-jerk could be the accompaniment of purely referred pain. Similarly actual weakness of muscles supplied by the sciatic nerve suggests direct involvement of the parent root. Although anaesthesia is not to be expected from the involvement of a single root, changes in sensation are common and may be due to partial denervation of the skin as suggested by Weddell [*Proc. Roy. Soc. Med.* (1941), 34, 777 (Sect. Neurol., 33)] in recovering nerve lesions. Though local signs are therefore usually present in a well-marked case they are generally insufficient to prove or to localize the lesion. The history is of great importance; although considerable trauma might reasonably be required to rupture a disc such a story is often not obtained, mere forward flexion or weight lifting being held responsible.

One curious thing is the rarity of sciatic pain following compression fracture of the spine and tuberculous disease, in both of which the intervertebral disc is grossly affected.

The diagnosis of referred sciatic pain is largely made by elimination of the second class, by finding evidence of fibrositis or tenderness due to local trauma in the low back. Kellgren's method of novocain injection is both diagnostic and curative. I believe that cases of this class rarely present clinical signs in the affected limb.

Treatment.—Movement as well as other factors being usually required to produce sciatic pain, it is obvious that immobilization of the lumbar spine will usually give relief. This may vary from rest in bed, through the fixation in plaster, to fusion of the spine and it is questionable at what point the patient will consider the treatment worse than the disease. Application of a plaster jacket in suspension, possibly prolonged into a short hip spica will give relief even in many cases of protruded disc. It is possible that here in addition to immobilization reduction of the disc may occur. It is doubtful whether such a reduction can be permanent by healing of the ruptured fibrous ring and relapse often occurs on mobilization. I have had what appears to be permanent relief in non-fibrotic cases of sciatic pain both by this method and by simple traction of the affected limb in recumbency. A 10 lb. weight applied by strapping extension with the recumbent patient on a tilted bed is easily applied. It will give relief in two or three days if at all. It is not too tiresome for the patient and is without risk. If it gives relief it should be continued for three weeks. Although manipulation of the spine under anaesthesia gives relief to some cases it is dangerous unless the possibility of a ruptured disc is first excluded.

Omitting the effects of the trauma of operation which are rapidly decreasing with improved technique, the mechanics of the spine are abnormal both before and after operation. Removal of the protruded disc may relieve the symptom but one hydraulic shock absorber is out of action, one set of interspinous ligaments may still be seriously strained, and the faulty mechanics which allowed the original lesion are still present. Orthopaedic treatment to correct posture or improve muscle tone may prevent chronic low back pain if not a possible recurrence of sciatic pain.

Dr. Frank A. Elliott: Since the sciatic nerve and its radicles are the final common path for impulses from a number of important structures, it is to be expected that sciatic pain may have many causes. So much is generally admitted. What there is no agreement

below the knee, a diminished or absent ankle-jerk, and slight sensory impairment over the lateral border of the foot and ankle. But not all of these signs are present in every case. The commonest seems to be slight muscular weakness, especially in the dorsiflexors of the foot when these muscles are tested in action against resistance. This slight kinetic weakness is rarely of an order to have been noticed by the patient or to cause any abnormality of gait. Next most frequent is a slight impairment of cutaneous sensibility: loss of the tickle sense in testing with cotton-wool, and slight impairment to pin-prick over the lateral border of the foot and ankle. This latter examination demands some care, but the information it yields is worth the time spent in eliciting it.

The cerebrospinal fluid in the majority of cases is normal; a slight increase in the protein content is not uncommon, but a great increase should direct attention to one of the rarer causes of sciatic pain, such as an inflammatory process, tumour, angiomatous malformation, &c. At this point, I should say that in the cases of massive protrusions causing compression of the cauda equina, a great increase of protein is a common finding, and it is interesting to note that in these cases the fluid is usually withdrawn from above the level of the lesion.

Ordinary X-rays of the spine are generally of little help. They will show the flattening of the lumbar curve, and the scoliosis which is commonly present, but the lesion itself is not visible. Nor is it often possible to say with certainty that a given intervertebral space is narrower than it should be, and hence the likely site of a protrusion of nuclear material.

Short of operation, the lesion can be demonstrated most convincingly by contrast myelography. In some clinics, air is used as the contrast medium and it is said to be very satisfactory. My own limited experience of the technique has not encouraged me to pursue it in the investigation of these cases. Lipiodol myelograms are easier to interpret, but as the stock of lipiodol in this country dates from before the fall of France, most of it now contains so much free iodine that it may set up troublesome irritant reactions in the theca, and on the whole it is better not to use it. With an adequate history and examination, it should be possible to make the diagnosis without contrast myelography, and if there was any doubt I should prefer an exploration to the instillation of lipiodol.

Cases which have been submitted to operation have been (1) cases of severe sciatica which in a given attack (usually not the very first) have not responded to rest or other measures; or (2) cases of recurring sciatica in which the frequent recurrences have led to domestic or economic hardships, or (3) more rarely, cases of compression of the cauda equina resulting from a massive protrusion. In this last connexion I have seen one such case following a manipulation for sciatica, and I know of others. Even though this is a rare sequel of manipulation, it is such a serious one, that I would be very hesitant about advising manipulation for this complaint.

As to the technique, the lesion is much more easily seen and dealt with by the extradural than the intra-dural approach. I have seen several cases where there was nothing at all abnormal to be seen or felt within the theca, and yet the lesion was perfectly obvious when approached extrathecally. This may explain a certain number of negative explorations. In some cases, it is possible to deal with the lesion without removing any bone: the sacrospinalis muscle is cleared and retracted from the spinous processes and laminae on the side of the lesion, the ligamentum flavum excised, and the lesion removed in the interval between the laminae. In this manner the operation is limited to soft tissues, and it seems that convalescence is a little more rapid, and there may be less disturbance of the mechanics of the vertebral column than is entailed in the usual hemilaminectomy.

Group Captain C. P. Symonds said that in the discussion opened by Dr. Love in 1939, he had stated that the clinical syndrome reported in patients with prolapsed disc proved at operation was one which he had observed in scores of patients who had recovered with rest and warmth, without surgical intervention. He concluded that if prolapsed disc was anything but a very rare cause of sciatica it was a lesion in most cases capable of spontaneous repair. He had nothing to retract from this statement but something to add from his experience with Service patients in the past two and a half years. He was convinced that in this group prolapsed disc was by far the commonest cause of sciatica. He further believed that there was a clinical syndrome characteristic of prolapsed disc, and that the diagnosis could therefore be made without recourse to contrast media. He was not yet convinced that operation offered a short cut to recovery in an attack of sciatica from this cause, or that the prospects of recurrence were any less in cases treated by operation than in medically treated cases. As far as Service patients were concerned operation, even in the best hands, had proved on the whole so unsuccessful in getting men back to duty that his present policy was to advise a prolonged period of immobilization and if this failed to render the man fit for duty within a reasonable time, to recommend invaliding.

conditions from this small series does not mean that they were not looked for. They were, and with orthopædic assistance. I am surprised that Mr. Ellis should not have seen sciatica as a symptom of spinal tuberculosis; these three patients presented cold abscesses and were old neglected cases which would not normally have been seen by an orthopædic surgeon. There were seven cases of secondaries in the lower spine; in all seven the pelvis was clinically free from growth. Unless this is the case it is of course difficult to know whether the sciatic pain comes from the pelvis or the vertebrae. Tumour of the cauda equina was found three times, and it is interesting to note that one of these had suffered from lumbago and sciatica for seven years, dating from a strain of the back while lifting a heavy weight. She was in consequence treated as a low back sprain until neurological signs appeared. A confident diagnosis of herniated disc was then made, and at operation a neurofibroma was discovered to be the sole abnormality present. In the course of six laminectomies a herniated disc was found in two cases, radiculitis was present in two cases, in one there was a chondroma of the second lumbar disc; no abnormality was found in one, but the operation was curative and there has been no recurrence of pain for two years. Finally, there was one malingerer who had an absent ankle-jerk dating from a previous attack of sciatica, which the patient was able to reproduce in order to secure his discharge from the Army. This information was forthcoming during light narcosis. I include it because the psychological aspect of sciatic pain is a definite problem. Long-standing cases sometimes develop hysterical symptoms, and if seen for the first time, give the impression that there is nothing organically wrong. Oppenheimer described this long ago as "reflex hysteria". Another variation is an anxiety state grafted on to the organic nucleus and aggravated by the doubts and dissensions of too many bedside consultations.

Mr. Joe Pennybacker: It seems reasonable to approach the larger problem of pain in the lower limb by limiting the discussion to that group of cases complaining of pain in the sciatic distribution, with physical signs referable to the sciatic nerve or its component roots, and accepting only these as cases of sciatica. It is true that even this limitation will carry us to the borderland of the low back pain country, as a great number of cases of ordinary sciatica have had periods of low back pain.

My experience in the last few years has led me to the belief that the majority of these cases of sciatica are due to compression of one of the lower lumbar roots by a damaged intervertebral disc. When I tell you that I have seen this lesion verified at operation in some sixty cases in the past two and a half years, you will appreciate my difficulty in believing that this is a rare cause of sciatica. It is nearly three years since Love addressed a combined meeting of these sections, and focused attention on this lesion as a common cause of sciatica. That we have been slow in accepting it as such is perhaps characteristic of our conservatism in matters of treatment, and as long as we are willing to assess results without bias, there is something to be said for this attitude.

The cases on which I base these remarks were cases of ordinary sciatica; at least, all of them had been regarded as such by numerous therapists who had met with indifferent success in dealing with them. You may say that the fact that they had not responded to treatment takes them out of the class of ordinary sciatica, which in the vast majority of cases recovers spontaneously or in response to various physical measures, and never recurs. I doubt the validity of that reasoning, and mere impressions based on a long experience are not enough. It would be interesting to follow up a series of cases of sciatica treated conservatively, to see what has happened to them over the years. I know that some of the cases which would figure in such an analysis have ultimately had an operation for the removal of a prolapsed intervertebral disc.

There are several points in the syndrome which are not sufficiently appreciated. The possibility of such a lesion must be remembered in every case of sciatica, and the diagnosis can often be made from the history. Although the primordial pathology is not clearly understood, it seems reasonably certain that the lesion is traumatic. But the injury is usually a trivial one, and perhaps strain is a better term. The strain commonly occurs in flexion of the trunk: in lifting from the bending position, for instance. The injury may have been so trivial that it escaped notice or was forgotten, and frequently its discovery in the history is only at the expense of direct questioning. The immediate damage to the disc is followed by low back pain, often miscalled lumbago even by doctors who have had the affection in their own persons. This pain may be severe and disabling for a few days or it may only cause a slight discomfort in the back. It is easy to see how such an incident may be forgotten and not related to the sciatica. Even in the cases in which the sciatica follows more closely on the back pain, the memory of the back pain may be swamped in the more severe and present sciatic pain.

The neurological examination in the classical case reveals pain on the straight-leg raising test, some tenderness over the course of the sciatic nerve, weakness of the muscles

Section of Laryngology

President—E. D. D. DAVIS, F.R.C.S.

[March 6, 1942]

DISCUSSION ON INJURIES OF THE NOSE AND THROAT

V. E. Negus: The *proportion of injuries* in the various regions is illustrated by a table showing the cases of injury to the head and neck treated at Horton Emergency Hospital. The table demonstrates that of all the head injuries admitted, just over half were cranio-cerebral, while the remainder affected regions other than the cranium. The nose and sinuses suffered relatively infrequently and the pharynx and larynx scarcely at all.

INJURIES OF THE HEAD AND NECK. ADMISSIONS TO HORTON EMERGENCY HOSPITAL FOR 8 MONTHS.

| | Civilian | Service | Total |
|-----------------------------------|----------|---------|-------|
| Total cases | 1,476 | 2,441 | 3,917 |
| Total head and neck cases | 372 | 205 | 577 |
| Cranio-cerebral | 214 | 113 | 327 |
| Other regions of head and neck | 157 | 126 | 283 |
| Face | 55 | 32 | 87 |
| Eye | 92 | 34 | 126 |
| Nose and sinuses | 10 | 5 | 15 |
| Mouth and tongue | 0 | 2 | 2 |
| Mandible | 5 | 7 | 12 |
| Neck | 4 | 10 | 14 |
| Pharynx | 0 | 1 | 1 |
| Larynx | 0 | 1 | 1 |
| Trachea | 2 | 1 | 3 |
| Bronchus (F.B.) | 0 | 1 | 1 |
| Ear | 39 | 35 | 74 |
| Wounds of other parts of the body | 51 | 31 | 82 |

NOTE.—The added total of head and neck cases is 610; the excess of 33 over the number given as 577 refers to combined injuries.

Nose

Hæmatoma of the septum is to be expected and if present, free incision and evacuation are required, particularly if infection has occurred.

Much can be done to prevent infection if it is realized that the vestibule of each nasal fossa is heavily infected, while the regions lined by mucous membrane are practically sterile.

Mercurochrome in 2% solution or biniodide of mercury 1:500 in spirit, can be used, both for the vestibule and exterior of the nose. To maintain asepsis the patient should wear a small roll of gauze held in position over the nostrils by tapes tied behind the head.

For fracture of the nose with displacement, I have found a splint made of successive strips of broad ribbon gauze, to which collodion is applied with a brush, very convenient. Each layer is allowed to dry partially before the next is applied; eight thicknesses are sufficient.

Para-nasal Sinuses

Simple injuries to the sinuses may be confined to effusion of blood into the antrum. Such a collection of fluid is best left alone, as it will in all probability absorb without immediate or subsequent trouble; here again, prevention of infection from the nostrils is of importance.

Simple fracture of the walls of sinuses may not call for treatment, except in special instances.

Depression of the lachrymal bone associated with inward displacement of part of the ethmoid plate, may lead to obstruction of the fronto-nasal duct, with subsequent headache; manipulation into normal position can be carried out with Walsham's forceps.

Compound fractures into the sinuses may present both cosmetic and functional diffi-

Dr. Henry Wilson: The complaint of pain simulating neuralgia and neuritis interests the medical psychologist since he is asked to see such cases when medical, surgical, and physiotherapeutic measures have been unsuccessful.

That hysterical pain may occur in any organ is well known, but it only occurs if one of two conditions have been present: (a) Previous organic pain in that part; (b) in a dubious and much smaller number of cases, as a result of suggestion.

I have reviewed the notes of eighty patients with primary sciatica admitted to the London Hospital between 1937 and 1941 inclusive.

It was remarkable to find that house physicians, or registrars had mentioned irregularities in the patients' mental attitude in no fewer than twenty-five of the cases. In arriving at this figure I have disregarded the word "anxious", since any patient with serious sciatic pain may well be this, nor have I included the word "exhausted" unless that has been expanded by other suggestions of neurosis. Thus to give a few examples: "always been a worrier", "suspicious", "psychoneurotic", "each form of therapy seems to occasion a fresh burst of-complaints", "came on when he had a considerable amount of worry". In one woman with prolapsed discs it is stated that after ten days in hospital "a remarkably quick improvement occurred when she wanted to go home"; one case immediately recovered when an epidural injection was proposed.

There seem to be two possible explanations of the frequency with which neurotic manifestations have been noted by non-skilled observers. The first is that quite apart from the initial pain the prolonged rest in bed produces those symptoms of irritability which may be shown by the most healthy when inactive. My impression from the notes is that the deviation from the normal in these cases was greater.

The second explanation is that a crippling and fairly lengthy illness encourages the persistence of originally organic symptoms as a hysterical aftermath used for escapist or other purposes.

If the question of suggested pain is disregarded hysterical pain in any part of the body is never primarily (in time) psychogenic. Pain at some time (remembered or not) has affected that area. Given the neurotic constitution together with the current problems organic sciatic pain may merge into hysterical sciatic pain, or hysterical sciatic pain may recur some time after the original attack of organic pain. The former wasting and altered reflexes may persist, and suggest a recurrence of organic disorder. Epidural injections and manipulations may be welcomed by the patient as focusing the attention away from the real seat of hysterical pain, i.e. the mind.

Is there any means whereby the perpetuation of symptoms for unconscious purposes may be lessened?

If the first explanation advanced above is correct, i.e. that neurotic manifestations are the results of inactivity and invalidism, it is essential that directly the acute symptoms have subsided the patient has sufficient occupation for his active upper limbs and for his mind. Ways of achieving this were suggested by Jefferson [*Proc. Roy. Soc. Med.* (1942), 35, 295 (Sect. Neurol., 1)].

What of those other cases where the pain persists for escapist motives? The first necessity is that this possibility should be borne in mind, and that its frequency be recognized. Physical signs alone are misleading since absent ankle-jerks and wasted muscles may persist.

Although non-organic pain may serve exhibitionistic, masochistic, or propitiatory motives it begins as an escape mechanism, and may sometimes, possibly frequently, be a form of malingering. This occurs more often than is recognized, and is the more likely the longer the period of primary inactivity. To assert that sciatic pain following trauma or neuritis is sometimes hysterical does not imply the necessity of long psychotherapy.

What is essential is early diagnosis of a psychological perpetuating cause, which should be followed by prompt adjustment of the environment so that the gain of the hysterical illness is reduced, and the disturbing mental factors fairly faced. My impression is that the moment of psychiatric diagnosis is sufficiently early, long psychotherapy would be unnecessary, indeed it might well take the form of stimulating activity, once the escapist tendencies are recognized and discussed.

Larynx

Indirect effects on the larynx are produced by certain wounds of the neck in which the vagus nerve is bruised or pressed upon by a hæmatoma. It is improbable that a patient with a divided vagus will survive, owing to its position in relation to the great vessels, and, therefore, the injury is unlikely to be due to division of the main trunk.

The effect is seen in *paralysis of the recurrent nerve* supplying one vocal cord, with disturbance of phonation, but with no severe upset of respiration; bilateral paralysis, with dyspnœa, is unlikely to occur. No treatment other than removal of the foreign body, is practicable; the paralysis may recover.

Simple direct injuries are produced by falls on the larynx or by motor or aeroplane crashes; they are unusual owing to the protection afforded by the lower jaw and sternum. They do occur, however, sometimes with such severe effects as complete occlusion of the glottis.

It is a general rule, in simple injuries of the larynx, to find that fracture of the thyroid cartilage is not of itself of serious import, but that fracture of the cricoid is dangerous because of subsequent obstruction to respiration. Tracheotomy is then called for as a life-saving measure, and later, relief of stenosis may be possible; it is wise to wait until all active inflammatory changes have subsided.

The method recommended is opening of the larynx, division of adherent surfaces, which may include the whole of the opposed surfaces of the vocal cords and subglottic areas, and removal of prominent masses of scar tissue; a lumen can thus be restored. To maintain patency, an effective method is the insertion of a rubber tube, held in position by transfixion with silver wire, as recommended by Schmiegelow.

An essential addition is the application of a skin graft to the divided surfaces; healing is thereby accelerated and recontraction avoided.

It may be necessary to keep the rubber tube in for as long as two months, if the cartilaginous framework is much damaged. In other cases, where the thyroid and cricoid cartilages are intact, as short a time as twelve days may be sufficient to obtain a permanent result.

Sometimes it is impossible to restore the airway sufficiently to permit of normal respiration and no more can be done than reconstruction of a passage sufficient for purposes of speech.

Compound fracture is of more serious account because of the danger of infection and particularly of perichondritis. Free exposure of the wound is required, with excision of lacerated skin edges; sulphonamide powder should be dusted into the wound, which is then kept widely open by a loose packing of gauze impregnated with iodoform. Tracheotomy may be required if much swelling is produced by hæmatoma formation or by inflammatory swelling.

Penetrating wounds of the larynx or trachea are rarely seen because of the liability to associated injury of the carotid sheath or the vertebral column. Should the patient survive these immediate dangers, he will be exposed to various others; such as hæmorrhage, fatal of itself or from flooding of the trachea, sepsis in the wound, with spread to the lungs causing septic bronchitis or bronchopneumonia, and finally perichondritis.

The immediate treatment consists in keeping the lungs clear of blood by allowing the patient to cough in the sitting posture and without the centrally depressing effect of morphia, or by the use of a suction pump; and in controlling hæmorrhage by pressure or the application of ligatures.

If there is a wide enough wound into the lumen of the larynx or trachea, it is permissible to insert through it a tracheal cannula as a temporary measure; it is imperative, however, that the tube should not remain in contact with the laryngeal cartilages for more than a few hours, as otherwise perichondritis will follow. The right procedure is, therefore, a correctly placed tracheotomy as soon as practicable. The wound itself should be laid freely open.

Possible sequelæ are immobility of the crico-arytenoid joints, with mechanical fixation, of the cords, or stenosis from organization of granulations or from perichondritis.

The glottis and lumen of the trachea may be so narrowed that normal respiration is impossible; a permanent tracheotomy may be necessary, but sometimes it may be possible to restore the lumen by excision of scar tissue, with skin grafting, after all active inflammation has subsided.

Certain cases do not lend themselves to the operation described and it may be necessary to turn in skin flaps, to be united to the mucosa of the larynx or trachea and subsequently divided and folded in, so as to restore the airway.

Laryngo-œsophageal fistula.—A condition disturbing to the patient and difficult for the surgeon, is traumatic fistula between the larynx or trachea and the œsophagus. Food

culties. If the wound is clean, it may be possible to effect immediate closure without drainage. If, however, potential or actual sources of infection are present, it is desirable to provide free drainage, to cleanse lacerated skin edges, to remove foreign bodies and pieces of bone and then to dust the wound with sulphonamide powder. A full prophylactic course of sulphapyridine, 1 g. four-hourly, is given for three days. Failure to carry out this form of treatment, or excess of zeal in opening up bony layers or venous and lymphatic channels in early septic cases, may precipitate osteomyelitis, or thrombophlebitis, with possible spread to the meninges or the cavernous sinus.

After an interval of at least fourteen days, a further operation can be undertaken if necessary, according to the site and type of injury.

The *maxillary sinus*, if containing pieces of metal or fragments of bone, must be opened by the sublabial route as soon as possible after the injury, unless the wound itself is of sufficient size for the removal of foreign bodies. If infection is present or impending, it is wise to provide an opening into the inferior meatus of the nose.

Clearance of the *ethmoidal labyrinth* may be required in injuries of this region, if infection is present; the operation should be postponed for at least fourteen days and is best carried out by the external route.

If it appears that obstruction of the fronto-nasal duct might follow the operation, it is well to make a free communication between the nasal fossa and the frontal sinus and to insert a skin graft, held in place by a rubber tube.

The *frontal sinus*, if injured, may present the double difficulty of cosmetic repair and functional restoration. If the anterior wall is depressed, but still more or less intact, elevation may be possible. If, on the other hand, the wound is severe and the cavity of the sinus infected, there will be swelling of the mucosa and blockage of the fronto-nasal duct, particularly if the injury extends downwards to the nose. In such a case it is essential to provide a free passage into the nose by enlarging the fronto-nasal duct; the operation should include the insertion of a skin graft.

If there be destruction of the anterior bony wall, it may be desirable to restore the lumen of the sinus and to replace its mutilated lining with a skin graft in the form of a sac, held in position by a bag of oiled silk packed with ribbon gauze. I have on two or three occasions adopted this procedure with satisfactory results.

It is extremely difficult, if not impossible, to obliterate a frontal sinus and a pocket is liable to be left, with continuation of suppuration. The restoration of the lumen and provision of a free new fronto-nasal duct, eliminates this source of chronic infection and at the same time avoids the severe disfigurement resulting from destruction of the anterior wall. Insertion of fat or bone grafts in such cases offers difficulty because of the probability of sepsis.

Compound fracture involving the posterior wall of the frontal sinus is of serious import and a radical operation may be indicated.

A further complication of a wound involving the ethmoidal and maxillary sinuses, in one case under my care, was penetration of the *pterygo-maxillary fossa* by a piece of high explosive shell.

Fracture of the base of the skull may be of serious moment if it throws the meninges into communication with the nose or pharynx. The regions where this may occur are the nose, from fracture through the roof of the ethmoidal cells or cribriform plate, or in the nasopharynx. Escape of blood is not of so much significance as the presence of cerebrospinal fluid, indicating tearing of the dura.

If the cleft in the roof of the nose remains open, it may be necessary to refer the case to a neuro-surgeon, for closure with a graft of fascia lata or by some other method.

Pharynx

Wounds of the pharynx are rare, because of the fatal result produced in most cases by projectiles penetrating the neck.

There may be no marked symptoms following wounding of the pharynx by a piece of metal or other object, except for dysphagia, possibly of mild degree. I have removed from a bronchus a piece of high explosive shell which had passed through the side of the neck to enter the pharynx, whence it passed through the glottis; the patient was never seriously ill and suffered no apparent ill-effects.

If the patient survives perforation of the pharynx, he may be placed in a position of danger from infection of the para-pharyngeal space. Such an occurrence may cause death by spread to the mediastinum, by thrombophlebitis in veins of the neck, or by sloughing of one of the great vessels in the carotid sheath or of one of the branches.

It is imperative, therefore, to open widely any such infected wound, and sometimes it is advisable to tie the internal jugular vein.

Malar and Zygomatic Regions

Fractures in this region are constantly overlooked. The immediate swelling is liable to mask the deformity but the clinical picture is usually a very clear one and radiological examination confirms the diagnosis. When the fracture involves the roof of the antrum, as it commonly does, the effects on the eye are so serious that early treatment is of paramount importance. In uncomplicated cases replacement is readily achieved by passing a lever from an incision in the temporal region on the surface of the temporal muscle until it lies deep to the zygoma. Manipulation then disimpacts the fracture and brings out the malar eminence in a most convincing manner. Occasionally difficulty is encountered in maintaining the bone in position and recourse is had to packing of the antrum through a Caldwell-Luc opening or to external support as suggested by Mowlem by wire passing through the antral wall to a projection on a head cap.

In established deformity from this type of fracture much may be achieved by fat or cartilage graft. Diplopia is corrected by inserting cartilage or fat under the periosteum of the depressed orbital floor.

Nose Loss

When only the skin of the nose is lost it is often possible to replace it by means of a free full-thickness skin graft. In cases where there has been both skin and bone loss it is sometimes possible to elevate the scarred area by cartilage graft but it is usually wiser to excise the scar and bring down a forehead flap before introducing supporting material.

Most cases of loss of part or the whole of the nose can be converted into a standard type of defect to which a suitably shaped flap, rolled in distally to make the nostrils, can be applied. Such a flap is best obtained from the forehead, but excellent results can be obtained by means of a short abdominal tubed pedicle flap temporarily transplanted to the wrist. Alternatively the necessary flap may be obtained from the chest wall and swung upwards from its upper attachment near the point of the shoulder.

Here are certain simple principles for all reconstructive work:

(1) The three layer construction of the face—covering, supporting (skeletal) and lining—should always be borne in mind.

(2) Accurate diagnosis must be made particularly in regard to loss or displacement of tissue.

(3) Careful search should be made for foreign bodies: ingrained dirt, wood, glass and clothing are as important as metal.

(4) Routine excision of wound margins, desirable elsewhere, has no place in facial surgery.

(5) Drainage of a wound for twenty-four to forty-eight hours is infinitely less troublesome than the treatment of a hæmatoma or its usual sequel in this region—an abscess.

(6) Close and friendly collaboration between throat, plastic, ophthalmic and dental surgeons is essential to success.

J. F. Simpson said fractures of the nose often produced obstruction of the nasolachrymal duct and should be dealt with by the rhinologist and ophthalmic surgeon working together. In performing the Toti operation in such cases the duct was found torn or compressed by flakes of bone. He supported the advisability of making an intra-nasal opening in cases of fracture involving the antrum when these required surgical intervention.

The President said that he saw many of these injuries in their early stages during the last war. He was convinced that they should be treated within the first twelve hours, by restoring the conditions to normal as much as possible, not forgetting the skeletal layer underneath and also the mucous membrane. The maxilla and mandible should be restored as far as possible with the help of the dental surgeon.

In fractures of the nose, frequently one nasal bone was driven in and the other nasal bone driven outwards, and the replacement of the nasal bone driven outwards was forgotten. First of all the depressed nasal bone should be elevated, then the septum replaced, and finally the nasal bone which had been driven outwards should be pressed inwards. He agreed that a general anæsthetic should be used; these cases could not be manipulated effectively under local anæsthesia.

D. F. A. Neilson asked what was the best manner of dealing with an injury which involved the soft part of the nose. In one case he made a Stent splint for the patient to wear in the nose to try to establish the opening, but the patient took it out after two days and wore it at night for only about ten days.

What was to be done with fractures of the nasal bones where there was nothing more

flows through into the airway on deglutition and seriously endangers the life of the patient. A case under my care was operated on by the laryngo-fissure route, with the turning in of a skin flap; a successful result was obtained.

T. Pomfret Kilner: The plastic surgeon's contribution to any discussion of this kind must be of a pictorial character for his results are judged by external appearance. Nevertheless, it is essential in all reconstructive work that restoration of function should be constantly in the surgeon's mind.

The Fractured Nose

Very few nose and throat surgeons are sufficiently radical in dealing with simple fractures of the nasal bones and many come into the hands of the plastic surgeon later when treatment is a much more difficult problem. Free disimpaction and mobilization under general anaesthesia are essential. It is a useful check on the efficacy of this treatment to make sure that the nose can be made to deviate readily to the side opposite to the original displacement. I usually employ a splint of dental modelling-wax moulded over lead but this is retained only until risk of displacement during the stage of recovery from the anaesthetic is over. I have often used with satisfaction the collodion-gauze splint described by Mr. Negus.

In more extensive fractures in this region Mr. Negus's advice that treatment should be delayed for ten to fourteen days in order to avoid risk of meningeal infection must be accepted though it has always been my practice to try to get the bone fragments back into normal position as soon as possible, feeling that by doing so I was re-establishing normal drainage channels. In cases where the crookedness of the nose is confined chiefly to the cartilaginous parts submucous resection is usually required for restoration of free nasal breathing and frequently this, if sufficiently radical, is followed by depression of the bridge line requiring a hinged cartilage graft.

Where, in addition to depression of profile, there is excessive breadth of the bony bridge a two-stage procedure is required, narrowing of the nose by infraction being followed by the insertion of a cartilage or bone graft. In many severe injuries a crush fracture of the nose is associated with posterior displacement of adjacent parts of the maxillae. Where early treatment has failed to give correction of contour these cases should be treated as for syphilitic deformities. Building out the nasal bridge line alone is insufficient: restoration of the maxillary contour calls for freeing of the soft tissues from bone by skin graft introduced on Stent mould from an incision in the upper gingival sulcus. The mould is later replaced by a small vulcanite prosthesis or a projection from a denture.

Frontal Sinus Region

No restoration of contour in this region should be undertaken for at least six months after all discharge has ceased. Excision of a depressed scar can usually be carried out without reopening the sinus and after suitable undermining of the skin over the depressed area, a cartilage graft, suitably shaped to fill the defect, is inserted. In one of my cases the whole forehead region was sheared off by a propeller blade. There remained only scar tissue over the dura and both frontal sinuses lay open. After making sure that the sinuses opened freely into the nose, I closed them by folding over part of the mucosa and applied a large skin flap to cover the whole forehead area. At a later date contour was restored by strips of costal cartilage.

Maxillary Sinus Region

Many penetrating wounds in this region close spontaneously but usually with deeply depressed scars, and frequently with troublesome oedema of the lower eyelid. In many such cases it is possible to restore contour by excising the adherent scars and building out the depression by local flaps of deep tissue. Occasionally free fat grafts are needed while, where loss of tissue is extensive, both lining and covering skin flaps have to be provided.

In cases of fracture of the maxilla we use at Queen Mary's Hospital, Roehampton, a simple upper dental plate or metal cap splint with extra-oral projections (Kingsley) held up to a head cap or band. I aim at immediate rather than gradual replacement.

In the lantern slide records which I have shown you I have included a case of gross deformity following the removal of the maxilla for growth and would put forward a plea for the prevention of such deformity. Had the cheek cavity been filled out at the time of operation by a mould attached to a dental plate and carrying on its surface a Thiersch graft no contraction would have occurred and contour would have been readily restored by a simple dental appliance.

Section of Otology

President—F. W. WATKYN-THOMAS, F.R.C.S.

[February 6, 1942]

DISCUSSION ON SKIN DISEASES OF THE EAR

Dr. Reginald T. Brain: Infections of the external auditory meatus often produce secondary lesions in the path of the purulent discharge, and these lesions are unlikely to resolve until the otitis has cleared up. This is the task of the aural surgeon, and, because of anatomical factors, it is a difficult one, especially when œdema closes the meatal orifice.

The pus-coccal infections.—These are usually caused by streptococci and staphylococci, and clinically it is rarely possible to decide which organism is responsible. The pneumococcus may also give rise to similar lesions, and occasionally diphtheritic bacilli are recovered instead of the expected pus-cocci.

Impetigo contagiosa may affect the ear. The common scabbed type of sore is easily recognized but bullous lesions also occur in erythema multiforme and in those grave mysterious diseases of pemphigus and dermatitis herpetiformis. In hydroa æstivale, vesicular and bullous lesions erupt on the ears, face and hands during the summer, and unless it is noticed that the lesions affect all parts exposed to light, the ear eruption may be mistaken for impetigo. Circinate impetigo often resembles tinea, but its course is more rapid and its colour deeper red. Impetigo pityroides, the scaly variety, is similar to seborrhœic dermatitis. Ulcerative lesions are rare in impetigo, and cultures should be made to find the causative organism. The skin folds are commonly involved in impetigo, and retro-auricular intertrigo and septic fissures occurring under the lobule and in the fossa of the helix may be very resistant to treatment. From a moist lesion of impetigo, including intertrigo and fissures, a patchy pus-coccal dermatitis may spread to the face, neck or scalp. Treatment has a good deal to do with the cause and prevention of this variety of dermatitis. Follicular infection from staphylococcal impetigo may result in a boil, but it is surprising how often boils arise as primary lesions. In the Near East cutaneous leishmaniasis produces a similar lesion but chronic in its course. Certain individuals are very prone to staphylococcal infections, and the seborrhœic subject is a well-known dermatological type. Plump and placid or thin and nervous, the seborrhœic subject has a moist greasy skin, a liking for carbohydrate and fat, and a marked susceptibility of the skin and mucous membranes. Apparently the skin is attacked by the pityrosporon of Malassez, the benign bacillus abundantly found in scurf, for it is certain that many a scurfy scalp becomes inflamed and a scaly dermatitis spreads from it to the ears, face, and neck; also to the mid-line of the back and chest and to the folds and flexural surfaces. A retro-auricular intertrigo of seborrhœic origin is very common and is easy to recognize if the pale pink lesions covered by a flaky, soft, greasy scale are seen to be continuous with the scalp condition. Pus-coccal intertrigo has a deeper colour, and when it spreads into the scalp it does so asymmetrically, and the unaffected parts are not scurfy. The seborrhœic lesions invade the auricle and external meatus and, if they are a part of the condition described above, the diagnosis is not in doubt. This suggests that seborrhœic dermatitis is a specific infection, but unfortunately the pathogenicity of the pityrosporon has not been established, and the scaly lesions of impetigo suggest that mild coccal infections produce similar lesions, so that seborrhœic dermatitis is still a controversial subject for dermatologists. So is eczema which is obtusely confused with dermatitis.

Dermatitis is surely an inflammation of the skin, having a cause and classical signs and symptoms. If the cause is removed the inflammation should subside. Frequent examples are met during the treatment of coccal and seborrhœic lesions when some application irritates the skin. The affected area becomes erythematous and œdematous, and burns, and the exudate causing the œdema may raise vesicles or blebs or ooze through the horny layer and make a moist surface. If this dermatitis venenata is recognized early and the causative irritant removed, the inflamed skin will soon respond to cooling lotions.

Eczema is a reaction peculiar to a hypersensitive skin, and the hypersensitivity can be inherited or acquired. In varying degrees most irritants can sensitize the skin, and if the eczematous reaction occurs while the skin is inflamed the eruption appears on an area of dermatitis which is then described as eczematous. The first essential feature of eczema is that when the cutaneous hypersensitivity has been established the eruptions are often

than the evidence of the X-rays for the displacement or fracture? In the case of the boy who had been injured in a game at school it was always a very difficult point to know whether to interfere.

When he had been called in to deal with an infected antrum due to injury he had always found the cases apparently do better after operation than was customary with infections of the sinus due to some ordinary cause. There might be some more basic physiological disturbance in the case of an ordinary sinus infection than in that of a sinus infection from trauma.

J. H. Otty said that in early cases it had been found surprisingly simple to reduce these fractures of the malar-maxillary region through the Caldwell-Luc approach. Many of these cases had considerable comminution of the orbital floor and of the posterior wall of the antrum. The displacement of the posterior wall of the antrum caused the patient considerable difficulty in opening the mouth. The comminuted orbital floor was easily moulded into position and then held there with a firm pack of "bipped" gauze. The end of the gauze was pulled out through the naso-antral opening and removed after ten days or so when a certain amount of consolidation had taken place. He asked whether Mr. Kilner had any experience of the use of refrigerated cartilage grafts preserved in merthiolate.

W. G. Scott-Brown said that in the treatment of adhesions in the nose, he had found it advantageous in getting grafts to take on the septum after division of adhesions to use wire cages on which to build up the Stent instead of putting in the Stent moulds. He exhibited a wire frame which was more usually made up as a cage. The discharges would escape through the framework and the graft could be left in position without being disturbed for a long period.

V. E. Negus (in reply) said that the remarks which Mr. Kilner had made about the use of the obturator applied equally well to operations on the upper jaw for malignant disease. It was sometimes necessary to remove the greater part of the maxilla. If the obturator was put in about the fifth day the soft tissues adapted themselves to the obturator instead of the obturator adapting itself to the soft tissues.

In reply T. P. Kilner said that in cases of nasal obstruction due to scar tissue free excision followed by the insertion of a skin graft on a mould was necessary. He fully approved of the contrivance which had been exhibited for retaining such a mould.

When there was any doubt about the diagnosis of fracture of the nose following an accident, confirmation should be obtained either by X-ray examination or by waiting until the subsidence of swelling made the deformity obvious.

He had never used refrigerated cartilage but there was ample evidence that it fulfilled its purpose of contour restoration satisfactorily. He thought that anything other than an autograft of cartilage tended to produce more reaction and was more liable to be replaced by fibrous tissue in the course of time.

He did not consider the presence of blood in the antrum in cases of malar fracture an indication for opening the antrum but he recognized the importance of ensuring intranasal drainage in cases in which the antrum had to be packed to support the comminuted walls of that cavity or when closure of fistulous openings was being undertaken.

Warts and molluscum contagiosum may occur on the ear and their virus origin should be remembered because cure necessitates the destruction of every infected cell. The virus lesions of variola, varicella and vaccinia may be seen on the pinna and bromides and iodides produce good imitations of these lesions. The rare condition of painful nodular growth of the ear is of obscure ætiology but its pathology is that of focal inflammation with necrosis of cartilage and a foreign body reaction, the epidermis being thickened above it. This chronic painful nodule develops upon the upper part of the helix and often makes the pressure of a hat or pillow unbearable. It will respond to some method of cauterization but rarely to other treatment.

The keratoses are interesting dermatological lesions and some varieties remain as obscure to dermatologists as keratosis obturans does to the otologists. Hyperkeratosis occurs in certain congenital abnormalities such as ichthyosis. It may be a functional disorder and is quite common after any severe erythema, whether that is due to local irritation or to infection, toxins or drugs. It is characteristic of seborrhœic dermatitis, psoriasis, pityriasis rubra and follicular lesions such as keratosis follicularis and keratosis pilaris. The latter condition and possibly some of the diffuse keratoses may be manifestations of vitamin A deficiency.

Senile keratoses are not uncommon on the helix and may be recognized as horny wart-like papules. Removal of the horny cap reveals a bleeding shallow crater with the raised and irregular features of an epithelioma. The histology is suggestive of a pre-cancerous lesion in the early phase and eventually most senile keratoses develop into squamous-celled epitheliomata. Tar warts and actinic keratoses have a similar pathology and all these keratoses should be treated with radium or failing that, be destroyed by some form of cautery. In the congenital disease of xerodermia pigmentosa young subjects are very sensitive to sunlight and in consequence rapidly develop freckles, atrophic macules and actinic keratoses which become malignant. The natural prominence of the pinna exposes it to sunlight so that it is a common site for actinic lesions.

Rodent ulcers and the primary type of squamous-celled epitheliomata occur on the ear and, since invasion of the cartilage is almost inevitable, the prognosis is not so good as when the lesion is solely dermal, and disfigurement is common. Moreover cartilage reacts badly to irradiation, and a very painful necrosis with perforation sometimes follows.

Dermatological principles of treatment.—Treatment should be governed by two important facts. The first is that our protection from external irritants and infections depends upon a dry and intact horny layer, and treatment should be designed to maintain it. Pyogenic lesions are often spread by wet dressings or by ointments which retain sweat and exudates. The second fact of therapeutic importance is that living cells are instantly damaged by water or by grossly hypotonic solutions, and this applies to the skin when the protective stratum corneum is missing or its continuity is disrupted by exudation. Thus the washing of a weeping eczematous area with plain water is usually quickly followed by an increase of the irritation, erythema and exudation, and in consequence resort is made to the inferior procedure of cleansing with oil instead of trying isotonic solutions.

Before considering local applications in detail, thought should be given to the general condition of the patient and his or her reaction to the skin lesion. Eczema and lichen simplex are often manifestations of a neurosis and many a pus-coccal infection fails to clear up with proper treatment because the patient scratches. Reassurance and small doses of phenobarbitone make the difference between success and failure in such a case. Although it is obviously sensible to consider the patient's diet and to advise the use of protective foods, dermatologists have found few indications for rigid dieting. It would also appear that too much has been expected of the vitamins. Vitamin A has been used hopefully in the keratoses; vitamin B₁ for neurogenic and herpetiform lesions; vitamin C in the infections and to protect the capillaries; vitamin D for its effect upon calcium metabolism and so diminishing the irritability of, and the exudation from, capillary vessels; and nicotinic acid for its influence in desensitizing the skin to sunlight, and possibly as a factor in the resistance of nerve tissues to herpetic infections. Sulphonamide is very valuable when a pus-coccal lesion of the skin shows its virulence by a bright erythematous flare.

In the chronic staphylococcal infections small doses of thyroid and injections of colossal manganese or of manganese butyrate appear to be beneficial. Toxoid and vaccine constitute more rational therapy but often fail because the general immunity they undoubtedly confer does not influence the carrier state nor the susceptibility of the skin to the staphylococcus.

Therapeutic agents in dermatology.—Of these, lotions are the most useful. Calamine lotion is well tolerated even by the eczematous skin and the addition of 2% phenol or 0.1% mercury perchloride gives it antiseptic qualities. To allay irritation 2% of tar solution

independent of external factors, or at least the irritating properties of those factors are imperceptible to the normal skin. The second essential feature is the morphology of the lesions. Eczema appears as grouped, minute papulo-vesicles set in non-inflamed skin. The vesicular character, microscopically constant, becomes obvious when slight scratching reveals a weeping spot. Itching is marked in the eruptive phase but subsides when weeping begins. The exudate dries into golden crusts or maintains a moist surface upon which the skin cocci flourish and produce a dermatitis sometimes obscuring the eczema. Since eczema is the reaction of a hypersensitive skin the appearance of the eruption is the call to suspend the use of active remedies and to apply bland lotions and emollients. General treatment with sedatives to allay irritation and to restrain scratching is important, and calcium and vitamins help to reduce the reactivity and permeability of the skin capillaries. Ultra-violet light has a good effect upon the skin, upon metabolism and upon the mental outlook of the patient.

The chronic infections of tuberculosis, syphilis, leprosy, yaws and leishmaniasis may attack the ear, and the granulomatous eruptions due to bromide or iodide are somewhat similar in appearance to the lesions of these infections.

Lupus vulgaris more commonly affects the face than the ear, and can be recognized by its brownish-red translucent nodules which have a close resemblance to apple jelly when compressed under glass. This may be difficult to observe in the lobule, which often becomes reddened with congestion. Lupus vulgaris is an active tuberculosis of the skin leading to ulceration, and the lobule and a considerable part of the pinna may be destroyed. It also occurs in the purple congested lobules of patients with chilblains, and is then called lupus pernio. The sarcoid of Boeck shows similar lesions, and nodular infiltrations of the skin also occur in leukaemia and in Hodgkin's disease.

Lupus erythematosus is quite distinct from lupus vulgaris but its final atrophic phase results in just as much destruction of the pinna but without ulceration. The erythematous lesions are superficial and scaly and horny plugs mark the orifices of the sebaceous follicles. It is thought that lupus erythematosus is an allergic reaction to streptococcal or tuberculous infection and certainly the most dramatic cures result from the extirpation of a septic focus, usually found in the nose or throat. When a focus cannot be found, treatment with sulphanilamide or sulphapyridine gives a fair measure of success and some support to the view that the streptococcus is of aetiological importance.

Mycotic infections of the ear are rare and it is not proposed to discuss them. When they do occur perhaps the experience of the dermatologist with the common fungus diseases would be of value to the otologist.

The virus lesions of the skin are also rare, but they are not without interest. Herpes simplex and zoster are often confused and indeed if the eruptions are scanty it may be impossible to distinguish between them. Both have an eruption of grouped vesicles on an inflamed base and are unilateral. Zoster is the more painful and its eruption is often spread over the distribution of the cutaneous sensory nerves belonging to the ganglion attacked. The pinna is mainly involved with the adjacent parts of the face and neck when zoster attacks the second and third cervical ganglia and the extent of the eruption makes the diagnosis clear. Of greater interest and difficulty is zoster of the geniculate ganglion, for the sensory supply of the 7th nerve is somewhat rudimentary and overlaps that of adjacent nerves to the tympanic membrane, the external auditory meatus and the adjacent parts of the concha, so that a few vesicles in these inconspicuous sites or on the anterior part of the tongue may be the only direct evidence of zoster infection as the cause of facial palsy. In 1907 Ramsay Hunt [1] drew attention to these facts in his classical paper on "Herpetic Inflammations of the Geniculate Ganglion" and in 1933 Aitken and I [2] were able to confirm Ramsay Hunt's thesis by using a method of complement fixation for zoster which I had found to be reliable [3]. The sera of 9 cases of Bell's palsy with zoster-like eruptions in the areas of the sensory supply of the geniculate ganglion were all found to contain antibodies to zoster virus while the sera of 22 cases of Bell's palsy without such eruptions had demonstrable antibodies to zoster in only four instances, or in less than 20%. Since evidence exists to support the view that symptomatic zoster, and herpes, are the result of the activation of latent virus by infection, trauma or shock, it is possible that the unexpected post-operative Bell's palsy is a zoster lesion even in the absence of a vesicular eruption. The possibility would be a probability if, after a few weeks, the presence of zoster antibodies could be demonstrated in the patient's serum. With good antigens the complement-fixation test for zoster is as reliable as the Wassermann reaction, but the test is rarely used outside a virus research laboratory.

Zoster should not be confused with herpes simplex. Zoster virus is probably a modified strain of varicella virus. Immunity after infection is usually solid and lasting, so that zoster rarely recurs. It is non-infective for the ordinary laboratory animals. On the other hand herpes is not related to zoster. Recurrent attacks are common and the rabbit and guinea-pig are readily infected.

that a drying source of heat is most comfortable and healing in the treatment of boils, and use should be made of infra-red rays, diathermy or X-rays. The last-mentioned in fractional doses is of the greatest service in many chronic skin diseases of the ear and it is unnecessary and less effective to attempt to restrict the irradiation to the meatal walls. Radium and radon are invaluable agents in the treatment of the senile and actinic keratoses and of the epitheliomata.

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The President asked, with regard to allergic reactions, could a patient become sensitized to preparations commonly employed? He had noticed that a patient would do well for a time on one particular preparation and then suddenly get worse. Was that true of mercury and sulphur?

The abuse of peroxide was responsible for many of their troubles. Patients with discharge were too often sent away, week after week, with peroxide drops. Could peroxide set up a chronic dermatitis?

As to zoster lesions, the complement-fixation test should establish the diagnosis in doubtful cases of Bell's palsy between the true palsy *a frigore* and a missed zoster.

Lastly, in keratosis obturans. Was this a true keratosis or was it a cholesteatoma which had burst its way through and so dilated the cavity?

Mr. R. J. Cann said that he had occasionally met a condition which he had not seen described. The patient complained of a chronic discharge, with possibly a history of bleeding, and after cleaning out the meatus there was seen on the deepest part of the walls of the meatus or even on the membrane, patches of granulation which bled when touched. Apart from the granulations the membrane was seen to be intact. One or two applications of a caustic to each of the granulations would clear the condition up in a week or a fortnight. He did not know the pathology.

Dr. Hugo Frey said that he was glad to hear Dr. Brain attribute importance to the mental condition of the patient, because he had also come to the conclusion that in a number of cases eczema or dermatitis was only secondary to itching sensations of nervous or psychic origin. Itching in the meatus was very common. It was known that the Chinese were accustomed to carry about with them a small instrument for the purpose of scratching their ears.

Another reason for the eczema was possibly an over-indulgence in personal cleanliness. There was a general idea that the meatus must be most thoroughly washed, and he had seen even nurses and mothers continually trying to cleanse babies' ears, and this was frequently the cause of eczema. The complete removal of cerumen might make the skin of the meatus too dry, and so produce the itching. Many ointments and oily compositions were dangerous and likely to make an existing eczema even worse.

For local treatment in acute cases resorcin solution 1%—2% seemed to be very efficient.

Mr. F. C. Ormerod said that amongst civilians the most frequent type of external ear disease was an eczematous condition associated with seborrhoeic dermatitis.

There were, after the last war, many soldiers and other service men who required treatment for a chronic thickening of the skin of the meatus. The meatus was found to be almost impervious to anything but a small probe. It was assumed that most of these men had chronic middle-ear suppuration, although in many cases the drum head was never seen.

During the last two years he had seen many young soldiers with a similar condition in a much earlier and more acute state. He did not think the condition was one of seborrhoeic dermatitis, but most of them had infection of the tonsils. They often required several weeks of treatment. He felt that there must be many more people in the Forces with conditions of this kind, and that there was great danger of chronic external otitis resulting under Army conditions. It would be interesting to learn whether the members in the medical services were having much trouble from this external otitis.

Lieut.-Colonel M. L. Formby said these cases caused much trouble in the Army. Difficulties had arisen firstly because from 1937 onwards men were accepted into the Army with discharging ears, and secondly when civilian medical boards were introduced in 1939 no instructions were issued regarding otitis externa. In future chronic cases would not be admitted. The present problem was the large number already in, many of whom had now become extremely useful soldiers. If possible these men must be retained. Many lost a great deal of time attending sick parades and having treatment, others spent

or of strong solution of lead subacetate may be added and for seborrhœic dermatitis 2% of precipitated sulphur. A lotion with a powder basis adds to the debris of the meatus and this can be avoided by using simple solutions. Gentian violet, methyl violet, brilliant green or malachite green can be used in 1 or 2% solution in water. Acriflavine 0.1% in water has the advantage of not obscuring the skin lesion, and for exudative conditions 4% of tannic acid may be added. This lotion is useful for moist pus-coccal lesions and for eczema. The metallic salts are used when an astringent effect is indicated; they are antiseptics too but of a low order in the presence of exudate; they have little power of penetration unless ionized and idiosyncrasy towards them is not uncommon. Examples are: mercury perchloride 0.1%; l'eau d'Alibour which consists of zinc sulphate 6 gr., copper sulphate 4 gr., and camphor water to one ounce; silver nitrate 1.5% in distilled water is also useful.

When a quickly drying application is indicated a spirituous paint should be used. For example, the aniline dyes already mentioned can be dissolved in 50% industrial methylated spirit or 10 gr. of silver nitrate can be dissolved in 60 minims of distilled water and made up to one ounce with spirit of nitrous ether. Eczematous lesions are very intolerant of spirit which is painful unless the horny layer is intact but spirit paints penetrate the follicles better than watery lotions and are useful to localize boils.

A 5% sulphanilamide solution in glycerin is a useful paint for crusted pus-coccal lesions and as a dressing for septic ulcers. Powders are used to dry up exudates and to limit infections of the skin, and powdered sulphanilamide is certain to prove of value in the treatment of meatal lesions. Bismuth subgallate is another useful astringent powder and 5% of calomel may be added to it as an antiseptic or added to a zinc oxide and talc basis.

When lotions are not tolerated or when the skin becomes too dry and tends to crack, creams or pastes are indicated.

The simplest cream consists of equal parts of a vegetable oil and lime water with perhaps 5-12% of hydrous lanolin to make it more stable. 5 or 10% of zinc oxide or calamine added to this emulsion makes a zinc or calamine cream which may be medicated with 2% each of phenol, tar solution, ichthyol or sulphur; the latter being of service in seborrhœic dermatitis. A paste should contain equal parts of powder and grease; for example zinc paste consists of starch 25%, zinc oxide 25% and vaseline 50% and to this may be added 2% of salicylic acid, or 3% of coal tar or of yellow oxide of mercury and similar percentages of ichthammol, resorcin or sulphur are used for seborrhœic lesions. Pastes with their high content of powder can absorb water and lose it, so that the skin surface is drier and cooler than when it is covered by ointment. Besides this therapeutic gain the saving of 50% of the fatty base is a valuable economy in war.

Ointments are made with bases of vegetable and animal fats with the addition of hard and soft paraffin or of the latter alone as a base. Recent advances have been made with new emulsifying bases which are less greasy and bring the active ingredients into more intimate contact with the tissues. These new bases are readily miscible with water and so are much more easily cleansed from the skin. Comment has been made upon the limited use of ointments in dermatology, and no reference need be made to those in common use. Ointments containing sulphanilamide are certainly potent on streptococcal lesions of the skin and quinolor ointment is often very effective on staphylococcal lesions. So little of these ointments is required for an individual patient that there can be no objection to using a proprietary preparation.

Since seborrhœic lesions of the ear usually resist treatment or soon recur unless the scurf condition of the scalp is cleared up, here are a few hints on the treatment of the scalp. For a dry scurf scalp an ointment of 3% precipitated sulphur and salicylic acid should be rubbed in at night and shampooed out after a day or two with sulphur soap and weak sodium carbonate solution. Instead of the ointment a liniment of salicylic acid 25 gr., industrial methylated spirit 1 drachm and castor oil to one ounce may be preferred. 5% of phenol or of liquor picis carbonis may be added to the ointment or to the liniment to allay irritation. When the scurf has been cleared relapse may be prevented by the continued use of a lotion such as: resorcin 10 gr., industrial methylated spirit 2 drachms and liquor hydrarg. perchlor. to one ounce. If this proves to be too drying 20 minims of castor oil or of glycerin should be added to each ounce. Weekly shampoos are indicated in subjects prone to pityriasis capitis or seborrhœa.

Physiotherapy.—Orologists are familiar with methods of ionization and are no doubt aware that although the penetration of ions is slight the surface effect of the solutions employed is greatly intensified. In consequence eczematous lesions are usually aggravated by ionization. Although lesions of the meatus can be treated with local ultra-violet light by quartz applicators and a Kromayer lamp the greater beneficial effect of general ultra-violet ray therapy in chronic infections is often unsought. It is not generally realized

Section of Neurology

President—GEORGE RIDDOCH, M.D.

[February 19, 1942]

DISCUSSION ON CEREBRAL ŒDEMA

Dr. J. G. Greenfield : *Criteria of Cerebral Œdema.*

Three criteria have been suggested so far. These are (a) physical, (b) chemical, (c) histological.

(a) The *physical criterion* rests on the relationship between brain size and skull capacity. Following Rieger and Reichardt, Alexander and Looney define cerebral œdema as an increase of brain size beyond 96% of skull capacity. This is satisfactory except for the difficulties of the technique of measurement and the possibility of vascular dilatation. The normal variations associated with age also introduce an error of unknown dimensions. In childhood, as is well known, the brain fills the skull almost completely; in old age the vertex is separated from the bone by a varying depth of subarachnoid space; and the disproportion between skull capacity and brain size varies with different people at the different age-periods. Le Beau (1938) has simplified this definition by using tentorial herniation as the criterion of cerebral œdema.

(b) The *chemical criteria* depend on the ratio of wet to dry weight of the brain substance. Much work which has been done on this question has not taken into account the normal difference between grey and white matter in this respect although this was established by Halliburton in 1894. Normally about 70% of white matter and 83% of grey matter consists of water and as œdema is rarely sufficiently intense to add more than 10% of water to the brain tissue, it is necessary to examine grey and white matter separately in making this analysis. Stewart-Wallace (1939) added the valuable criteria of the contents of sodium and chlorine ions, since the fluid added to the swollen brain tissue brings with it sodium chloride in a concentration at least as high as in blood plasma. Owing to the virtual absence of chlorine from the nervous tissues the percentage increase of chlorine ions is especially great, rising to 500% or even 600% of the normal. That of sodium may rise to 200% and rarely to 300%. These figures should be corrected for added blood by the determination of excess of iron, as has been done by Shapiro and Jackson (1939), and this is especially important in examining the grey matter. Even with this correction the figures do not reveal whether water and sodium chloride are added to normal brain tissue or merely fill the spaces left by atrophy of cerebral tissue, as Alexander and Looney (1938) have shown that they do in senile and shrunken brains. The method is, however, of value in cerebral tumours in which œdema is usually confined to the homolateral side.

(c) *Histological criteria* of œdema have been denied by many, though certain histological changes have long been known to occur in the more severe forms. The false distinction between cerebral œdema and simple swelling of the brain has for long confused thought and delayed progress in this direction. At the present time, we can define the histological characteristics of cerebral œdema in the white matter only, and again only in the forms related to cerebral tumours, granulomas, and abscesses. In such lesions we can form some opinion also of the severity and duration of the œdema on histological grounds alone, but we may be unable to tell whether an old-standing lesion has resulted from œdema or from some other cause.

If it were possible in any given case to apply all these criteria the diagnosis of cerebral œdema would be certain. Actually with any two of them in agreement the diagnosis is reasonably definite. But many conditions have been described as œdema in which there is no further evidence than an increase in size of the brain in relation to the capacity of the skull, that is to say flattening of convolutions or tentorial herniation. Many of these are probably susceptible of a different explanation.

Conditions Associated with Cerebral Œdema

(1) Of the known causes of cerebral œdema probably the commonest is *thrombosis of cerebral vessels*. It has long been recognized that brain tissue swells when its arterial

a considerable time in hospital. The aim now was to investigate the cases in the Army, co-operating with dermatologists, in an endeavour to ascertain their nature, whether seborrhœa, eczema, &c., and to treat on appropriate lines. From now on instructions to civilian medical boards would be that these cases should be sent for an otologist's opinion before they were accepted.

Men in the Army with chronic otitis externa for which no definite cause could be found and in whom treatment was unsuccessful would be boarded out unless they were extremely useful persons whom it was desirable to retain.

One Army otologist had taken a great interest in otitis externa and had been struck by its association with dental trouble, particularly impacted wisdom teeth. He had been encouraged to collect these cases and publish findings.

Dr. Agnes Savill mentioned the case of a man who had suffered for four years with a condition of infective eczematoid dermatitis of the ears, with blocked auditory canal and much œdematous swelling of the whole ear. Ionization given deep down into the canal with zinc, and then salicylic acid, had effected a cure in about six weeks.

A simple remedy for dandruff of the scalp consisted of half to one dram of liq. carbonis detergens to the ounce of water.

Lieut.-Colonel Mitchell-Heggs supported Dr. Brain and others in their views on the importance of the treatment of septic foci in certain cases and the value of post-operative rest and adequate diet, especially for those patients who relapsed after apparent recovery following an effective local application such as that of a weak silver nitrate lotion. He mentioned the importance of bacteriological examination and recalled a case of *B. pyocyaneus* infection of the meatus successfully treated by Major Philip Scott. He also recalled a case of recurrent pruritus and dermatitis which responded well to general treatment for gout, and stressed the importance of keeping hair away from the pinna and avoiding hats which compress the ears in women suffering from active or convalescent dermatitis of the pinna with a sensitive ticklish skin.

Mr. W. Stirk Adams said that he also had seen the granulations on the drumhead associated with a discharge; he had regarded it as a secondary condition, but he agreed with Mr. Cann. As to chronic otitis externa affecting the deep meatus, it was a difficult condition, and the most successful treatment was 5% silver nitrate in aqueous solution. The patient was also asked to use 2% nitrate of silver drops daily for about a fortnight, and after that to keep it by for use at longer intervals. Generally the condition cleared up after two or three weeks' treatment.

Mr. R. D. Owen asked Dr. Brain for his opinion as to the use of the "wet" or the "dry" treatment in patients with chronic eczematous meatitis; in other words the use of drops as compared with powders.

Dr. Reginald T. Brain in reply said that the skin could be sensitized by simple chemical substances but this was not common with the preparations usually employed. Mercury and sulphur were irritants and dermatitis was often seen after their use. He had never seen a case of dermatitis which could be attributed to hydrogen peroxide but cases had been reported by Sabouraud. Probably the continual wetting was the main factor but the protective horny layer would undoubtedly suffer mechanically and by oxidation from peroxide.

He was in entire agreement with Dr. Frey's remarks and several types of prurigo were recognized by dermatologists in which the skin lesions, usually eczema or lichen simplex, were the result of scratching. A common site for this neurogenic lichen was just behind one ear.

With regard to the "wet" or "dry" treatment of chronic eczematous meatitis the continued and casual use of ear-drops by a patient was bad for the skin. An œdematous skin was usually aggravated by moisture and in dermatological practice drying lotions and paints would be used. Since the meatus was badly ventilated it must be carefully dried by expert swabbing or by a warm jet of air. Powders might be useful in the terminal phase but they formed concretions with exudate; an obvious disadvantage. In conclusion Dr. Brain paid tribute to his teacher, Dr. J. H. Sequeira and to Professor S. P. Bedson for help in researches upon virus diseases of the skin.

contusion has been the treatment of the œdema which is supposed to follow the trauma. But the experimental and post-mortem investigations which have been made in the United States are definitely against this assumption. Probably it was Apfelbach's work (1922) that formed the basis for the American belief in traumatic œdema. He determined the ratio between wet and dry weight in brains of patients dying after cerebral trauma and found an increase of water in some cases up to 3.7%, although in many cases there was no increase at all. He concluded that œdema frequently occurs when the cranial bones are fractured and that it is most common in patients dying more than a few hours and less than three days after the accident, rarely earlier or later. But as he used an undetermined mixture of grey and white matter his results are worthless.

Nevertheless much clinical treatment continues to be based on the assumption that traumatic cerebral œdema is a major danger to life. Pathological investigations on the other hand have provided very little basis for this assumption. For example, Vance (1927) reviewed 512 cases of death from head injury. He found that during the first few hours there was an increase of intracranial pressure, with flattened convolutions. At this stage the blood-vessels were engorged. Later, twelve to twenty-four hours after the injury, there was distinct swelling of the brain and the brain tissue was moist, but he says: "No case presented the degree of swelling that Apfelbach mentioned and no example of death arising solely from this condition was found." Connor and Wright (1934) reviewed 1,760 clinical cases of cranial and intracerebral injuries with many autopsies. They could not find a single instance of generalized traumatic œdema coming on forty-eight hours after the injury. They say: "We are also somewhat sceptical of its late occurrence as we have never seen it." Shapiro and Jackson (1939) reported some investigations on cerebral œdema. Among other things they investigated the relationship of wet to dry weight, in grey and white matter separately, correcting for blood by estimating the iron content. Contrary to Apfelbach they found that injured brains contained less water but more blood than normal brains. They thus could find no evidence for true cerebral œdema after trauma.

Valuable experimental work on this subject was published by Pilcher (1937), who performed 60 experiments on dogs, allowing a weight of a half or one kilogramme to fall on the heads from a height of 5 feet. In no case did he produce either fracture of the skull or intracranial hæmorrhage, and only one dog died as a result of the trauma. Examination of the intracranial pressure showed a sharp initial rise after the trauma followed by a return to normal within thirty minutes. A subsequent more sustained rise of pressure began in some of the dogs one to two hours after the trauma. Examination of the brains at various intervals after the injury showed a slight increase in water in the grey matter, and an increase of not more than 2% of water in the white matter. These ratios were not corrected for added blood. A matter of some clinical interest in these experiments is that in dogs treated by intravenous injections of hypertonic dextrose the increase in the ratio of wet to dry weight was at least as great as in untreated dogs. He concluded that there was, after trauma, an increase of fluid in the grey matter, but he did not find any evidence that marked cerebral œdema occurred. As he did not correct his figures for added blood, by estimation of the iron, the increase of fluid in the cortex might well have been due to vascular congestion.

My own investigations in the matter have been histological. In the 31 traumatized brains which I have so far examined since the outbreak of war I have found no evidence of generalized post-traumatic œdema. Where the brain is bruised, especially after fracture of the skull, or where it is penetrated by a missile there is often a zone in which the myelin stains poorly, and the astrocytes are swollen. The ill-defined margin of this area and its demarcation by the subcortical arcuate fibres resemble what is seen in œdema round tumours. This zone may be a few centimetres in width, but rarely more than 2 cm. unless there is gross displacement of the bones of the skull with overriding of one fragment by another.

In three cases a fairly large artery had become thrombosed and this had produced an area of degeneration of myelin, more intense in character and with much sharper edges than that due to œdema of the brain. This would have been associated with temporary swelling of the tissues in the affected area.

These investigations lead me to conclude that œdema following injury to the brain is limited to the neighbourhood of the bruised area. Its extent appears to be related to the amount of deformity of the skull caused by the accident, and it is only great when fairly large fragments of skull are driven inwards and when there is overriding of the edges of the fracture. Even in such cases it usually only affects one pole. Naturally sepsis or abscess will increase its extent and severity. But apart from these complications it is usually very slight and is limited to the white matter underlying the bruised area of cortex.

blood supply is cut off or its venous drainage blocked. The cause of this phenomenon is not fully understood. In the grey matter the swelling is probably chiefly due to congestion of capillaries, at least in the early stages, during which this tissue looks redder than normal. But white matter which is swollen as the result of ischæmia does not differ appreciably in colour from the normal, and blood-vessels form so small a part of its bulk that any distension of these, without hæmorrhage, could not cause the great swelling which occurs. The swelling appears to be due to chemical changes in the myelin and axons and to be allied to the post-mortem swelling of these tissues. It seems to be a true œdema in the sense that the water content of the white matter increases. The histological changes in this form of œdema in its milder grades are similar to those in the severer grades of œdema surrounding cerebral tumours. The importance of cerebral ischæmia as a cause of œdema is twofold. It explains swelling of the brain in some cases of hypertensive encephalopathy, and in some cases of cerebral contusion in which thrombosis of cerebral vessels occurs. And it offers a possible theory for the œdema which occurs in relation to cerebral tumours.

(2) *Cerebral tumours, granulomas and abscesses.*—Much work has lately been done on œdema in relation to cerebral tumours. Here the first criterion of brain swelling (i.e. relationship of brain size to skull capacity) cannot be applied. But the fact that in most cases œdema is limited to, or is greater in, one hemisphere, makes it possible to apply the second and third criteria with the normal hemisphere as a control. As regards histological criteria, Spielmeyer, Spatz (1929) and others in Germany described swelling and degenerative changes in the astrocytes (clasmato-dendrosis and amœboid glia) in the œdematous area round cerebral tumours. But they regarded these as chance accompaniments and not, as I have lately shown, as one of the most constant and characteristic changes. They paid little attention to the alterations in the nerve fibres and myelin sheaths, regarding these as simply separated from one another by interstitial fluid. Jaburek (1936) made a very important contribution to this subject. He found that œdema associated with cerebral tumours affects the white matter exclusively, usually only on the side of the tumour, and that it spares the large commissural tracts such as the corpus callosum, optic radiations and anterior commissure. He considered that the brain-stem was also spared but of this I have no confirmation. It certainly is not affected in tumours of the hemispheres. He also noticed that œdema is much more common in rapidly growing, poorly differentiated tumours, than in slow-growing gliomas, endotheliomas and angiomas. He could find no sharp distinction between cerebral œdema and brain swelling, which he considered to be merely two stages of the same process. Scheinker (1938, 1941) noted swelling of myelin and of axis cylinders as well as swelling of astrocytes in œdematous areas. He, however, still adheres to the distinction between œdema and swelling of the brain. I was able to confirm the observations of Jaburek and most of those of Scheinker in 1939. I did not, however, find in any of my cases the dilatation of perivascular spaces described by these authors. This may have been due to differences in technique, but it is remarkable that even when the myelinated fibres are widely separated by fluid the perivascular spaces appear closed. Swelling and pallor of the myelin sheaths were constant and irregular swellings on the axis cylinders were common in my cases. Every astrocyte in an œdematous area shows swelling of the cell body, slight at first, and irregular in the more acute cases, whereas large fibre-forming astrocytes are seen in the more chronic cases. Mitoses in astrocytes is not a very uncommon finding in fairly acute cases, and many of the cells are binucleated. In contrast to the astrocytes the oligodendroglia showed little change, and the mobilization of the microglia appeared to depend on the changes in the myelin. As regards the distribution of the œdema, it always has an indefinite border, merging gradually with the neighbouring normal tissue. The escape of the subcortical arcuate fibres and of the corpus callosum and optic radiations was very striking in many of my cases. In some cases changes similar to those of ischæmia were found in the overlying cortex, but these were never very severe, and the cortex for the most part appeared normal. If therefore œdema in cases of cerebral tumour is due to ischæmia, the rich capillary network of the cortex must be able to supply it with oxygen.

In cases of abscess, tuberculoma and gumma of the brain, œdema is often very severe. To some extent it has the same histological appearances as the œdema surrounding cerebral tumours, but its inflammatory nature is shown by the escape of leucocytes from the vessels, and the more severe degenerative changes in the nervous tissues. Such changes are still more marked in diffuse septic encephalitis. This is a true inflammatory œdema.

Trauma of the Brain

In trauma of the brain œdema has been regarded as an important complication, so important in fact that for some years in many American clinics the treatment of cerebral

is associated with gross swelling of all parts of that half of the cerebrum. An abscess of the frontal lobe may clinically cause hemiplegia which quickly clears up after the abscess is aspirated, and a comparable improvement in local signs is sometimes observed after a brain tumour is decompressed. These well-known clinical observations suggest that the improvement observed depends on the relief of circulatory embarrassment of which œdema is the most visible expression.

The effect produced by obstructing a cerebral vein may also throw light on the subject. Symonds (1940) has described the transient but widespread loss of function which follows thrombosis of a cerebral vein, while Mr. Pennybacker tells me that if a cerebral vein from the frontal lobe to the superior sagittal sinus is ligatured at operation there may be widespread loss of function of one cerebral hemisphere which temporarily causes hemiplegia, but from which there is a good recovery.

A recent operation provides a further example of the effects of obstructing a cerebral vein. An aircraftsman developed signs of increased intracranial pressure two months after being momentarily knocked out at boxing. At operation a large subdural hæmatoma was evacuated from the left side. The right hemisphere was then explored through a burr-hole high up in the right posterior parietal region: a large vein which presented beneath the dura looked so like the wall of a blood cyst that it was deliberately opened. The mistake gave then appreciated, hæmorrhage was controlled with a muscle graft, but some swelling of the underlying brain was noted. On the day following operation in addition to there being complete astereognosis and some paresis of the left hand there was complete left homonymous hemianopia. The visual fields recovered in two or three days while stereognostic sense recovered almost completely in about three weeks.

In head injuries, areas of contusion or hæmorrhage are also surrounded by an area of œdema which may contribute to the transient hemiplegia frequently observed after local cerebral contusion.

All these examples of local circulatory disturbance have this in common, that they are associated with a local disturbance of cerebral function which is under certain circumstances only temporary. It is impossible, however, to conclude that the œdema in itself is the cause of the loss of function as other associated circulatory abnormalities may in fact be more important.

Recent experiments on cats by Denny-Brown and Russell (1941) on what we termed acceleration concussion contributed little to this problem. We found that in the absence of gross intracranial hæmorrhage there was no important change in intracranial pressure or other evidence of œdema after severe concussion.

It is an interesting practical question whether local œdema surrounding a tumour or abscess is readily influenced by intravenous hypertonic solutions or whether the brain shrinkage observed in these circumstances is due more to a reduction in volume of the relatively normal brain tissue. If the œdema in these cases is due simply to venous obstruction it is difficult to see how an intravenous hypertonic solution can readily influence the œdematous area. It is possible therefore that these dehydrating measures affect the relatively normal brain to a greater degree than the abnormal œdematous brain. This hypothesis would give some explanation for the disappointing results and the delayed relapses which may follow this form of treatment.

We have attempted to investigate this matter by observing the effects of injecting 50 c.c. of 50% sucrose intravenously following air encephalography or ventriculography in cases of ventricular displacement due to cerebral tumour. Radiological examination of the air-filled ventricles was repeated at intervals of up to half an hour following the intravenous infusion. No significant change in the position of the ventricular system was, however, demonstrated, but we have no evidence that the few cases investigated were in fact complicated by cerebral œdema of marked degree.

In conclusion, therefore, while it seems evident that local cerebral œdema is associated in pathological conditions with loss of cerebral function of the œdematous area, it does not seem possible with our present knowledge to decide whether this local abnormality is in fact due to the œdema or is due to other circulatory disorders with which the primary condition is associated. There is a danger that the diagnosis of cerebral œdema is used as a cloak to conceal our ignorance of the cause of certain cerebral states.

I am indebted to Lieut.-Colonel G. O. Chambers, M.C., R.A.M.C., for permission to refer to cases in the hospital under his command, and to my colleagues for assistance.

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Other causes.—As regards other supposed causes of cerebral œdema we are on very uncertain ground and I shall therefore refer to them briefly. *Uræmia* is often said to cause œdema of the brain, but I have no first-hand evidence for this. In *hypertensive* states the well-known encephalopathy is probably due primarily to foci of ischæmia and any swelling that may follow is of the nature of ischæmic cerebral softening. This interpretation at least appears to be warranted by the histological changes which I have found in such cases. Alexander and Looney's cases of uræmia and hypertension gave inconclusive evidence. They had three cases with this diagnosis in which there was an increase in the ratio of brain size to skull capacity. One of these had a subarachnoid hæmorrhage and one a hæmorrhagic softening in one occipital lobe. The latter was the only case in their series in which there was œdema of the white matter, as judged by the ratio of wet to dry weight of the tissue. Another case, that of a woman of 44, showed only 1.15% difference between skull capacity and brain weight, but the ratio of wet to dry weight of the brain tissue was reduced rather than increased.

Cerebral œdema has also been described in catatonia, and narcotic poisoning (Struwe, 1931), but more evidence is needed on these conditions. One condition, however, deserves mention, i.e. the swelling of the white matter of the brain which may occur in *hydrocephalus*, especially when it comes on rapidly. This is not a true œdema, but rather a passage of cerebrospinal fluid from the ventricles into the brain tissue through the stretched ependyma. The formation of diverticula of the ventricles into the surrounding white matter which has been described by Northfield and Russell (1939) appears to be a later result of this process. This condition has little clinical significance, as there is no increase of the contents of the skull except that of cerebrospinal fluid.

In closing I would urge the need of clarity in defining cerebral œdema. Neither congestion of the cortex, nor wetness of the surface of the brain need have anything to do with œdema. The œdematous brain, when cut, may appear wet, but does not always do so, and post-mortem changes may simulate œdema macroscopically. In fact the only certain criterion of œdema, on examination of the fresh brain post mortem, is diffuse yellowish discoloration of the white matter and this is seen only in the severer grades of œdema.

Since cerebral thrombosis ranks high among the causes of œdema, we should bear it in mind where there is rapid increase of intracranial pressure in such conditions as uræmia, cerebral contusion, and after operation for cerebral tumours.

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Major W. Ritchie Russell: In attempting to determine the clinical results of cerebral œdema attention may be directed first to diseases which cause a general cerebral œdema and secondly to those conditions which cause local swelling of part of the brain.

In cases of uræmia for example there is some general œdema but the relationship of this to the comatose condition of the patient is unknown. In some cases of head injury there is a moderate degree of general swelling of the brain but there is no satisfactory evidence which supports the common belief that this is a cause of the associated disorders of consciousness. This œdema will, however, contribute to the slightest increase of intracranial pressure which is often demonstrated in the early days after injury. The remarkable bradycardia (Russell, 1934) which often continues for two or three weeks during recovery from closed head injuries may also be due to an œdematous condition of the brain or brain-stem, but the spinal fluid pressure in these cases may be quite normal, and it is important to note that this post-concussional bradycardia is not necessarily associated with impairment of consciousness.

The most definite example of local cerebral œdema occurs in the type of case studied by Greenfield (1939) in which a tumour or abscess involving one cerebral hemisphere

To set up the apparatus the lumbar puncture needle is introduced into the subject, who reclines on his side with the head on the same level as the lumbar spine. Patients have been found to tolerate this procedure quite well for periods up to six hours. The apparatus is then filled with sterile normal saline and is connected to the lumbar puncture needle which is firmly strapped to the subject's back. After calibration of the system the tap on the needle is opened and the recording drum is started.

Blood-pressure and pulse readings are taken every quarter of an hour. A sufficient time is allowed to elapse for a satisfactory basic level of lumbar cerebrospinal fluid pressure to be established—usually a period of about an hour—after which the injection of 100 c.c. of 50% sucrose solution is given intravenously. Continuous records of the lumbar cerebrospinal fluid pressure are made over periods up to three and a half hours following the injection.

Controls

Adequate controls of the apparatus without the injection of sucrose were carried out in ten subjects with a normal and nine with a raised intracranial pressure. Typical tracings from controls with normal pressure are shown in fig. 2.

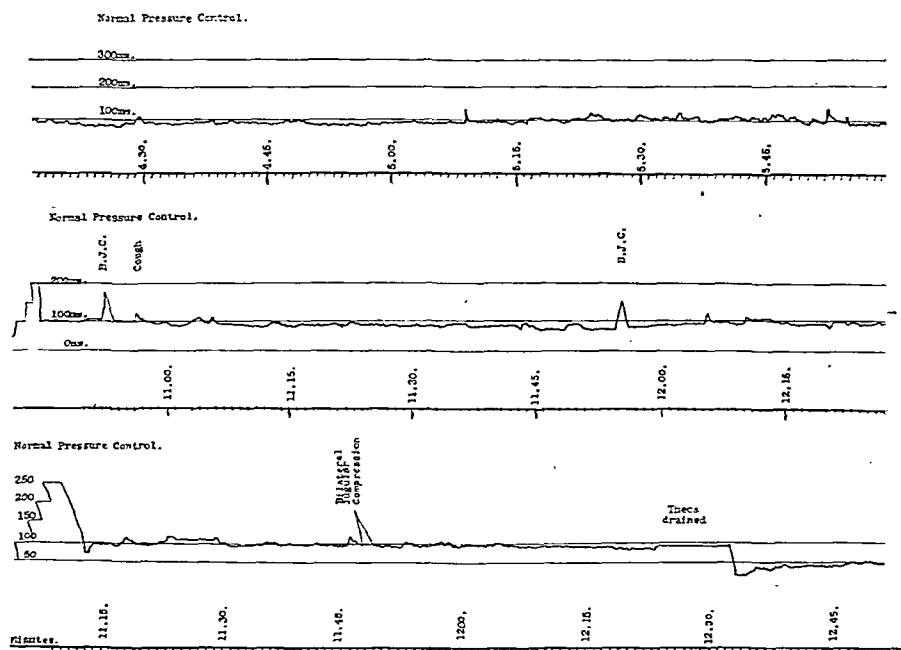


FIG. 2.

The cerebrospinal fluid pressure was steadily maintained and the recording apparatus was quite sensitive, the tambour quickly responding to sudden transient rises in pressure produced by coughing or by jugular compression.

Figure 3 shows three similar tracings from high pressure controls.

Sucrose Injection. Normal Pressure Subjects

The effect of an injection of sucrose on eight subjects with a normal cerebrospinal fluid pressure—i.e. below 150 mm. of water—was next investigated, seven of the subjects receiving 100 c.c. of 50% sucrose solution, the eighth receiving only 50 c.c.

A definite reduction in pressure was obtained in four of these cases, the maximum effect being a reduction in pressure of 30 mm. of cerebrospinal fluid which, maximal in fifteen minutes, was maintained for only one hour. In each of the other three cases showing a reduction in pressure, a fall equivalent to 25 mm. of water was obtained in ten to fifteen minutes but which lasted only from one to two and a half hours. The case receiving only 50 c.c. of sucrose showed but a transient and negligible reduction in pressure.

Three typical tracings from records are shown in fig. 4, the uppermost being a

An Investigation into the Effect of Intravenous Injections of Sucrose on the Cerebrospinal Fluid Pressure as Measured by Lumbar Puncture

By J. HAMILTON PATERSON, M.B., B.S.

THOUGH the use of concentrated solutions of sucrose administered by intravenous injection for the purpose of effecting a reduction in intracranial pressure has been frequently practised in many neurosurgical units during recent years, it has been felt desirable to inquire more fully into the degree of actual reduction of intracranial pressure so obtained. The effect of sucrose thus administered was therefore investigated in a small series of cases with normal and with raised intracranial pressures by means of a continuous recording apparatus. A standard quantity and concentration of hypertonic sucrose solution—namely 100 c.c. of a 50% solution—was used in all but two cases.

Two methods of performing such an investigation were open to choice, the simpler being repeated lumbar puncture, performed before and after the injection, the cerebrospinal fluid pressure being measured on each occasion. We felt that this method was open to the grave objection that cerebrospinal fluid has been shown to leak from the initial puncture hole in the theca, a fact which has been noted on several occasions by other observers. This leakage has occurred in our own experience and is well illustrated by the following record (fig. 1).

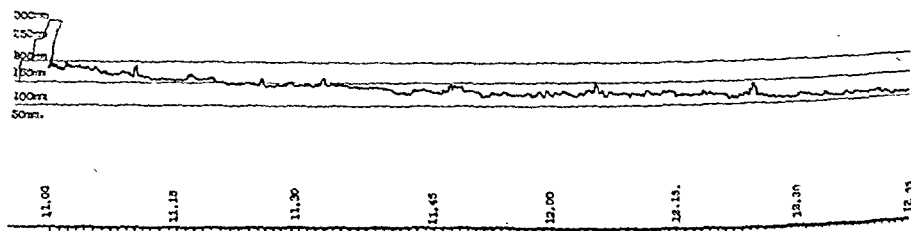


FIG. 1.—High pressure. ? Thecal leakage.

A continuous record was attempted, but owing to difficulties in obtaining a satisfactory record, the lumbar puncture needle, the point of which had been in the sub-arachnoid space, was removed and reinserted. A steady decline in pressure resulted, which could only be reasonably attributed to fluid leaking through the theca from the initial puncture hole.

The other available method is to insert a lumbar puncture needle into the spinal theca in the usual way and, after attaching a suitable manometer, to strap the apparatus securely to the subject for the duration of the investigation. Readings of the cerebrospinal fluid pressure can then be made at regular intervals both before and after the injection of sucrose. This method was the one we employed in earlier experiments. It was found, however, that in many cases the normal fluctuations in the manometer level accompanying the slightest movement on the part of the patient, as well as the respiratory excursions, rendered accurate readings impossible. A recording apparatus was accordingly devised which not only gave a continuous graphic representation of the cerebrospinal fluid pressure, but effectively damped down such extraneous fluctuations in pressure.

This recording apparatus connects the lumbar puncture needle through pressure tubing with a vertical glass tube of medium calibre connected with an air tambour which actuates an inkwriting pen recording on a slowly moving drum. The whole length of rubber tubing and the lower part of the glass tube is filled with normal saline which thus transmits the pressure of the cerebrospinal fluid to the air in the upper part of the glass tube and in the tambour. It should be noted that considerable changes in the pressure can thus be recorded without the displacement of more than one or two cubic centimetres of cerebrospinal fluid. For convenience in calibrating the records a T-piece is fitted in the pressure tubing connected to a saline container which can be raised or lowered to the requisite level and put in communication with the rest of the system by opening a cock. In calibrating the record the level of saline in the glass tube (and consequently the pressure in the tambour) can be controlled against the centimetre rule alongside the glass tube, the zero level of which is set to the level of the lumbar puncture needle.

The lumbar puncture needle used is fitted with a 3-way tap and vertical bayonet mount carrying a rigid glass manometer.

To set up the apparatus the lumbar puncture needle is introduced into the subject, who reclines on his side with the head on the same level as the lumbar spine. Patients have been found to tolerate this procedure quite well for periods up to six hours. The apparatus is then filled with sterile normal saline and is connected to the lumbar puncture needle which is firmly strapped to the subject's back. After calibration of the system the tap on the needle is opened and the recording drum is started.

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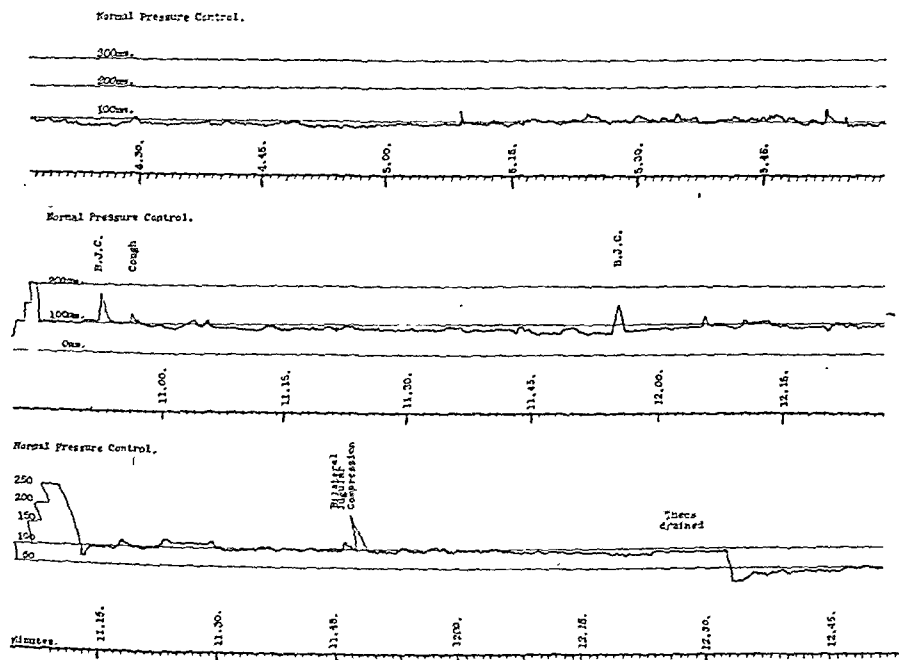


FIG. 2.

The cerebrospinal fluid pressure was steadily maintained and the recording apparatus was quite sensitive, the tambour quickly responding to sudden transient rises in pressure produced by coughing or by jugular compression.

Figure 3 shows three similar tracings from high pressure controls.

Sucrose Injection. Normal Pressure Subjects

The effect of an injection of sucrose on eight subjects with a normal cerebrospinal fluid pressure—i.e. below 150 mm. of water—was next investigated, seven of the subjects receiving 100 c.c. of 50% sucrose solution, the eighth receiving only 50 c.c.

A definite reduction in pressure was obtained in four of these cases, the maximum effect being a reduction in pressure of 30 mm. of cerebrospinal fluid which, maximal in fifteen minutes, was maintained for only one hour. In each of the other three cases showing a reduction in pressure, a fall equivalent to 25 mm. of water was obtained in ten to fifteen minutes but which lasted only from one to two and a half hours. The case receiving only 50 c.c. of sucrose showed but a transient and negligible reduction in pressure.

Three typical tracings from records are shown in fig. 4, the uppermost being a

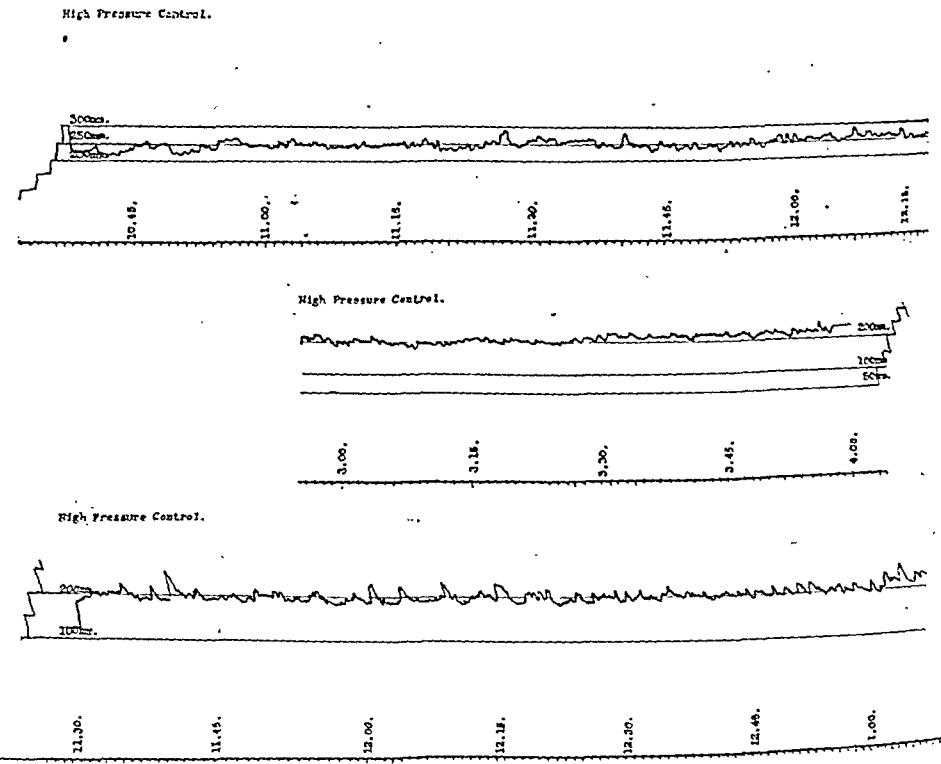


FIG. 3.

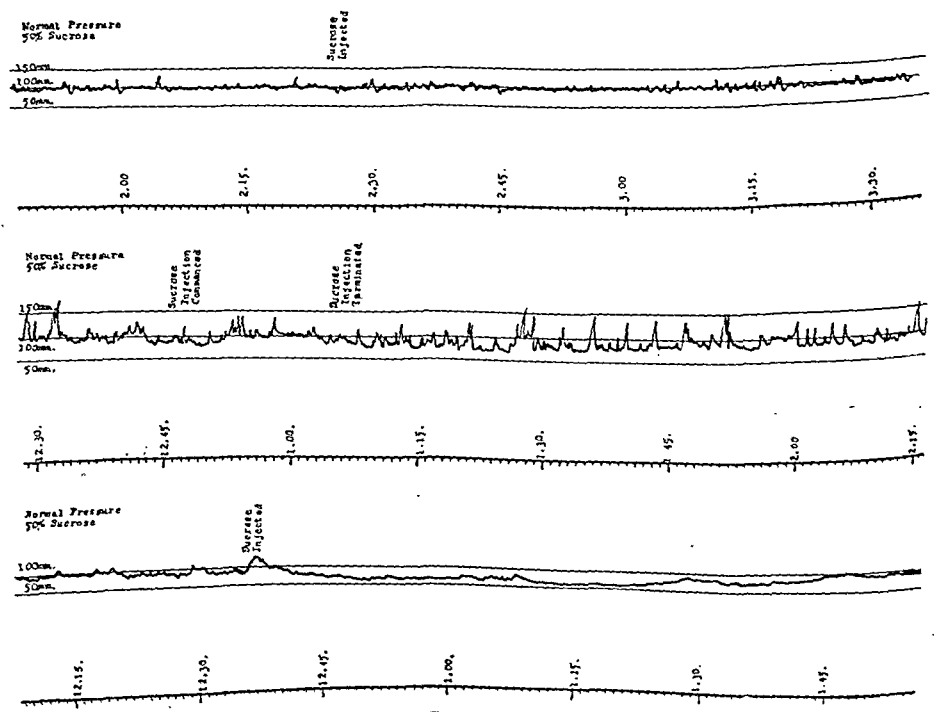


FIG. 4.

case in which no effect was obtained; the middle showing a reduction in pressure of about 25 mm. water; whilst the lowermost illustrates the maximum effect obtained of 30 mm. water.

Sucrose Injection. High Pressure Subjects

Finally the effect of sucrose on eight cases with a raised cerebrospinal fluid pressure was investigated, seven subjects receiving 100 c.c. of 50% sucrose solution, the eighth receiving 50 c.c. only.

A definite reduction in cerebrospinal fluid pressure was obtained in five cases, whilst in three cases despite initial pressures ranging from 200-300 mm. water, no effect was obtained. The maximum reduction in pressure which occurred was obtained in a subject with an initial pressure of 420-440 mm. water; a quarter of an hour after injection of sucrose the pressure had dropped to 380 mm., but in fifty minutes the pressure had risen to 480 mm.

Another case with an initial pressure of 200-210 mm. showed a reduction in pressure after sucrose injection to 170 mm., maintained for two and a half hours. In the other three cases in which an effect was observed, reductions in pressure of 20-30 mm. occurred in fifteen to twenty minutes lasting from fifteen minutes to two hours. This last group included the subject who received only 50 c.c. of sucrose solution.

The three tracings (fig. 5) illustrate typical effects obtained in high pressure cases

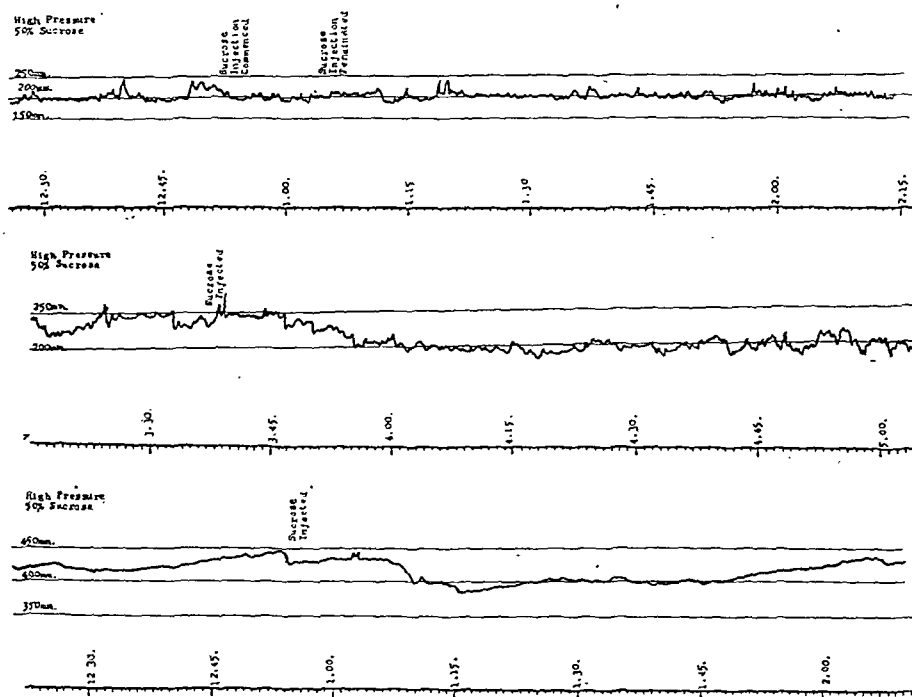


FIG. 5.

after injection of sucrose, the uppermost tracing showing no effect, the middle a reduction in pressure of 30 mm. of water, maintained for nearly two hours, whilst the lowest tracing was taken from the subject who showed the greatest reduction in pressure of all, about 50 mm. of water.

It should be noted that in all the cases investigated no significant alteration in blood-pressure or pulse-rate was noticed, whilst subsequent to the sucrose injection no improvement in the mental state or general condition of the subject was noted during the period of investigation.

Mention should be made of the fact that the subjects under investigation were mostly patients with intracranial tumours, especially those in the high-pressure class. Though it might have been valuable to have performed such an inquiry on a series of acute head injuries, such cases were not deemed suitable for investigation.

CONCLUSION

These results show that, in the type of cases with which we have been dealing, great reductions in lumbar cerebrospinal fluid pressure are not produced by the intravenous injection of 100 c.c. of a solution of 50% sucrose. The greatest fall produced in this series was only 50 mm. of cerebrospinal fluid and was maintained for less than one hour, at the end of which time the pressure rose again to a point 40 mm. above the pre-injection level.

We are forced to the conclusion, therefore, that the reduction of lumbar cerebrospinal fluid pressure produced by such an injection is very small and in most cases is likely to be of little therapeutic value. We have observed no clinical change in the general condition of the patient under investigation and no significant alteration in respiration rate, pulse-rate or blood-pressure. Many observers have, at one time or another, noted striking changes in the general condition of patients suffering from intracranial tumours following injections of hypertonic sucrose solution, a fact which we have occasionally observed ourselves, but we incline to the view that this clinical alteration is not necessarily due to a lowering of the intracranial pressure as measured by lumbar puncture.

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Section of the History of Medicine

President—J. F. HALLS DALLY, M.D.

[February 4, 1942]

The Folk-Lore of the Acute Exanthemata

By J. D. ROLLESTON, M.D.

[Abridged]

DURING the last two congresses of the International Society of the History of Medicine held at Madrid in 1935 and at Zagreb and Belgrade in 1938, medical folk-lore formed a considerable part of the proceedings, as is shown by the fact that at Madrid 10 papers and at Zagreb 44 were devoted to the subject. The contributors of the papers on folk-lore mostly came from the Balkan countries, but communications of this kind were also made by Belgian, French, German, Italian, Norwegian, Portuguese and South American representatives. The only British contributions at these two congresses dealing with folk-lore were a paper by the late Dr. John Comrie on "The Medical Folk-Lore of Scotland" at the Madrid meeting and one by myself on "Some English Folk-Lore Remedies" at Zagreb. According to Dr. Albert Bazala, general secretary of the Yugoslav Congress (*Compte rendu de l'onzième Congrès international d'histoire de la médecine 1938-42*), the Proceedings of the Madrid congress were destroyed in the Spanish civil war, and it is more than probable that those of the Zagreb and Belgrade congress shared the same fate.

The present paper which forms a supplement of my Fitzpatrick Lectures on "The History of the Acute Exanthemata" (1935 and 1936) as well as one of a series of previous studies on medical folk-lore (1939-41) deals exclusively with the folk-lore of small-pox, measles, scarlet fever and chicken-pox. There is no folk-lore connected with rubella.

In contrast with other diseases, particularly whooping-cough, pulmonary tuberculosis, skin diseases, especially warts, and eye diseases, the folk-lore of the eruptive fevers is somewhat scanty though sufficiently rich to justify a special communication. The abundance of folk-lore connected with each exanthem bears a direct relation to the antiquity or the importance of the disease, so that the amount of folk-lore of small-pox, measles, scarlet fever and chicken-pox is represented in the order named.

Although the existence of the acute exanthemata in Ancient Greece and Rome was for a long time a hotly disputed subject of controversy, it is now generally agreed that the eruptive fevers cannot be traced further back than the Middle Ages. Nothing, therefore, suggestive of a popular conception of the acute exanthemata can be found in those two great storehouses of classical folk-lore. Pliny's *Natural History* and the *Deipnosophists* of Athenaeus. Nor indeed is there anything to indicate the existence of these diseases in Cockayne's work containing the folk-lore of Early England. There do not seem to be any popular causes suggested for their occurrence, such as catching cold, sexual excess, or punishment for an evil life, as in the case of pulmonary tuberculosis, skin diseases and venereal diseases. Except small-pox none of the exanthemata has been attributed to malignant spirits or demons. In comparison with curative measures and in accordance with medical folk-lore generally, prophylaxis is rarely employed in the acute eruptive fevers. There are several examples of the same treatment being applied to this group of diseases generally, such as overheating the patient and flagellation with nettles to bring out the rash, the use of red hangings and bedclothes and coprotherapy. Popular errors connected with the acute exanthemata generally include a tendency to regard them as trivial, a belief responsible for much infant mortality, and the view that they do not occur more than once in a patient's lifetime.

SMALL-POX

Although small-pox did not exist in classical antiquity, it is by far the oldest of the acute exanthemata, having been known from time immemorial in India owing to the temple worship of a deity whose protection and help were invoked on the outbreak of an epidemic. Central Asia was also an ancient focus of the disease as well as China. There has, therefore, been much more time for an abundant growth of folk-lore to have developed in the case of small-pox than in that of the other acute exanthemata. Another explanation of its richness in popular tradition is the fact that small-pox is widely prevalent in the tropics where scarlet fever is almost unknown, and measles is exceptional unless introduced from countries where it is endemic.

To small-pox alone of the acute exanthemata has a divine origin been assigned, although a few patron saints have been invoked in the case of measles. There are numerous examples on record of outbreaks of small-pox in different parts of the world, such as China, Korea, India, Japan and Malay Archipelago and West Africa, in which the epidemic has been attributed to the agency of a goddess or demon or much more rarely a god, to win whose favour or repel their visitation various methods were employed.

Closely associated with the supposed existence of a divine being causing small-pox is the avoidance of calling the goddess or disease by proper names and substituting more or less euphemistic terms.

Among the Gayos of Sumatra for example the name of small-pox may not be used in the house of a man suffering from it, and the words characteristic of the disease such as "ugly", "red", "stinking", &c., are also forbidden, the title of "prince of the averters of misfortune" being substituted (Frazer. "Golden Bough. Taboo and the Perils of the Soul", 410). A similar prohibition to mention the disease by its name is to be found in Bulgaria according to Stoianoff, who says that when a case occurs in a household a loaf of bread or pastry is made, spread with honey and given to the neighbours with the recommendation to eat it "for the health of the sweet and honeyed disease".

Allusion may be made in this connexion to the euphemistic modern Greek term for small-pox, viz. *εὐλογία* (kindly inflammation or blessing) which recalls the ancient Greek denomination of Eumenides applied to the Furies.

The idea of transfer of a disease to other persons, animals or inanimate objects which plays so important a part in medical folk-lore is plentifully illustrated in small-pox, examples of the kind being found not only in savage races but also in this country (Frazer, Moncrief).

Prophylaxis.—Lady Mary Wortley Montagu's letter to a friend in 1717 shows that inoculation was a folk-lore practice carried out by old women in Turkey long before it was adopted by the medical profession in England and France. The combination of vaccination with folk-lore methods is illustrated in India where the former is accompanied by worship of the small-pox goddess (Hovorka and Kronfeld), and in Morocco where the vaccinator, if the operation is to be successful, must have killed an enemy in battle (Legey).

In Balkan countries where vaccination is regarded as the work of the devil, other modes of protection are employed by the people such as heavy smoking, keeping grass snakes and swallows' nests and chewing bilberries or garlic (Hovorka and Kronfeld).

Treatment.—Hastening the appearance of the eruption by warm coverings and warming pans, administration of warm tea and brandy, and decoctions of lentils or cynoglossum was a favourite method of treatment. The use of red hangings and bedclothes was used in Japan in the tenth century A.D. or long before John of Gaddesden, who is usually credited with being the first to employ this method. Animal cures consisted in roast mice (Dawson), blood of pigs, pigeons and snails, and coprotherapy in the form of the feces of cats, cows and sheep (Delaunay, Moodie, Lean). Miscellaneous remedies were as follows: burying the patient up to the neck, a method frequently employed in Gloucestershire, stealing a bun from the shop of a person whose wife when she married did not change her name, and giving it to the patient to eat, a practice current in Cheshire (Wright), and washing the patient at the grave of a murdered man and taking him home by a different way from that by which he came (Spoer).

Patron saints.—Doubtless, owing to its more serious character more patron saints are connected with small-pox than with any of the other acute exanthemata.

According to Du Broc de Segagne the following saints may be invoked.—St. Bonose, St. Elie, St. Magin, St. Martin of Tours, St. Matthias, St. Saturnin and St. Rite di Cascia. St. Bonose is invoked because his form of martyrdom consisted in having his flesh torn by iron hooks which produced scars resembling those left by small-pox. According to Bonnerjea St. Martin of Tours may be invoked by those who object to vaccination. St. Nicaise was also invoked with the following words: "In the name of our Lord Jesus Christ, may the Lord protect these persons, and may the work of these virgins ward off the small-pox. St. Nicaise had the small-pox, and he asked the Lord (to preserve) whoever carried his name inscribed 'O, St. Nicaise! thou illustrious bishop and master, pray for me a sinner, and defend me by thy intercession from this disease. Amen'" (Pettigrew, p. 82; Black, p. 93).

MEASLES

Popular errors.—Measles has more popular errors connected with it than any other acute exanthemata, viz. (1) Measles is often considered as a trivial disease, whereas its severity and fatality rank very high among the acute diseases of children. (2) It is believed that measles clears out the system and makes a child less liable to contract other diseases. (3) The old "heating regimen" still survives in the popular belief that

children suffering from measles should not be washed or their body linen changed for the first week of the disease (Rorie).

Transfer.—There are several examples of the belief that measles can be best treated by transfer to animals or plants.

Animal remedies.—In none of the acute exanthemata is coprotherapy more frequently employed than in measles especially in the early stages of the disease with the object of bringing out the rash, especially in the form of sheep's dung, which is euphemistically called "nanny-goat tea", "lamb tea" or "nanny pill tea" (Levine).

Plant remedies.—The following plants have been recommended for bringing out the rash: rosemary boiled in water with a tablespoonful of rum added, dried elderberry blossom or marigold leaves with water, decoction of peas either externally or internally, oil of sweet almond sweetened with syrup of maidenhair, and one or two spoonfuls of the syrup of the flowers of the elder.

Mineral remedies.—The magic and curative properties attributed to stones, especially those with a hole at one end, are exemplified by the Long Stone at Minchenhampton (Glos.) where children are passed through its hole to cure them of measles and other diseases (Hole). In China pearls with honeysuckle are recommended for measles which affects children's eyes (Read). In the seventeenth century bezoar stone was much commended against measles or against small-pox, pestilence, malignant fevers and the like (Bishop).

Patron saints.—A certain number of saints, both in France and Belgium, are invoked for the cure of measles as well as for other diseases, viz. St. Adelard, St. Maginus or St. Maxime (Du Broc de Segagne) and St. Foy and St. Laurent (Tricot-Royer).

Other folk-lore remedies for measles are washing the patient at the grave of a murdered man and taking him home by a different way from that by which he came; abstinence from water for ten days, flagellation with nettles and administration of roast mouse.

SCARLET FEVER

Nomenclature.—More than a hundred years before it received its present name in the middle of the seventeenth century, scarlet fever was popularly known as *rossania*, *rossalia*, *sofersa*, *sturola*, *scurula* and *rosagia*.

Popular errors.—These include the belief, which is not yet entirely extinct even among the medical profession, that the term "scarlatina" indicates a mild form of the disease. Another popular error is that a mild case of scarlet fever is capable of transmitting only a mild attack to another person, whereas it often happens that a mild attack in one individual gives rise to a virulent form in another.

Transfer.—In certain parts of England and Ireland some of the patient's hair is cut off and passed down the throat of an ass who is thereby supposed to receive the infection. A reputed cure for scarlet fever in Cornwall is to drive sheep through the house containing the patient in the belief that they will take the fever with them.

Prophylaxis.—In New Hampshire a camphor bag worn round the neck is regarded as particularly effective in the prevention both of scarlet fever and measles. An amulet containing sulphur has also been used in Scotland in any place where scarlet fever is prevalent.

Treatment.—The fear of the rash striking inwards which is prevalent in the case of smallpox and measles is also illustrated in scarlet fever, and the patient is therefore made to sweat profusely by various means including the drinking of hard cider, decoctions of rue accompanied by milk and honey and "nanny-goat tea". A popular remedy for scarlet fever in Ireland is the application of the blood of a hare by means of rags which are afterwards burnt. This may be an example of the "doctrine of signatures". The same explanation may be offered for the tongue-shaped pieces of red cloth known as "red tongue" which were tied round the patient's head and were sold in Fleet Street as late as the early part of the nineteenth century as a cure for scarlet fever.

Plant remedies.—In German Switzerland a decoction of *Agrimonium Eupatoria* was given and in Ruthenia a decoction of rue and garlic. In Belgium mullein flowers mixed with an egg and olive oil and a spoonful of holy water were made up into a poultice and then thrown over the shoulder without looking back.

CHICKEN-POX

Nomenclature.—Although this disease is probably as old as small-pox owing to its usually mild character very little folk-lore is attached to it. Like rubella it has a multitude of synonyms in many countries which is out of all proportion to its real significance. In Britain the synonyms at one time were "water-pox", "water-jags", "the crystals", "mirls" or "blibes". In France it was called "crystalline" and "petite vérole volante"

and in Germany "Schiffspocken" and "Hühnerpocken". I am indebted to His Excellency, M. D. Caclamanos, the former Minister of Greece, for informing me that the modern Greek for chicken-pox is αἰμοβλαγιά, the termination βλαγιά being a corruption of εὐλογία which means "blessing". The term "chicken-pox" was first used in medical literature by Richard Morton, who in his *Pyretologia* (1694) speaks of a form of small-pox "called in the vernacular the Chick-pox". Fuller also in a paragraph on "Ritteln or Chicken-pox" in his *Exanthematologia* (1730) writes "I have adventur'd to think that this is that which among our women goeth by the name of Chicken-pox." Under the name of "crystalli", which was obviously chicken-pox Vidus Vidius (Guido Guidi), physician to François I, stated that the vulgar called it *ravaglione*. It is therefore obvious that folk-lore terms preceded the medical names for this usually trivial exanthem. Another example of folk-lore anticipating scientific medicine is furnished by the fact recently mentioned by W. N. Pickles that the connexion between herpes zoster and chicken-pox has been known among the people in Aysgarth in Yorkshire long before this connexion was demonstrated by Bokay in 1892.

Treatment.—Among the Czechs dry plums are applied to the eruption and St. Barbara is invoked. In Belgium the eruption is powdered with potato meal and starch.

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Clinical Section

President—PHILIP TURNER, M.S.¹

[February 13, 1942]

MEETING HELD AT KING'S COLLEGE HOSPITAL, LONDON

We regret to have to report that Dr. de Bec Turtle died suddenly on December 3, 1941. A tribute to his memory was paid at the Council meeting of the Royal Society of Medicine and at the meeting of the Council of the Clinical Section of which he was President. The Presidency of the Section has been taken over by Mr. Philip Turner, who was elected on December 22, 1941.

Atrial Septal Defect.—PHILIP ELLMAN, M.D.

E. M. M., aged 26, typist.

First seen May 1940 then completely free from symptoms. She stated that in course of routine examination by Civil Service M.O. (on account of twenty-five days' sick leave in five years) "a murmur was found by the doctor".

Clinical examination.—Somewhat high-coloured facies, no real cyanosis. Heart enlarged to left; apex beat in 5th space 4 in. from mid-line; rhythm regular; rate 84; rough systolic murmur best heard over pulmonary area; pulmonary second sound markedly accentuated; blood-pressure 130/90.

X-ray examination of heart.—Transverse diameter increased, owing to enlargement of right ventricle, with marked aneurysmal dilatation of pulmonary artery (this can be felt) and its branches. On fluoroscopic examination right branch of pulmonary artery shows marked aneurysmal dilatation and excessive pulsation ("hilar dance"); aorta unusually small (fig. 1).



FIG. 1

FIG. 1.—Antero-posterior film from case of atrial septal defect showing aneurysmal dilatation of the pulmonary artery, enlarged right hilar shadow due to enlargement of the right branch of the pulmonary artery and enlargement of the left border of the heart, due to enlarged right ventricle.

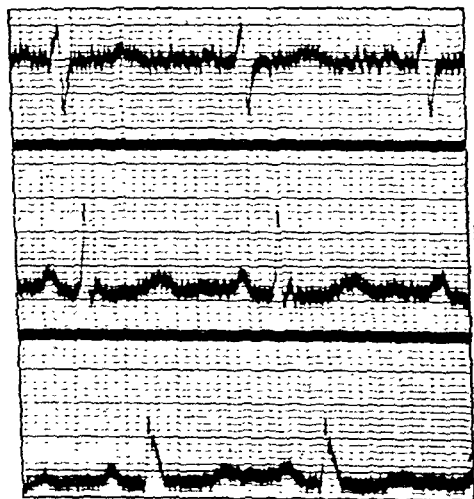


FIG. 2

FIG. 2.—Electrocardiogram (see text).

Electrocardiogram shows right axis deviation; rather large bifid P wave in lead II; widening and slurring of QRS in I and III (fig. 2).

The condition is interesting in that, until recently when breathlessness has become rather more marked, the patient has been free from symptoms. She has no clubbing or real cyanosis and has hitherto led a normal life.

Tuberculous Polyserositis.—PHILIP ELLMAN, M.D.

J. T., aged 20, gardener. Present illness began with an acute attack of "influenza" in February 1941, following which he complained of increasing severe breathlessness, a cough with a trace of mucoid sputum, and some swelling of the legs.

Previous history.—No noteworthy illnesses. *Family history.*—Nothing relevant.

On admission to hospital seven weeks after onset of illness, patient was very ill and grossly orthopnoic. Temperature 97° – 102.8° F.; pulse-rate 130; no appreciable chest pain; no cyanosis or engorgement of the veins of the neck.

Examination.—Chest: Marked limitation of movement of both sides of chest, especially left, and bulging of præcordium; marked dullness over almost the whole of left chest (except for 1st and 2nd intercostal space, anteriorly and posteriorly), and the whole of left axilla; also dullness over right lower zone, somewhat higher in the axilla; breath sounds absent over areas of dullness.

Heart: Apex beat not then palpable; heart sounds feeble and rhythm regular; rate 130; blood-pressure 130/75; for first three weeks after admission pericardial friction audible at base of heart; no significant murmurs.

Abdomen: Ascites. Liver palpable. Some oedema of legs. Urinary output at first diminished but now normal.

X-ray examination (15.4.41).—Lungs: Bilateral pleural effusion especially marked on the left side. Heart: Cardiac silhouette markedly increased. Appearances very suggestive of pericardial effusion (fig. 1).

Electrocardiogram (Dr. East).—Rather low voltage curves in limb leads; negative T waves in all leads, especially præcordial leads (fig. 3).

All these findings strongly suggested an acute polyserositis. This was confirmed by paracentesis of the pleuræ and later of the pericardium.

Pathological investigations (Drs. Dacie and Nabarro) (21.4.41).—Pleural fluid: Clear, straw-coloured, some clotting. Polys. 2%, lymphos. 98%. Culture sterile. Ziehl-Neelsen: no T.B. seen. Total protein 4.65 mg.%, serum albumin 2.06, serum globulin 2.59, albumin globulin ratio 0.79. Fluid from pericardium: Clear straw-coloured fluid. Polys. 90%, lymphos. 10%. Culture sterile. Ziehl-Neelsen: no T.B. seen. Total protein 4.7 mg.%, serum albumin 1.75, serum globulin 2.95, albumin globulin ratio 0.59 (Dr. Gray).

Note the difference between the cytology of the pleural and the pericardial fluids.

Guinea-pigs inoculated with pleural and pericardial fluids showed tubercles in spleen and lungs.

Urine: Albumin: a trace. Deposit: an occasional R.B.C. and W.B.C. seen. Direct smear: no organisms seen. Culture: no growth.

Blood: B.S.R. 34 mm. at the end of one hour. R.B.C. 4,260,000; Hb. 78%; W.B.C. 5,600. Polys. 74%.

Treatment.—The patient has been treated on general lines for his tuberculosis: complete rest, restricted fluids, paracentesis of pleuræ and pericardium. He has also had a course of salyrgan injections. The fluid in the pericardium has been replaced by air on several occasions. He had clinical evidence of a hydro-pneumopericardium with characteristic auscultatory physical signs of a "tinkling splash" over the præcordium. The introduction of air appears to have retarded the rate of re-accumulation of pericardial exudate.

The final X-ray film (fig. 2) shows a pure pneumopericardium with no fluid and the auscultatory signs have accordingly disappeared. The markedly thickened pericardium is now well seen by radiograph and the heart does not appear to be enlarged. The ascites and oedema of the legs have completely subsided; he still has a small right pleural effusion, the left having resolved. Now completely afebrile; pulse-rate 80–100. He is up all day and has gained weight. General condition good. Blood sedimentation rate and blood-count normal. Repeated examinations of pleural fluid have shown no changes in cytology. Last examination of pericardial fluid (January 1942) showed 88 leucocytes per c.mm., now mainly lymphocytic.

Comment.—Note the comparatively good response to treatment for the time being: the difference until quite recently in the cytology of the pleural and pericardial effusions and the question whether the introduction of a pneumopericardium can have any influence in the prevention of chronic adhesive pericarditis (Pick's disease).

Diabetic Dwarfism with Hepatomegaly.—W. G. OAKLEY, M.D.

Boy, aged 7½. Onset of diabetes about March 1939. Treated with diet and 6 units of insulin twice a day. Later admitted to Hutton Residential School (Diabetic Unit) where the insulin was increased to 10 units twice a day (soluble insulin).

November 1941: found to have characteristic appearance with "moon" face, infantile hands, and enlarged liver. Blood-sugar found to be swinging greatly; treatment changed to 12 units of zinc protamine insulin with 8 units of soluble insulin once a day in the morning before breakfast.



FIG. 1

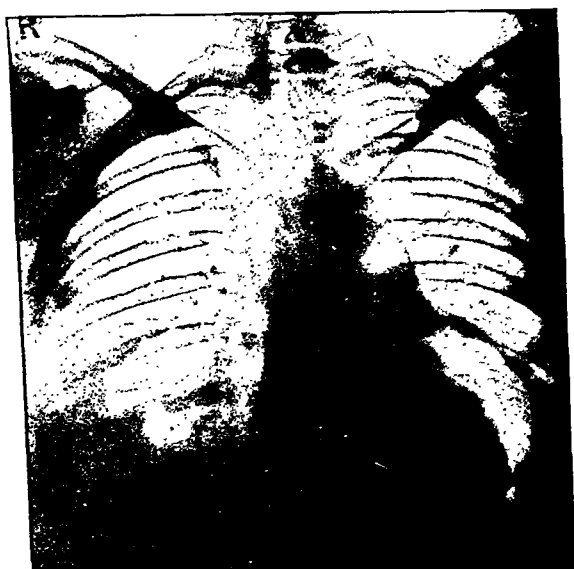


FIG. 2

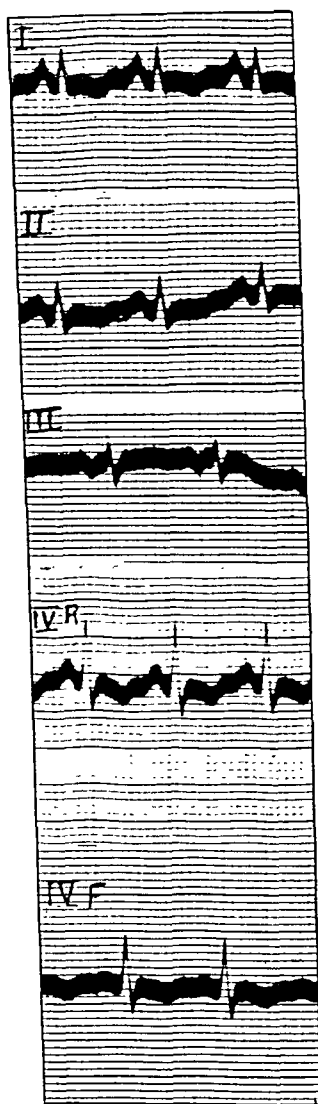


FIG. 3

FIG. 1.—Film of case of polyserositis, bilateral pleural effusions are well illustrated.

FIG. 2.—Pericardial effusion has now been aspirated and replaced by air. A pneumopericardium is well shown and the thick pericardial wall is well demonstrated.

FIG. 3.—Negative T waves in all leads—except III which is iso-electric. Precordial leads definitely negative T waves. Rather low voltage limb leads.

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Examination.—Chest: Marked limitation of movement of both sides of chest, especially left, and bulging of præcordium; marked dullness over almost the whole of left chest (except for 1st and 2nd intercostal space, anteriorly and posteriorly), and the whole of left axilla; also dullness over right lower zone, somewhat higher in the axilla: breath sounds absent over areas of dullness.

Heart: Apex beat not then palpable; heart sounds feeble and rhythm regular; rate 130; blood-pressure 130/75; for first three weeks after admission pericardial friction audible at base of heart; no significant murmurs.

Abdomen: Ascites. Liver palpable. Some oedema of legs. Urinary output at first diminished but now normal.

X-ray examination (15.4.41).—Lungs: Bilateral pleural effusion especially marked on the left side. Heart: Cardiac silhouette markedly increased. Appearances very suggestive of pericardial effusion (fig. 1).

Electrocardiogram (Dr. East).—Rather low voltage curves in limb leads: negative T waves in all leads, especially præcordial leads (fig. 3).

All these findings strongly suggested an acute polyserositis. This was confirmed by paracentesis of the pleura and later of the pericardium.

Pathological investigations (Drs. Dacie and Nabarro) (21.4.41).—Pleural fluid: Clear, straw-coloured, some clotting. Polys. 2%, lymphos. 98%. Culture sterile. Ziehl-Neelsen: no T.B. seen. Total protein 4.65 mg.%, serum albumin 2.06, serum globulin 2.59, albumin globulin ratio 0.79. Fluid from pericardium: Clear straw-coloured fluid. Polys. 90%, lymphos. 10%. Culture sterile. Ziehl-Neelsen: no T.B. seen. Total protein 4.7 mg.%, serum albumin 1.75, serum globulin 2.95, albumin globulin ratio 0.59 (Dr. Gray).

Note the difference between the cytology of the pleural and the pericardial fluids.

Guinea-pigs inoculated with pleural and pericardial fluids showed tubercles in spleen and lungs.

Urine: Albumin: a trace. Deposit: an occasional R.B.C. and W.B.C. seen. Direct smear: no organisms seen. Culture: no growth.

Blood: B.S.R. 34 mm. at the end of one hour. R.B.C. 4,260,000; Hb. 78%; W.B.C. 5,600. Polys. 74%.

Treatment.—The patient has been treated on general lines for his tuberculosis: complete rest, restricted fluids, paracentesis of pleura and pericardium. He has also had a course of salyrgan injections. The fluid in the pericardium has been replaced by air on several occasions. He had clinical evidence of a hydro-pneumopericardium with characteristic auscultatory physical signs of a "tinkling splash" over the præcordium. The introduction of air appears to have retarded the rate of re-accumulation of pericardial exudate.

The final X-ray film (fig. 2) shows a pure pneumopericardium with no fluid and the auscultatory signs have accordingly disappeared. The markedly thickened pericardium is now well seen by radiograph and the heart does not appear to be enlarged. The ascites and oedema of the legs have completely subsided; he still has a small right pleural effusion, the left having resolved. Now completely afebrile; pulse-rate 80-100. He is up all day and has gained weight. General condition good. Blood sedimentation rate and blood-count normal. Repeated examinations of pleural fluid have shown no changes in cytology. Last examination of pericardial fluid (January 1942) showed 88 leucocytes per c.mm., now mainly lymphocytic.

Comment.—Note the comparatively good response to treatment for the time being: the difference until quite recently in the cytology of the pleural and pericardial effusions and the question whether the introduction of a pneumopericardium can have any influence in the prevention of chronic adhesive pericarditis (Pick's disease).

Diabetic Dwarfism with Hepatomegaly.—W. G. OAKLEY, M.D.

Boy, aged 7½. Onset of diabetes about March 1939. Treated with diet and 6 units of insulin twice a day. Later admitted to Hurton Residential School (Diabetic Unit) where the insulin was increased to 10 units twice a day (soluble insulin).

November 1941: found to have characteristic appearance with "moon" face, infantile hands, and enlarged liver. Blood-sugar found to be swinging greatly; treatment changed to 12 units of zinc protamine insulin with 8 units of soluble insulin once a day in the morning before breakfast.



FIG. 1

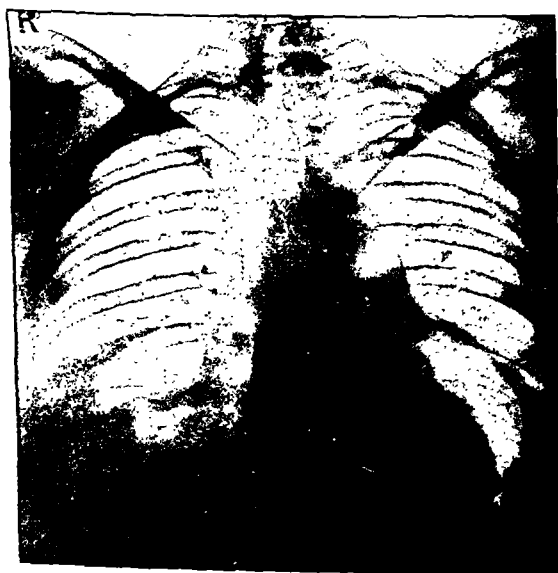


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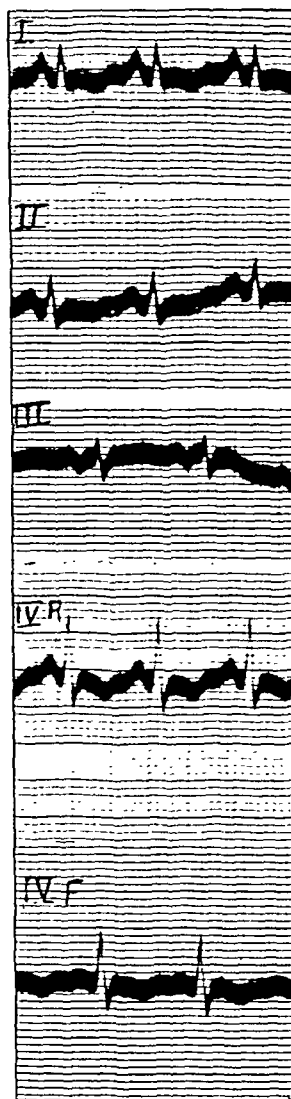


FIG. 3

FIG. 1.—Film of case of polyserositis, bilateral pleural effusions are well illustrated.

FIG. 2.—Pericardial effusion has now been aspirated and replaced by air. A pneumopericardium is well shown and the thick pericardial wall is well demonstrated.

FIG. 3.—Negative T waves in all leads—except III which is iso-electric. Præcordial leads definitely negative T waves. Rather low voltage limb leads.

Tuberculous Polyserositis.—PHILIP ELLMAN, M.D.

J. T., aged 20, gardener. Present illness began with an acute attack of "influenza" in February 1941, following which he complained of increasing severe breathlessness, a cough with a trace of mucoid sputum, and some swelling of the legs.

Previous history.—No noteworthy illnesses. *Family history.*—Nothing relevant.

On admission to hospital seven weeks after onset of illness, patient was very ill and grossly orthopnoëic. Temperature 97° - 102.8° F.; pulse-rate 130; no appreciable chest pain; no cyanosis or engorgement of the veins of the neck.

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On examination.—Shuffling gait; patient walks on a wide base with tendency to deviate to the right. No Rombergism; slight prosis and slight lateral nystagmus. Both arms show considerable ataxia, brisk deep reflexes and no sensory changes. Abdominal reflexes absent. Legs: Considerable inco-ordination; plantar reflexes indefinite, probably extensor. Mild hyperpiesis.

It is considered that he has a late diffuse cerebellar degeneration. There has been some improvement in symptoms since treatment with vitamin B complex.

Thoracic Swelling: Tuberculous Abscess.—A. GILPIN, M.D.

Female, aged 30. Complained of pain in the chest during the past year, with swelling of the right side of the chest for one month.

On examination.—General condition good. No abnormality in the cardiovascular system. A diffuse painless swelling, about the size of a plum, was situated over the 10th and 11th ribs in the posterior axillary line. There was impaired movement and air-entry over the lower part of the right chest. Aspiration of the swelling gave no result.

Investigations.—B.S.R. 52 mm. in the first hour. W.B.C. 9,400 per c.mm. X-ray of chest showed an opacity in the lower lateral part of the right lung, suggesting an encysted pleural effusion. X-ray of ribs and vertebræ showed no bony disease.

This swelling has since been operated upon, and proved to be a tuberculous abscess, although the source of the infection still remains obscure.

[March 13, 1942]

MEETING HELD AT UNIVERSITY COLLEGE HOSPITAL, LONDON

Cystic Disease of the Lung.—K. J. MANN, M.D. (for KENNETH HARRIS, M.D.).

Mr. L., aged 65. *History.*—Perfectly well up to 1938. No cough. No sputum.

1938: Catarrhal jaundice. Chest X-rayed as a routine—bullæ found at right apex and left apex. Discharged from hospital well, no cough.

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October 1941: Cough returned, sputum not purulent. Attended out-patients' department as bronchial for three months.

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Radiogram showed bullæ right apex with slight pneumonitis round it. Temperature fell after sulphapyridine. A few days later temperature rose again. Cough and sputum. Dullness right apex. Bronchial breathing. 30.1.42: Radiogram showed diffuse area of consolidation of right upper half with fluid levels in ? cysts. Sulphanilamide and sulphapyridine tried with no result.

Condition at present stationary with high temperature, cough and sputum.

Sputum showed no tubercle bacilli and no actinomycosis on direct examination or anaerobic culture. Bronchoscopy showed mucoid discharge from right upper bronchus but no evidence of growth.

Four Cases Showing Results of X-ray Therapy.—E. L. G. HILTON, M.B., D.M.R.E.

(1) Spondylitis.

A. H., male, aged 22. 3.2.41: History of pain and increasing stiffness in back of three years' duration. Stiffness and pain in hips for one year. Unable to work.

On examination.—Pale, thin man. Can only walk slowly and with difficulty with aid of two sticks. Great limitation of movement in spine from 6th dorsal vertebra downwards. Limitation of flexion of hip 130°.

Radiogram: Wedging of 6th dorsal vertebra. Spondylitic changes especially in lumbar region with new bone formation.

3.2.41: Deep X-ray treatment to whole spine commenced.

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4.3.42: *General condition.*—Looking much better. Still limitation of movement in spine and hips, but no pain. Can now walk moderately well without sticks. Has put on 1 st. in weight and is at work.

(2) Lymphadenoma.

O. G., female, aged 31. 28.3.41: Six months' history of swelling in groins and neck, and pain in lower part of back. Biopsy report: Lymphadenoma.

Radiogram: Mass in left hilum and mediastinum. ? deposits both sides of sacrum.

Hirschsprung's Disease.—R. C. F. CATTERALL, M.Ch. (for Col. H. C. EDWARDS and Dr. PHILIP ELLMAN).

Boy, aged 18. First seen in 1938 with a history of obstinate constipation with occasional attacks of diarrhoea for more than four years. A barium enema showed an enormously enlarged rectum. Spinal anaesthesia induced with 13 c.c. percaine 1/2,000 introduced between L. 3 and L. 4 failed to cause evacuation of the colon although skin anaesthesia was present up to the level of T. 9.

In January 1941 a presacral and lumbar sympathectomy was carried out. Section of the material removed showed the presence of ganglion tissue. The operation was followed by rectal incontinence and a barium enema taken shortly afterwards showed evidence of muscular contraction in the rectal wall. Patient discharged after a month, much improved.

June 1941: Readmitted with a return of constipation. This improved with colonic lavage and regular use of an anal dilator.

February 1942: Readmitted. The clinical condition has relapsed completely.

On examination his general condition is only fair, his tongue coated, and his temperature, pulse and respiration normal. His mental condition remains poor. The rectum is palpable, full of faeces, and extending almost up to the costal margin. It would appear that the operation has completely failed to produce any lasting change in the condition.

POSTSCRIPT (April 1942).—After a thorough course of colonic lavage and remedial gymnastics he has improved sufficiently to warrant his discharge.

Chronic Pancreatitis.—R. S. BRUCE PEARSON, D.M.

Male, aged 20.

First became jaundiced following a cold in February 1941. The jaundice lasted until June, when he was admitted to the West Kent General Hospital. Temperature varied between 97° F. and 99.5° F. He was obviously jaundiced. Stools pale but not typically putty-coloured; bile present in urine. His condition improved. He put on ½ st. in weight and the jaundice had practically gone by the time of his discharge early in August.

In September he had a further cold and again became more deeply jaundiced. He was admitted to King's College Hospital on 27.10.41 and again improved with rest and a fat-free diet. His liver was then palpable and hard. He was discharged in November for observation as an out-patient, but following a further cold, again became more deeply jaundiced in December and was readmitted.

He is now considerably jaundiced. He has a hard, palpable liver in the epigastrium. van den Bergh test shows a total bilirubin of 20 units.

Chronic Myeloid Leukæmia.—R. B. NIVEN, M.R.C.P.

Female, now aged 11½ years.

This case was presented at a Clinical Meeting in March 1941 (*Proc. Roy. Soc. Med.*, 34, 557, Clin. Sect., 9). The symptoms began in the summer of 1940, and in September 1940 she was discovered to have splenomegaly. In March 1941 the spleen was enlarged as far as the symphysis pubis. The total leucocytes at that time were 450,000 per c.mm.

She has had several courses of deep X-ray treatment and has maintained good general health. The splenic enlargement now reaches only just below the umbilicus.

Blood-count, 6.242 (Dr. Sheila Newstead): R.B.C. 4,990,000; Hb. 86%; C.I. 0.85; W.B.C. 54,800. Polys. 59.2%, metamyelocytes 24.8%, myelocytes 10.8%, lymphocytes 2.4%, eosinophils 0.8%, basophils 2.0%. No myeloblasts.

The condition is very rare in childhood and the case shows the response to deep X-ray treatment.

Two Cases of Cerebellar Degeneration.—WALLACE BRIGDEN, M.B., M.R.C.P. (for Professor S. NEVIN, M.D.).

(1) Male, aged 63. Complained of unsteadiness in walking which commenced about three years ago, and was associated with slight giddiness. Found great difficulty in balancing correctly, and now walks in a drunken fashion. Readily fatigued on slight exertion. Speech has tended to be slow and slurred. He had also noticed increasing stiffness in the legs during these three years.

On examination.—Mild dysarthria and slight nystagmus to the left. Ataxia of both arms. Some loss of power in legs with slight increase in tone, left being more rigid than right, associated with increase in deep reflexes. Plantar reflexes doubtfully extensor.

These symptoms and signs are thought to be consistent with a late cerebellar degeneration.

(2) Male, aged 65. Complained of inability to walk properly, associated with pains in the left leg. These symptoms had been coming on for five years. Difficulty in walking shown by a general unsteadiness, weakness of the left leg and a tendency to walk over to the right.

On examination.—Shuffling gait; patient walks on a wide base with tendency to deviate to the right. No Rombergism; slight ptosis and slight lateral nystagmus. Both arms show considerable ataxia, brisk deep reflexes and no sensory changes. Abdominal reflexes absent. Legs: Considerable inco-ordination; plantar reflexes indefinite, probably extensor. Mild hyperpæsis.

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18.3.41: End of treatment. No pain. Movement slightly less limited. Walking improved.

9.9.41 to 22.9.41: Deep X-ray treatment to both hip-joints, for pain and difficulty in walking. Considerable improvement at end of treatment.

4.3.42: *General condition.*—Looking much better. Still limitation of movement in spine and hips, but no pain. Can now walk moderately well without sticks. Has put on 1 st. in weight and is at work.

(2) Lymphadenoma.

O. G., female, aged 31. 28.3.41: Six months' history of swelling in groins and neck, and pain in lower part of back. Biopsy report: Lymphadenoma.

Radiogram: Mass in left hilum and mediastinum. ? deposits both sides of sacrum.

General condition.—Patient looked ill, with a swinging temperature; high irregular pulse, profuse sweating. Enlarged glands in both cervical chains, groins and axillæ. Spleen just palpable.

28.3.41 to 16.5.41: Course of deep X-ray treatment to all glands and mediastinal mass.

16.5.41: General condition much improved. All glands smaller. Temperature remains normal. No sweating. Discharged fit.

28.10.41: Developed typical erythema nodosum.

28.1.42: Swelling and induration of outer side and middle of left leg. ? lymphadenomatous deposit. This subsided with a course of deep X-ray therapy.

(3) Pharyngeal Carcinoma.

G. L., male, aged 58. *History* (10.12.40).—Difficulty in swallowing and irritation at back of throat for eight weeks.

On examination.—General condition good. Local condition: Swelling of posterior pharyngeal wall in mid-line, occluding view of larynx. Hard mobile gland in right cervical chain, 3 × 2 cm.

Biopsy: Carcinoma with no differentiation into cell nests. Several mitoses present.

Radiogram showed soft tissue swelling of posterior pharyngeal wall.

11.12.40 to 21.1.41: Course of deep X-ray therapy. Swelling much reduced and gland much smaller at end of treatment.

16.1.42: General condition good. Symptom-free. Local condition: Slight glazing of mucous membrane. No signs of recurrence. Gland not palpable.

Radiogram: No sign of the soft tissue swelling.

(4) Carcinoma of Left Breast with Secondary Gland in Axilla. Inoperable.

E. P., female, aged 80. *History* (5.7.40).—Lump in left breast for four months.

On examination.—Lump 4.5 cm. in diameter in nipple region. Nipple retracted. Attached to deep structures. Large, hard, fixed gland in left axilla.

10.7.40 to 13.9.40: Course of deep X-ray therapy.

13.9.40: Lump in breast and axillary gland much smaller.

12.2.42: General condition good. Breast: No tumour palpable. Glands: None palpable. No distant metastases.

Pancreatic Deficiency.—J. F. STOKES, M.R.C.P. (for KENNETH HARRIS, M.D.).

L. W., male, munition worker, aged 39.

Complained of diarrhoea for six months and loss of 4 st. in weight during the same period; slight tingling of hands and feet for a few weeks. Stools have been pale and unformed.

Examination.—Generalized wasting; knee and ankle jerks not obtained. Urine loaded with sugar. Blood-sugar curve diabetic. Stools contain 43% fat, of which 95% is unsplit; no muscle fibres seen in stool; no occult blood. Wassermann reaction negative. Blood-count: R.B.C. 4,000,000; Hb. 94%; C.I. 1.1; W.B.C. 4,400 (polys. 39%, lymphos. 57%, monos. 2%, basos. 2%).

Treatment.—3,000 calorie diet; diabetes controlled on insulin 35-15.5. Pancreatin tabs. ii t.d.s. Stools after treatment contained 20% fat, of which 80% was split; excess of indigested muscle fibre present. Insulin requirement now rising; weight steady.

Malignant Tumour Pressing on Brachial Plexus Complicating Paget's Disease.—HUGH BURT, M.R.C.P.

S. C., male, aged 54.

History.—Pain down inner border left arm and forearm and in 4th and 5th fingers of left hand with increasing weakness of limb nine weeks. Tingling left side of face three weeks, right-sided deafness eight months. Loss of 1 stone in weight six months.

On examination.—Visible swelling left posterior triangle of neck, hard in consistency and firmly attached to underlying structures. Left arm: wasting of all muscles especially small muscles of hand. Anaesthesia to pin-prick and light touch 4th and 5th fingers and ulnar aspect arm and forearm.

Slight left ptosis, weakness of left masseter, right nerve deafness.

Radiograms: High first rib of cervical type on left side with small area of erosion anterior to angle. Opacity in upper part of chest, corresponding to lump. Pelvis: advanced Paget's disease; skull shows Paget's disease with narrowing of left foramen ovale.

Exploration of neck (Professor Pilcher): Tumour fixed posteriorly to prevertebral fascia infiltrating roots C.8 and D.1.

Biopsy: Spindle cells with numerous mitotic figures, some foam cells and giant cells.

Comment.—Paget's disease with cranial nerve involvement; malignant tumour pressing on brachial plexus. ? sarcoma secondary to Paget's disease.

Section of Anæsthetics

President—A. D. MARSTON, D.A.

[May 1, 1942]

Convulsions

By Flying Officer E. A. PASK, M.B., B.Ch., D.A.

ANÆSTHETIC convulsions first began to concern anæsthetists in the year 1926 and in 1927 a number of cases of convulsions occurring in patients under di-ethyl ether anæsthesia were reported to a meeting of this Section [*Proc. Roy. Soc. Med.*, 20, 185 (Sect. Anæsth., 1)].

It has become impossible to regard di-ethyl ether as their specific cause, since convulsions have been reported during anæsthesia induced by many other agents. The convulsions have at different times been attributed to the following: Impurities in the anæsthetic agent; impurities in the oxygen administered; impurities derived from the anæsthetic apparatus; the effect of administering vapours which were too hot; the stimulation of vapours which were too cold; excessive concentrations of carbon-dioxide; concentrations of carbon-dioxide which were too small; excessive amounts of oxygen; too little oxygen, anoxia; excessive morphinization; too little morphine premedication; excessive atropin premedication; respiratory obstruction; surgical stimulation; overheating of the patient; diminished level of calcium in the blood; cerebral congestion; epilepsy; disturbance of renal function; a specific convulsant organism in the nasopharynx.

Despite this variety of causes, the impression has been widespread that the convulsions constitute a homogeneous group, with a specific nature and for which a specific cause should be discoverable.

Are we, in fact, dealing with "anæsthetic convulsions" or with convulsions occurring while the patient is anæsthetized?

In 1926 anæsthetists seemed to have been convinced that they had to deal with a completely new complication of anæsthesia. So strong was this opinion that at first some change in the nature of the ether used, due to an alteration in the method of manufacture, was suspected, but none was discovered.

On reflection, however, this proposition that in 1926 an entirely new complication of anæsthesia arose and has continued ever since, even though the methods of administration prior to that date were substantially similar to many which have been used since, lacks supporting evidence.

At the meeting of this Section in 1927 [*Proc. Roy. Soc. Med.*, 21, 1705 (Sect. Anæsth., 39)], when the first cases described as "Ether Convulsions" were discussed, Dr. Kirby Thomas recalled five cases of convulsions under ether anæsthesia which had occurred prior to 1926, though his comment does not seem to have received great attention. In 1937, in the *British Medical Journal* (ii), 996, Mark Taylor recounts a case of convulsions under ether anæsthesia which occurred in the early eighteen fifties. F. W. G. Smith, in the *Irish Journal of Medical Science*, 1936, p. 582, states that: "Osler and Macrae, writing at a time when ether convulsions were not recognized, in a description of thymic death say that, in some cases, convulsive attacks occur causing death during operation or several hours after the patient has come round from the anæsthetic."

In looking through the Hyderabad and Lancet Commission report on chloroform and other calamities the first thing one notices in this record of disaster is the frequency with which the word "convulsion" occurs.

Of the many cases of convulsions and convulsive movements recorded, I will quote two.

Case 55.—Patient, male, aged 30. Anæsthetic, chloroform. "Raised hands and trembled, kept spitting at the lint, appeared about to vomit. Suddenly he was convulsed as if in an epileptic fit . . . convulsions lasted only a few seconds, he began to breathe with difficulty . . . pulse almost imperceptible and irregular, relapsed and could not be recovered."

The early part of the description suggests that the patient was lightly anæsthetized, yet the cause of death is finally ascribed to "deep coma". If this should not now be described as a case of anæsthetic convulsions, what should it be called?

Case 18.—Female, aged 40. Anæsthetic, ether. "Removal of urethral polyp and two sebaceous cysts. Anæsthesia incomplete, ether was not pushed further because stage of excitement did not come on. When the operation was completed coldness increased with clammy sweats, convulsions, foaming at the mouth. Attack passed away but returned, more severe. After fourth attack the patient died from eclampsia attributed to etherization."

Again the patient is said to be incompletely anæsthetized but it is clear from the account that the anæsthetist was rather confused about the situation. Though the stage of excitement, on which he seems rather unwisely to have relied, did not appear the patient seems to have been deep enough for the operation to be performed. It is not easy to recognize a condition under light ether anæsthesia in which the patient would become convulsed, collapsed and die, after the operation is completed.

Neither can we state that this is unquestionably a case of anæsthetic convulsions, but that is not our problem. If the proposition we have been discussing is to be supported, we must be able to say that such cases as these certainly are *not* cases of anæsthetic convulsions and this we cannot do.

We may, therefore, conclude that the evidence for the assumption that anæsthetic convulsions did not occur prior to 1926, is wholly inadequate, and that the initial case for considering that anæsthetic convulsions constitute a specific entity must fail.

Are we in fact dealing with "anæsthetic convulsions" or are we rather dealing with convulsions arising as a symptom in an unsatisfactory anæsthetic situation?

Many case-histories and commentaries have appeared in the literature in which a particular feature of the symptomatology or pathology has been selected, and a theory of the nature and causation of anæsthetic convulsions built up around it. It is not always realized that though the theory raised may to some extent account for the particular incident described, there is no warrant for assuming its relevance to anæsthetic convulsions in general.

Even more dangerous perhaps, is the practice of drawing deductions from the effectiveness or otherwise of a particular form of treatment. As causation of anæsthetic convulsions is almost certainly complex in all cases we may anticipate their inhibition by different lines of treatment. It is impossible to deduce anything as to causation from the effectiveness of the barbiturates in checking these convulsions. It seems very likely in this case that the agent stops anæsthetic convulsions for much the same reason, whatever it may be, that it stops a variety of other convulsions. We would, perhaps, do well to inquire whether the same observation might not be made concerning other "curative" measures which have on occasion been quoted in support of a particular theory of causation.

At least two published papers, however, have taken a wide view of the situation. In 1937, Brennan in a paper in the *British Medical Journal* showed that many differing factors may combine together to produce a condition of "overheating" in an anæsthetized patient. When in this condition the patient may be convulsed in response to a neuro-genic stimulus.

F. W. G. Smith (1936) reviewed many of the suggested causes of these convulsions and produced evidence to refute a number of them. Evidence was given that this disorder did exist before 1926, and reasons were suggested which might account for an increase in frequency since that date. He too concluded that there is a resemblance between the convulsions of heat-stroke and anæsthetic convulsions.

We know that over-heating of an anæsthetized patient may determine a convulsive state and further that it is particularly easy to cause such over-heating in an anæsthetized patient. In view of our knowledge of the general effects of over-heating, it would, indeed,

be surprising if these two statements were *not* true, but it is quite another matter to assign a "Heat-stroke" basis to *all* cases of anæsthetic convulsions.

It has been suggested that the clinical appearance of the convulsions is in some way pathognomonic of the condition and that because these convulsions resemble, clinically, convulsions arising in different circumstances, then a similarity of causation may be assumed. I remember, for example, being told when I was a student, that a "true" ether convulsion could always be recognized because it commenced with twitchings around the eyes and that this sign was quite pathognomonic. It is true that this sign may enable the "late ether convulsion" to be differentiated from what we may perhaps describe as "induction hyperventilation tetany", but I suggest that neither this nor any other element in the appearance of the convulsions is specific to the condition. Such factors do not permit us to draw either comparisons or differentiations in respect of convulsions occurring in other circumstances.

Here is an account of two cases of convulsions.

PARFITT (1937): "The twitchings which have been noted as occasionally occurring around the mouth and eyes generally begin to increase and spread during this period. Small myoclonic movements are seen in the muscles of the hands and then in the arms, lower limbs, and body. . . . These myoclonic movements increase in force and numbers until combinations of contractions produce jerking of whole limbs and finally violent jactitations may throw the patient out of bed."

BEHNKE (1934): "During the 44th minute, however, a temporary twitching of the left eyebrow was observed. The subject uttered a short cry and developed a convulsion characterized in the beginning by violent clonic movements and then by tonic contractions of the muscles of head, trunk and extremities. Cyanosis did not appear, sphincter control was maintained. With the subsidence of the convulsions the subject was in a stuporous condition for about thirteen minutes. During this period the face turned ashen grey colour, beads of perspiration appeared on the forehead and the breathing became stertorous."

With the elimination of one or two minor "clues" either of these descriptions would pass very fairly for a description of a case of anæsthetic convulsions. The first description applies, however, to a case of convulsion occurring during insulin shock therapy and the second, and this may account for the absence of cyanosis, to convulsions in a subject who was breathing pure oxygen under the increased pressure of three atmospheres.

Convulsions clinically similar to these and to anæsthetic convulsions have been described in subjects submitted to anoxia, to an excessive alveolar concentration of carbon-dioxide, in heat-stroke, in hyperpyrexia and in other conditions. Convulsions are, after all, recognized in general medicine as quite non-specific signs.

CONCLUSIONS

(1) That we may not assume that convulsions occurring under anæsthesia are specific in nature nor that they necessarily have a single specific causation.

(2) That whether a patient is anæsthetized or not, in the presence of certain predisposing conditions, a sufficiently strong stimulus may evoke a convulsion. The strength of the stimulus required will depend on the weight of the predisposing conditions. The nature of this final stimulus will not necessarily be the same in all cases, even though the clinical appearance of the resulting convulsions may be similar. We must also accept the possibility that several different stimuli may act together to produce a stimulus level adequate to excite a convulsion.

We may divide the factors surrounding the development of anæsthetic convulsions into three groups: Predisposing Conditions, Inhibiting Factors, and Immediate Stimuli.

Such a grouping of a certain number of possible factors is shown below. This classification is incomplete and some of the factors are incorrectly placed, and its only basis is that of convenience.

| Predisposing Conditions | Inhibiting Factors | Immediate Stimuli |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------|
| Youth Infection Pyrexia Loss of power to regulate body temperature, due to, e.g. atropinization or deep anæsthesia Low blood calcium level ? ketosis ? apprehension | Barbiturates Nerve block ? morphine | Excess alveolar tension of carbon-dioxide Anoxia Overheating Surgical Trauma Respiratory obstruction ? hyperpnœa ? cerebral engorgement |

Many of the predisposing conditions listed here are recognized as predisposing to the development of convulsions in conscious patients.

I shall illustrate the idea behind this table by two hypothetical examples.

I think we might assume that a patient who is young, has a high temperature, and whose ability to lose heat by sweating has been impaired by a large dose of atropin, might become convulsed if subjected, for example, to a high alveolar tension of carbon-dioxide. Had a barbiturate been given, it is possible that in similar circumstances a convulsion would not appear, since barbiturates seem in general to act as inhibiting factors to convulsions.

For our second hypothetical case we might imagine a more elderly patient in whom a severe infection has been present for a long time. Perhaps deep anaesthesia interferes with the function of the central temperature regulating mechanism, while the final stimulus may be represented by a persistent degree of anoxia, due maybe to the anaesthetic, upon which is superimposed at one point a profound surgical stimulus.

When faced with a case of convulsions under anaesthesia have we perhaps been a little too anxious to inquire: "What is the cause of Anaesthetic Convulsions?" May it not be more profitable to inquire rather: "What combination of factors has led to this disorder, which manifests itself by convulsions, in this particular patient?"

The result of such inquiries may be that eventually a single theory of nature and causation will suffice.

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Section of Psychiatry

President—Lieutenant-Colonel A. A. W. PETRIE, R.A.M.C.

[January 20, 1942]

The Role of Mild Cerebral Commotion in War Neurosis

By W. LINDESAY NEUSTATTER, M.D.

In the 1st war commotional disturbance was at first held responsible for "shell-shock" until its emotional determination became apparent. Although this is generally so, nevertheless under the comprehensive heading of war neurosis a number of symptoms occur, where one is not always satisfied of their psychogenic origin.

Moreover the part played by commotional disturbance in the production of psychiatric disorders is of particular interest in this war, as in the case of civilians, injury allowances are only payable for physical injury, and not for "symptoms induced merely by apprehensions and fears occasioned by enemy activity". And though there is nothing in the Royal Charter to prevent an ex-Service man receiving payments for a purely emotionally determined condition, his claim is far harder to establish if this is the case.

The importance, therefore, of making sure that none of the patient's symptoms is attributable to physical injury is obvious, and the difficult question frequently arises of the assessment of symptoms which may be psychogenic or physiogenic. (1) In particular there are a number of subjective symptoms accepted as due to trauma, but which closely resemble those of psychoneurosis, especially neurasthenia. These are headache, dizziness or giddiness in relation to posture, impaired memory, concentration and judgment, and undue fatigability, a syndrome commonly known as "post-traumatic neurasthenia" (Oppenheim, 1911), a term rightly criticized by Schilder (1940) as suggesting psychogenesis. Mapother (1937) has called it "post-traumatic psychasthenia", but Symonds' "minor confusion syndrome" is perhaps the best term (Symonds, 1940).

(2) Is one justified in assuming that symptoms of anxiety are always psychogenic? Cases have been reported of stable individuals with complete amnesia for a head injury, and with no compensation involved, who have developed anxiety symptoms which are generally regarded as caused physiologically. May not some anxiety reactions, which occur subsequent to some degree of "cerebral trauma", in otherwise completely stable individuals be at least partly physiologically determined? Even if they are caused by fear, may not physiological factors be preventing the regression of fear, which is normal in stable individuals?

(3) How far is it justifiable to argue that when a previously stable individual develops a psychiatric disorder subsequent to enemy action, nevertheless constitutional factors are solely responsible? (A line of reasoning adopted by some invaliding boards.)

(4) How far are the patients symptoms motivated, i.e. hysterical?

(5) What degree of disability do psychoneurotic symptoms produce?

Procedure.—Three groups of 30 cases showing psychiatric disorders were investigated by questionnaire and, where possible, reference to documents, as follows:

(1) Soldiers developing psychiatric disorders who had not been in action. (2) Service men and civilians subjected to enemy action, but not subjected to blast. (3) Cases subject to blast, but where there was no evidence of gross head injury. They were chosen as far as possible to conform with what Symonds (1940) calls slight general cerebral confusion, i.e. where the unconsciousness does not exceed five minutes, and there is no gross evidence of injury.

The cases in group 1 exhibited symptoms for a variety of periods from months to years, depending on when their M.O.s referred them on. In groups 2 and 3, from 6 to 15 months had elapsed since the time of the trauma. (Acute cases were not included in this series.) Cases of definite head injury, e.g. fractured skull, or where there was evidence of prolonged unconsciousness, were excluded, as irrelevant to this inquiry.

Results.—Table I shows the frequency of occurrence of symptoms generally recognized as psychoneurotic, i.e. expressions of anxiety. Insomnia and irritability might well go

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states; one strongly coloured by depression, and one schizophrenia. The latter was especially interesting, as there seemed to be definite evidence that his condition originated on the crossing from Dunkirk, but there was no definite evidence of head injury. Reports confirmed by his mother showed that before this he was perfectly normal.

Headaches.—Brend (1941) points out that headache is the presenting feature in organic cases, an observation with which I would agree, although noticeably in my series it occurred in about half the cases in all groups. As the patients were asked if they had headaches their number not unnaturally looms large, but they were not necessarily severe or complained of spontaneously.

Hysteria.—There is a not uncommon assumption that pension or Workmen's Compensation cases are generally purely gold-diggers. It is worth while, therefore, drawing attention to (a) the small number of hysterics in this series of cases, which appears to be in conformity with general findings in this war to date,¹ and (b) to the fact that their reactions are often, not a desire to make something out of the war, *per se*, but the result of a very real anxiety and worry. For example a small shopkeeper had his shop and business destroyed, and a seamstress had part of her house blown away and her clients evacuated from the district, leaving her workless; both had an anxiety hysteria the motivation of which was easy to understand.

DISCUSSION

Before considering the role trauma plays, it is useful to see how far constitutional factors are a sufficient explanation in themselves.

Constitution obviously plays a part in any disorder, but the degree to which it is brought in is usually directly proportional to the prevailing ignorance of the pathology of the condition; hence its frequent appearance in psychiatry. But to invoke it as an explanation of a war neurosis, without any evidence of past personality disorder, is to argue *post hoc ergo propter hoc*. Therefore it is significant to note that out of 60 cases, selecting only those which strictly conformed to the standards laid down, as many as 16, or approximately a quarter of those whose disorders developed subsequent to enemy action, appeared to have had previously normal and stable personalities. In such cases it is quite unjustifiable to dismiss the precipitating factors as of no importance compared to constitution, yet this is often done.²

Commotional factors.—As the anxiety symptoms in Table I are so evenly distributed throughout the groups, it is not possible to argue from this inquiry that cerebral contusion plays a causative part in producing them. The fact that the symptoms of "minor contusion" feature equally in all groups in Table II confirms their ambiguity as diagnostic criteria in themselves. On the other hand, it does not exclude the fact that some of them may be organically determined, and that in a given case a mixture of psycho- and physiogenic symptoms may be present. A clinical as opposed to a numerical analysis would appear to substantiate this, as exemplified by the following case.

Mrs. S., a widowed chiropodist of 67, was blown through a doorway by blast. She had no memory of the explosion, but remembered picking herself up, and feeling very dazed. The next day she had a headache, and felt extremely shaky. These symptoms persisted for seven months, and she was unable to work because her hand was too unsteady. On examination she was very anxious and emotional. This was quite unlike her former self, she declared. She had unequal pupils, which reacted normally, and commotio retinæ. Her right ankle-jerk was diminished and there was a coarse tremor of the right hand. W.R., C.S.F., and X-ray of the skull were normal. After two months in hospital only the commotio retinæ and absent ankle-jerks persisted. Her headaches had almost disappeared. She was far less emotional, but her tremor, which was obviously hysterical, persisted. It was probably a continuation of a tremor originally due to fright. Its motivation was clear. Her business, actual and potential, had been entirely destroyed by bombing, and her confidence in her ability in wielding a chiropodist's knife had gone, and at 67, with no capital, she had nothing but public assistance to look forward to.

The case illustrates the coexistence of three parallel disorders, viz.: (1) Anxiety symptoms presumably due to fright. (2) A hysterical perpetuation of these arising out of her precarious financial position. (3) Headache, vomiting, emotional lability, and commotio retinæ, the outcome of physical trauma. The signs of the latter had been masked by the patient's own pre-occupation with psychogenic symptoms.

The Role of Commotion in the Possible Production of Anxiety Symptoms

The clinical study of certain cases in the series still leaves a doubt as to whether commotion can so readily be completely excluded, as nowadays there is a tendency to do. The occurrence of similar symptoms irrespective of cerebral trauma suggests fear as the common factor causing the condition, but may not commotional disturbance interfere with their subsidence? Following head injury or cerebral arterial disease patients may be emotionally labile, presumably the inhibiting functions of the cortex

¹ I have differentiated between the patient all of whose symptoms were hysterical, and those cases where, for example, there is an exaggeration of a solitary symptom at examination, e.g. a tremor.

² Curran and Mallinson (1940) have shown that unpredictable cases of breakdown often have a positive family history, but unfortunately I had no reliable records of these.

having been interfered with. Might not some similar interference with an inhibiting mechanism prevent the normal resolution of anxiety symptoms?

The following are examples of case-histories where such queries appear to arise:

A stolid railway-guard of 63, with forty years' excellent service, who had never been nervous in any way, was blown from his guard's van. He lay dazed on the permanent way, picked himself up to fight a fire in his train, was blasted into this, but luckily escaped quickly enough to avoid burns, ran to a signal box only to have it blown all around him—again without apparent physical injury. Six months after he still had recurrent nightmares of his experiences and was intensely nervous of raids. He was then discovered to have commotio retinae, showing that he had suffered physical as well as psychic trauma.

The other cases were merchant sailors of over 60. Two were at Zeebrugge without nervous sequelae in the last war. The third said this was his fourth war. None had previously known what it meant to be nervous. All three had become intensely nervous after their ships were bombed for the first time, in this war, and remained so.

In such cases, how far is cerebral arteriosclerosis a factor? None of the cases had objective signs of this, which of course does not exclude it. In none of these cases can I produce evidence that physiological changes caused the symptoms, nevertheless I wonder whether the persistence of these symptoms can be attributed entirely to psychogenesis, even though they were caused by this? Then the immediate question arises: Is it right that claims for an injury allowance should depend on the examiner's views on pathology? Would it not be more satisfactory to inquire not whether the symptoms are merely caused by fear and apprehension, but to decide instead whether they are being perpetuated by psychological causes? This would still leave the examiner latitude in deciding whether enemy activity had produced or materially aggravated a condition, but would get away from the anomaly of having to reject claims wherever there was no apparent physiological injury, and yet severe psychic trauma existed. (For example: A woman patient of mine was trapped for seventy-two hours beneath a bed. She was physically unhurt but she could hear the screams of her four children whom she could not reach, three of whom died before they could be rescued.)

The undesirability of paying pensions for functional disorders is axiomatic, and provision has been made for free in-patient treatment for these cases instead, but unless they are discharged as completely fit there is still the problem that during the continuation of the war there will be many vulnerable areas in which they are unable to work. They are therefore placed at a disadvantage compared with the average man in the labour market. As these facts are accepted as grounds for paying an allowance, I contend there is a case for granting a partial injury allowance for these with a genuine "conditioned fear" state—an allowance which would naturally cease with the termination of hostilities.

SUMMARY AND CONCLUSIONS

- Three groups of 30 cases are compared, viz. (1) Soldiers not exposed to enemy action, (2) soldiers and civilians exposed to immediate enemy action, but who were unscathed, (3) cases who had suffered slight cerebral commotion but no physical injury.

The evidence does not suggest a physiogenic cause for the psychoneurosis, but it is argued that physiological causes may prevent the subsidence of anxiety symptoms.

Organic post-concussive symptoms may co-exist with, and be masked by, psychogenic ones where the latter are prominent.

In view of the frequent difficulty of determining the psycho- or physiogenesis of symptoms the desirability of assessing purely on the degree of disability irrespective of the cause is contended.

Some war neurosis may be the cause of genuine partial physical disablement and a case for injury allowances for the duration of the war is stated.

Where no positive evidence of previous personality disorder exists, it is not justifiable to dismiss the precipitating factors in favour of constitutional ones.

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Section of Therapeutics and Pharmacology

President—R. D. LAWRENCE, M.D.

[April 21, 1942]

DISCUSSION ON TRINITROTOLUENE POISONING

Dr. J. C. Bridge: The earliest cases of toxic jaundice before the last war were attributed to dinitrobenzol. Then jaundice, associated with liver damage, was noted in 1914-16 (some 70 cases) among those employed in doping aeroplane parts with a solution containing tetrachlorethane [Willcox, W. H., Spilsbury, B. H., and Legge, T. M. (1915) *Trans. Med. Soc. Lond.*, 38, 129]. Then came similar cases of liver damage from T.N.T., which continued until the end of the war in 1918. A few cases occurred after the cessation of hostilities, when chlorinated naphthalene appeared as another cause of liver damage, and produced the same pathological changes as T.N.T. Why the chlorination of the non-toxic naphthalene, not its nitration, should produce the same pathological effects as the nitration of toluol is an unsolved problem.

It is impossible to give the incidence of cases to the number of workers exposed to risk, but it is probably fair and safe to say that the number of persons employed and exposed to risk has not been less, and has increased at a no less equal rate from 1939 in comparison with 1914. If that is true, then the incidence is not greater than in the previous similar war period. The exposure to risk before the onset of symptoms, has varied from three weeks to six months among females and from three weeks to ten months among males. The average age of the 40 females affected since 1939 has been 30 years (19-49 years) and that of the 28 males in the same period 42½ years. The interesting lag period between the cessation of contact and the onset of jaundice has again been noted; seventeen cases showing varying lag periods up to thirteen and a half weeks. Of the 475 cases between 1916 (when statutory notification of toxic jaundice came into force) and the end of 1941, 125 or 26·3% have ended fatally, the male death-rate being 21·9% and the female death-rate 28·4%.

The occurrence of anæmia of the aplastic type is also of interest. Up to February of this year we have had knowledge of nine cases of anæmia amongst T.N.T. workers, three of which were associated with jaundice. On March 1 of this year toxic anæmia was made notifiable under the Factories Act.

Furthermore, we must not lose sight of the fact that in addition to known toxic liver damage, cases of liver damage occur in industry without evidence of contact with any chemical substance. Such cases should be studied carefully, as they may be of value in analysing the factors determining liver damage in known toxic cases.

In 1917, Moore showed to our then satisfaction, that the skin was the main channel of absorption, and Legge, at the end of the last war, anticipated little risk in T.N.T. poisoning in the future, because he, optimistically, visualized the elimination of hand contact. The T.N.T. is eliminated in the urine and its presence can be shown by Webster's test, but this unfortunately gives no indication of liver damage, but only indicates contact. Early evidence (from a simple test) of such damage would be invaluable.

Finally, as regards the prevention of illness from T.N.T., I would summarize this as cleanliness of the air breathed, secured by effective ventilation or, failing the practicability of that, filtration through an effective respirator, cleanliness of the implements used and the cleanliness of the person, secured by protective clothing and by personal attention to the care of the skin.

Dr. Catherine Swanston: *Incidence of T.N.T. symptoms in a filling factory* (Abridged).—Pure T.N.T. is used either molten, powdered, or as a biscuit. It is also

being mixed with other substances, such as ammonium nitrate in amatol and with barium nitrate in baratol and many other mixtures. Contact hazard may therefore exist, either from T.N.T. dust or fumes, according to the process under consideration.

Dust hazard is met with in the preparation of amatol and baratol, aerial bomb and trench mortar filling, and in the breaking of biscuit. This last process we considered as one of the most dangerous processes in the factory. Women were kept out of it as far as possible, and we attempted, not too successfully, a rota system, limiting the time spent in this process by the workers. Later, coloured men worked on this job and whatever the reason, we have never had a case of dermatitis or T.N.T. illness on one of these workers. They appear to stand up to it amazingly well.

Fume hazard occurs where T.N.T. is melted and the temperature of the shops is always above the optimal. It also occurs where shells and anti-tank mines are filled.

In all the above processes there is, to a greater or lesser extent, direct skin contact with solid T.N.T.—spilled on the benches, allowed to cool, handled as biscuit, adhering to stemming rods, pouring cans, funnels, &c. Also the cleaning of these tools and shops themselves, unless done properly and under supervision, very often entails a higher contact than the filling itself.

T.N.T. is mainly absorbed through the skin, but working as at present, in ill-ventilated shops with dust and fumes constantly present in the air, a certain amount must be inspired into the lungs and ingested into the alimentary tract. Nevertheless, the skin is the most important absorbing surface in considering suitable protective clothing and safe methods of working.

In one factory about 2,000 persons are employed on T.N.T. work, three-quarters of them women. The ages of the women vary from 20 to 50 years, the men up to 60. Very few have done this work before. The general physical standard is low, especially among the men. Despite preliminary medical examinations, our standard must to a large extent depend on the state of the labour market.

The following are some of the conditions considered as an absolute bar to employment in T.N.T.: a history of jaundice or gall-stones, severe or chronic gastric illness, including gastric and duodenal ulcers, multiple or severe abdominal or pelvic operations, nephritis or nephrectomy, tuberculosis in any form, moderate or severe anemia, chronic chest complaint, chronic skin lesions, or previous trade dermatitis, rheumatic fever and Graves' disease.

These workers are inspected twice in three weeks by the medical officer, on two of every three shifts. The shops are also visited, and the workers watched while on their jobs. Inspection of night shifts is valueless, cyanosis and early jaundice cannot be seen in artificial light.

The T.N.T. section was the first to go into production and labour was drafted there rapidly from the beginning. As a result, twelve to eighteen months ago, there was a large unsalted population in these shops, and this may partly explain the many cases of T.N.T. poisoning of that period. During the twelve months ending 31.12.41, we notified 495 cases of minor T.N.T. illness, including dermatitis, 15 cases of serious poisoning, comprising 12 of toxic jaundice and 3 of aplastic anemia, one of whom had previously had an attack of toxic jaundice, from which she had apparently recovered. All our aplastic cases died, and three of our jaundice cases, i.e. 6 deaths, a 40% mortality. The sex incidence was 1 male to 14 females. The only male case had been employed exclusively for two months in sieving T.N.T. by hand.

The minor cases can be summarized as follows:

| | | Male | Female |
|----------------|--------|------|--------|
| (1) Anilism | | 49 | 132 |
| (2) Gastritis | | 24 | 83 |
| (3) Dermatitis | | 92 | 115 |
| | Totals | 165 | 330 |

These are appalling figures, representing 25% of the present strength, but I cannot give the true percentage incidence, as I have been unable to get accurate figures of the monthly increase in the T.N.T. population in 1941.

These cases arose in every part of the factory, and it is impossible to inculcate specifically any one process, but one of the most dangerous processes for the women is the anti-tank mine filling, where both solid and liquid T.N.T. is used. Ten of our serious cases in these shops, 4 had worked exclusively on the job and of the 4, 3 died. The girls became very deeply stained and "filling mines" occurs more frequently else in our case-histories. Among the men the filling of aerial bombs caused a great deal of minor illness, and many cases of dermatitis.

In both these jobs contact is very heavy and, at that time as well, workers were inadequately supplied with the proper tools and ventilation was non-existent.

All these cases were removed from T.N.T. work, as soon as they were spotted, a few permanently, but the majority were allowed to return after a spell of non-contact varying from a few weeks to several months. In some of these, symptoms recurred, and they were taken off for good. In such cases Webster's test is of little value; all it means when positive is that T.N.T. is being absorbed and excreted and until we have an accurate simple test to show damage to liver or bone marrow, the moving of workers out and back to contact will remain a hit and miss affair.

Of the 495 cases notified, only a few were ill enough to be off work altogether. These were:

| | | | | | | | Male | Female |
|----------------|-----|-----|-----|-----|-----|--------|------|--------|
| (1) Anilism | ... | ... | ... | ... | ... | ... | 6 | 5 |
| (2) Dermatitis | ... | ... | ... | ... | ... | ... | 10 | 9 |
| (3) Gastritis | ... | ... | ... | ... | ... | ... | 1 | 2 |
| | | | | | | Totals | 17 | 16 |

None of these cases was allowed to return to T.N.T. work. We felt that in these cases, except perhaps in those with dermatitis, there was a possibility of a higher degree of liver involvement and we could not risk further damage.

Dermatitis has been rather overlooked in the consideration of T.N.T. illness, but the average time lost by men and women in my series, was forty-eight and thirty-nine days respectively, a serious loss to production. Nineteen of the above 33 workers drawing compensation for minor T.N.T. illness, had dermatitis. Only very occasionally does one find a patient who has both a dermatitis and toxic symptoms together. I have only seen one case of jaundice with a confirmed T.N.T. rash.

The conditions under which these cases occurred were such as to facilitate the development of the pathological changes described. The shops were badly ventilated, and during black-out ventilation was non-existent. Later, lean-to's were built along one side of the units, open at each end, so that one door in every shop could be open always. Ventilation is, however, still not good, and there is a complete lack of local exhaust draughts to remove dust. Labour was raw, and rapid increase in production rendered inadequate the available supply of trained workers. But training shops are now being installed, and to these all new workers are drafted for seven to ten days. Here they work under constant supervision with adequate tools and following definite rules. Production in these shops has been known to beat that obtained in the shops where the old hands work.

Difficulties were accentuated by the lack of trained supervisors to appreciate the dangers of this work and to insist on clean methods of filling, and by the lack of proper tools for the different jobs. The inadequate supply of protective clothing was another important detrimental factor. Workers were forced to wear the same garment for weeks or even months after it had been soiled with T.N.T. This combined with inadequate ablution facilities, inadequate cleaning tools, and the lack of any proper system of cleaning benches and workshops, formed an unfavourable background for any reasonable regimen of cleanliness at work.

Improvements have occurred and are occurring. The above conditions are getting better and it is possible that now we have developed a salted population. Medical supervision has improved. In June 1941 a Minor Surgery was opened on the T.N.T. Section, staffed by trained nurses, with previous experience of T.N.T. illness, drafted from the Central Surgery. These nurses stick to their own shifts, and are encouraged to go through the shops, and they assist the medical officer at the routine inspection of workers. They do valuable work and have become skilled at assessing T.N.T. illness. Dealing with a small number of people they can exert a thorough and continuous supervision over their own shifts. They have been well received by the management, and by spotting early cases they have reduced the amount of illness. They have power to remove workers from contact, but not to put them back.

Much still remains to be done. Conditions in the shops can still be improved by more ventilation, local dust extraction, better tools and increased methods of machine filling. This last would reduce not only actual contact, but also the number of individuals exposed to risk and would increase production. Ablution facilities still require extending, and the supply of protective clothing could be improved. Further, a stricter supervision of the workers by people who are sufficiently intelligent and interested to appreciate the dangers of the work and the methods by which such can be avoided, is needed. Workers and supervisors ought to be trained to the job, and penalized (especially the latter) if rules are not obeyed.

Dr. Ronald E. Lane: *Clinical aspect* (Abbreviated).—The clinical manifestations of T.N.T. poisoning may be summarized as follows: Dermatitis, Anilism, "Gastritis", Toxic Jaundice and Toxic Anæmia. The first three of these are comparatively frequently seen: Toxic jaundice is a serious and infrequent occurrence (0.2% in the last war), and toxic anæmia is an even rarer event.

Dermatitis.—None of the cases which I saw suffering from the other forms of T.N.T. poisoning had ever had any T.N.T. dermatitis.

Anilism.—A great number of those in contact show minor and varying degrees of cyanosis. This is quite symptomless and is the result of T.N.T. absorption in these otherwise healthy-looking individuals. There is little or no pallor, and probably the cyanosis is due to a minor quantity of methæmoglobin in the blood. The more serious cases present the definite T.N.T. facies so well described recently by Roberts (1941), pallor, with a lilac coloration of the lips, and lobes and tips of the ears. It is lessened by excitement or mild exercise, and for this reason is best detected by the medical officer when in the departments during work rather than on any set medical parade. It seems probable that these changes are mainly vasomotor in origin. These cases in the early stages are usually quite symptomless. Later, in some cases, symptoms may develop, e.g. breathlessness, retrosternal tightness, with more or less malaise.

T.N.T. "Gastritis".—In this category three different types of case have been seen, and their differential diagnoses are of practical importance.

(1) *T.N.T. "Gastric illness"*: Here the patient complains of a dull ache in the epigastrium, unrelated to food and relieved by rest. These are the important cases which must be differentiated from the other groups, though this may occasionally be difficult.

(2) *Anilism with retrosternal pressure*—sometimes described by the patient as "indigestion".

(3) *The group of chronic dyspeptics*, whose symptoms may be aggravated by the long hours without food, difficult journeys, and "shift" arrangements.

The true T.N.T. case usually gives no previous history of digestive trouble. He looks ill and very wretched and usually has the T.N.T. facies with marked pallor and looks "pinched" about the nose. He is weary, tired and intensely miserable. His loss of appetite is very marked. He is constipated, and his nausea and vomiting may be quite unrelated to food. Further, his heavy epigastric ache is quite unlike anything found in the ordinary dyspepsias. I believe these cases to be of hepatic origin, and therefore I consider their early detection of great importance. In half of my cases falling into this group the liver was enlarged and tender. Investigations disclosed little. Levulose tolerance tests were usually normal. Barium and fractional test meals disclosed no definite departures from normal. Blood examinations showed no abnormality. The urinary coproporphyrin content, however, was definitely raised in over half of the cases in which this estimation was made.

The treatment here, as in any industrial poisoning of this type, must start with the immediate arrest of intake of the poison. This means not only the removal of the patient from contact, but the removal of all T.N.T. from the skin of the patient. For this reason he is best removed immediately from his home surroundings into hospital, where a thorough bath is followed, as recommended by Moore, by the scrubbing of hands, fingers, feet and toes with ether until no pink reaction is obtained with alkaline alcohol. The nails should be closely cut. Bowels should be moved as soon as possible and kept open. A bland simple diet is allowed as soon as it can be tolerated. Fluids are given in large quantities, with as much fresh fruit and vegetable as can be obtained. Fats are kept to a minimum. Ascorbic acid was given in doses of 100 mg. daily in some cases, but I can adduce no evidence that it has affected the course of the disease.

These cases usually clear up so far as their major symptoms are concerned after a few days' rest in bed. In a few of the worst cases it has taken two or three weeks before they have been sufficiently free from symptoms to get up. In nearly all cases, however, the fatigue has hung on, and it is almost always the last to clear. These cases of true T.N.T. gastric illness should never be allowed to return to contact.

Toxic jaundice.—This is fortunately rare. In this series eight of these cases have arisen (five female and three male) with two deaths. This preponderance among the females is well recognized, and is even better demonstrated in a series already partly reported by Evans, 1941, for which details I am indebted to Dr. Swanston.

I know of no explanation for this difference in susceptibility, though it is a well-known finding with other industrial poisons. Is it in any way connected with possible thyroid dysfunction? The few B.M.R. investigations so far carried out in these cases have shown no very striking results. Or is it connected with dietary habits? Work so far published would lead one to believe that liver damage is best withstood on a diet low in fat but

high in protein and carbohydrate. In view of this one might question the advisability of giving extra milk to these workers. Perhaps skim milk might better serve their needs.

Jaundice usually appeared after short exposure—in most cases less than three months. It was usually preceded by symptoms similar to those just described, though in half the cases there was no history of previous cyanosis. There is little remarkable about the clinical findings or investigations. Stools were usually light coloured—urine dark. The liver was palpable at some stage of the illness in most of the cases. In some it was soft and tender in the early stages, and in three cases it became hard and of an indiarubber consistency towards the end of the illness. All cases were apyrexial. Serum bilirubin values fell between 2 and 15 mg.%. Levulose tolerance tests were abnormal in only two cases. One feels the lack of a simple yet comprehensive test of liver function. The colour-index was above 1 in half the cases. The one investigation which showed a definite departure from normal was the urinary copro-porphyrin. In the two fatal cases this figure was some 50 times greater than normal. The estimation was carried out immediately the patient was admitted to hospital some weeks before death and is therefore a striking finding.

Diagnosis: I know of no way in which these cases can be differentiated with certainty from catarrhal jaundice resulting from an infective hepatitis. These latter cases usually show some pyrexia for a week or two, but pyrexia is occasionally reported in the toxic cases. The only course open to us is to regard all "jaundice" among T.N.T. workers as of toxic origin, excepting always, of course, the obvious surgical cases.

Treatment: The treatment of toxic jaundice was on similar lines to that given in toxic gastric illness: admission to hospital, thorough cleansing, saline aperients, fluids and a high carbohydrate diet low in fat with high vitamin B and C intake. Zinc insulin (15 units daily) was given with a view to increasing glycogen storage and possibly producing an increase in glycuronic acid, which is needed in one of the detoxicating mechanisms for dealing with T.N.T. Whether or not any better results have been obtained as a result of this, I am not prepared to say.

Course: Of the six patients who survived in my series four have so far returned to work after an illness of approximately four months. In all cases unusual fatigue is the most persistent symptom. Recovery probably brings some measure of cirrhosis.

Aplastic anaemia.—Three cases of aplastic anaemia due to T.N.T. poisoning have been included in this series. All are in men—one of 41, one 51 and one of 55 years of age. Each case presented a similar picture—a profound normocytic anaemia associated with agranulocytosis and a low platelet count, all blood-forming elements being affected. Although there was no reason to suppose it would be successful, liver and nucleotide therapy was attempted without result. The only measure which met with any success was repeated transfusion. In this way our first case was kept alive for some months until his marrow began to regenerate. It is now some nine months since the beginning of his illness and four months since his last transfusion, since which he has not only retained his red count, but has increased his haemoglobin by 20%. His white count remains in the neighbourhood of 6,000, with a normal differential count, and his platelets have also returned to normal. The patient has still an enlarged liver, and over the last few months his spleen has become palpable. I believe this to be the first case of aplastic anaemia due to T.N.T. to survive. These are desperate cases, but there is hope that in the less acute cases life may be saved by repeated transfusion. The two other cases are still under treatment.

One other case has given rise to considerable interest, particularly in view of the recent work of Bomford and Rhoads, 1941. A woman with pernicious anaemia, who had been heavily exposed to T.N.T., has failed to respond normally to liver therapy, despite the fact that she has been on large doses of an active preparation. Has T.N.T. been responsible for this, and, if so, is there in all these serious T.N.T. cases some conditioning process rendering the individual susceptible to the effects of T.N.T.?

Copro-porphyrin estimations.—From a strictly practical point of view the prevention and care of the minor cases in the factory is more important than the supervision of the more serious type of case. There is so little we can do for these latter that every effort must be made to secure their prevention. One of our chief weapons is medical supervision in the factory. This is no easy task, but it would be greatly lightened if it were possible to evolve an objective test which would enable a closer control to be exercised on those exposed to the T.N.T. hazard.

Some assistance might be gained from urinary copro-porphyrin estimations. In Table I are given results so far obtained. In this work I have been fortunate in securing the assistance of Dr. Kench, who has made all these estimations for me. It will be seen that there is a considerable rise in most of the T.N.T. cases as one would expect from the

TABLE I.—URINARY COPRO-PORPHYRIN PER LITRE

| | Sex | Age μ g per 24 hours | |
|----------------|-----|--------------------------|--------|
| Gastritis | F. | 47 | 84 |
| | F. | 36 | 33 |
| | M. | 41 | 93 |
| | M. | 49 | 61 |
| | F. | 55 | 153 |
| | F. | 37 | 31 |
| | M. | 38 | 130 |
| | M. | 54 | 234 |
| Anilism | M. | 58 | 50 |
| | F. | 65 | 202 |
| Toxic jaundice | F. | 50 | 84 |
| | M. | 62 | 1,904* |
| | M. | 28 | 212 |
| | F. | 42 | 1,376* |
| | F. | 24 | 150 |
| | F. | 24 | 320 |

* Death.

work of Rimington and Goldblatt, 1940. The series is certainly far too small, but the findings in the fatal cases are arresting.

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Professor T. B. Davie (George Holt Professor of Pathology, University of Liverpool): *The pathology of T.N.T. poisoning.*—The lesions associated with the action of T.N.T. may be classified as follows: (A) Direct effects on the blood: (1) Reduction of O_2 -carrying capacity by formation of some methæmoglobin and NO-hæmoglobin. (2) Hyperplasia of marrow (compensatory to above). (B) Local toxic effects: (1) Skin—dermatitis. (2) Mouth and pharynx—œdema glottidis. (3) Gastric mucosa—catarrhal gastritis. (4) Subcutaneous—necrosis (experimental). (C) Severer systemic effects: (1) Liver—toxic necrosis. (2) Marrow—aplastic anemia. (3) Vascular endothelium—acute toxic purpura. (4) Other organs, e.g. kidneys—relatively mild degenerative changes.

Direct effects on the blood.—The clinical evidences of anilism have always been the first obvious signs of absorption of T.N.T. Benjamin Moore (1917) made much of the oxygen lack resulting from the replacement of some of the oxyhæmoglobin by methæmoglobin and nitro-hæmoglobin. Pantón (1917) in his earliest investigations on these cyanosed patients and again in his M.R.C. Report (1921), drew attention to a mild reactive leucocytosis, affecting mainly the neutrophil polymorphonuclears, but stressed the absence of anemia in the cases of anilism. His records show R.B.C. counts which to-day would be regarded as somewhat above normal. The only reference to reticulocyte counts is to be found in the post-war report on the American investigations by Minot (1919). He found them increased in the few cases examined, and incidentally also observed the presence of polychromatic red cells in 83% of a series of 233 workers.

We have been able to confirm this stimulant effect on the marrow in an investigation on the reticulocyte counts of small groups of new workers and old hands in the ordnance factory.

One of these groups comprised eight new workers, the average of whose reticulocytes before starting work was 5.9 per 1,000 red blood cells. During their first month of contact work counts taken at ten-day intervals showed increases in every case, the average count at the end of the month being more than doubled, to 12.2 per 1,000 red blood cells. This effect was also shown in another series consisting of workers who had been in contact operations for periods ranging from eight to fifteen months without developing symptoms necessitating their removal from contact work. The reticulocyte count of these eight varied from 5 to 26 per 1,000 red blood cells with an average of 13. This figure though within the normal range is probably about double the normal average.

Local irritative effects.—Among the local toxic effects of T.N.T. seen in our ordnance factories, the two commoner manifestations present little of pathological interest. The dermatitis is usually papular; the other common lesion, gastritis, is presumed to be associated with a catarrh of the gastric mucous membrane. Gastroscopy has not yet, to my knowledge, been employed to elucidate the pathology of this condition.

The serious complication of œdema glottidis was reported on a few occasions during the last war, but thus far it has not been reported in the factories in our area.

Severe systemic effects.—It is, however, with the grave systemic manifestations of T.N.T. poisoning that we are primarily concerned. The features of these toxic onslaughts were described in detail and with great accuracy by Stewart, Turnbull, Pantón and others

in the discussion arranged by this Society and reported in its *Proceedings* in 1917, 10. page 1.

Between 1921 and 1940 no publication of note on this subject has appeared. Recent articles by Hilton and Swanston (1941), Roberts (1941) and Evans (1941) have indicated that the cases occurring at present are of the same type as those of 1916-18. Now, as in the last war, the majority of the fatalities succumb to acute liver necrosis (or acute yellow atrophy), a small proportion die of an aplastic anaemia, and an occasional one manifests the features of an acute toxic purpura.

Toxic jaundice.—The changes in the liver are those of widespread necrosis which may, in the course of a few days, reduce the liver to half or even a third of its normal weight. The hepatic cells not destroyed are deeply bile stained and show up as yellow patches in a dull red field, particularly in the subacute cases. The red areas are those where necrosis has been complete and the red blood corpuscles from the disrupted sinusoids mingle with fatty fragments of liver cells and necrotic debris. In the subacute cases the necrotic areas are not so extensive and show ingrowths of fibroblasts and new capillary loops into the necrosed tissue. This highly vascular tissue is often bright red in colour and contrasts sharply with the jaundiced remains of liver parenchyma. Among the surviving liver cells are to be seen all grades of degenerative change. This is interpreted as indicating that the noxious agent is continuously at work. In all but the most acute cases there is evidence of chronic inflammatory cellular (lymphocytic) reaction and of regenerative proliferation of both bile duct epithelium and hepatic cells. The hyperplasia of the bile ducts leads to their appearance in great numbers in the necrotic areas, while that of the hepatic cells is seen first in the appearance of large active cells at the periphery of the surviving liver cells and later by marked nodular masses of liver cells with poorly demarcated lobular pattern. This nodular overgrowth is accentuated by the contraction of the fibrous tissue which eventually replaces the necrosed tissue. The outcome is the condition known as multiple nodular hyperplasia, seen to best advantage in those cases dying of aplastic anaemia, or other cause, some months after recovery from an attack of toxic jaundice.

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TABLE

| Name | Sex | Age | Cause of death | Liver appearance | Wt. g. | Marrow | Petechiae |
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| A. B. | F. | 21 | Aplastic anæm. | Subac. necr. Jaundiced | 1250 | Aplastic | ++ |
| R. B. | F. | 23 | Acute toxic purp. | Early necr. | 1500 | Hyperplastic | +++ |
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| M. D. | F. | 32 | Toxic jaundice | Acute necrosis | 530 | Hyperplastic | ++ |
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TABLE I.—URINARY COPRO-PORPHYRIN PER LITRE

| | Sex | Age | µg per 24 hours |
|-----------------------|-----|-----|-----------------|
| <i>Gastritis</i> | F. | 47 | 84 |
| | F. | 36 | 33 |
| | M. | 41 | 93 |
| | M. | 49 | 61 |
| | F. | 55 | 153 |
| | F. | 37 | 31 |
| | M. | 38 | 130 |
| | M. | 54 | 234 |
| | M. | 58 | 50 |
| <i>Anilism</i> | M. | 65 | 202 |
| | F. | 50 | 84 |
| <i>Toxic jaundice</i> | M. | 62 | 1,904* |
| | M. | 28 | 212 |
| | F. | 42 | 1,375* |
| | F. | 24 | 150 |
| | F. | 24 | 320 |

* Death.

work of Rimington and Goldblatt, 1940. The series is certainly far too small, but the findings in the fatal cases are arresting.

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Professor T. B. Davie (George Holt Professor of Pathology, University of Liverpool): *The pathology of T.N.T. poisoning.*—The lesions associated with the action of T.N.T. may be classified as follows: (A) Direct effects on the blood: (1) Reduction of O₂-carrying capacity by formation of some methæmoglobin and NO-hæmoglobin. (2) Hyperplasia of marrow (compensatory to above). (B) Local toxic effects: (1) Skin—dermatitis. (2) Mouth and pharynx—œdema glottidis. (3) Gastric mucosa—catarrhal gastritis. (4) Subcutaneous—necrosis (experimental). (C) Severer systemic effects: (1) Liver—toxic necrosis. (2) Marrow—aplastic anaemia. (3) Vascular endothelium—acute toxic purpura. (4) Other organs, e.g. kidneys—relatively mild degenerative changes.

Direct effects on the blood.—The clinical evidences of *anilism* have always been the first obvious signs of absorption of T.N.T. Benjamin Moore (1917) made much of the oxygen lack resulting from the replacement of some of the oxyhæmoglobin by methæmoglobin and nitro-hæmoglobin. Pantón (1917) in his earliest investigations on these cyanosed patients and again in his M.R.C. Report (1921), drew attention to a mild reactive leucocytosis, affecting mainly the neutrophil polymorphonuclears, but stressed the absence of anaemia in the cases of anilism. His records show R.B.C. counts which to-day would be regarded as somewhat above normal. The only reference to reticulocyte counts is to be found in the post-war report on the American investigations by Minot (1919). He found them increased in the few cases examined, and incidentally also observed the presence of polychromatic red cells in 83% of a series of 233 workers.

We have been able to confirm this stimulant effect on the marrow in an investigation on the reticulocyte counts of small groups of new workers and old hands in the ordnance factory.

One of these groups comprised eight new workers, the average of whose reticulocytes before starting work was 5.9 per 1,000 red blood cells. During their first month of contact work counts taken at ten-day intervals showed increases in every case, the average count at the end of the month being more than doubled, to 12.2 per 1,000 red blood cells. This effect was also shown in another series consisting of workers who had been in contact operations for periods ranging from eight to fifteen months without developing symptoms necessitating their removal from contact work. The reticulocyte count of these eight varied from 5 to 26 per 1,000 red blood cells with an average of 13. This figure though within the normal range is probably about double the normal average.

Local irritative effects.—Among the local toxic effects of T.N.T. seen in our ordnance factories, the two commoner manifestations present little of pathological interest. The dermatitis is usually papular; the other common lesion, gastritis, is presumed to be associated with a catarrh of the gastric mucous membrane. Gastroscopy has not yet, to my knowledge, been employed to elucidate the pathology of this condition.

The serious complication of œdema glottidis was reported on a few occasions during the last war, but thus far it has not been reported in the factories in our area.

Severe systemic effects.—It is, however, with the grave systemic manifestations of T.N.T. poisoning that we are primarily concerned. The features of these toxic onslaughts were described in detail and with great accuracy by Stewart, Turnbull, Pantón and others

in the discussion arranged by this Society and reported in its *Proceedings* in 1917, 10. page 1.

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present, apart from some lymphocytes, were hæmocyto blasts. As this particular case had recovered, some four months earlier, from a severe attack of toxic jaundice, and as at post-mortem the liver showed the result of this in the form of a multiple nodular hyperplasia, Evans (1941) who reported the case was led to suggest that the earlier damage to the liver may have repressed the formation or the storage of some maturation principle necessary for normal hæmopoiesis.

A third case (M. S.) of aplastic anæmia was admitted to another hospital in January of this year. Although her red cell count had been as low as 730,000 per c.mm. this woman is still alive, largely, we believe, as the result of repeated transfusions, of whole blood at first, and later of concentrated suspensions of R.B.C. In her case the aplasia affected mainly the erythropoietic tissues and her reticulocyte counts had remained at zero for some time, though latterly there had been some response. Her white cell count was at first slightly below normal limits but latterly has been maintained at an average of about 6,000 per c.mm. She cannot at present be regarded as fully cured, but there appears to be at least a chance that she may recover.

Acute toxic purpura.—The last case in my series (R. B.) died at home without any hæmatological or other examinations prior to death. At post-mortem she presented the features of purpura hæmorrhagica except that there was no splenomegaly. There was certainly no apparent anæmia and to naked-eye examination her liver appeared normal. Microscopically however the liver showed very early acute necrosis. Her marrow was markedly hyperplastic, the femur containing red marrow throughout the upper three-quarters of its length, and microscopically all the elements of the marrow were represented.

DISCUSSION

From a study of these seven cases it appears that in all of them the liver, the marrow and the vascular endothelium are affected simultaneously by the toxic action of T.N.T., though in different degrees. For reasons which are not clear the attack in some cases destroys the liver and, though affecting the marrow to the extent of calling forth an erythroblastic response, produces little or no anæmia. The vascular endothelium in these is damaged sufficiently to produce a variable number of petechiæ. In other cases the toxic effect falls most heavily on the marrow, usually repressing both the erythropoietic and the leucopoietic elements, but sometimes only the former. In these the liver is damaged even to the extent of a subacute necrosis with accompanying jaundice, and the vascular endothelium almost inevitably suffers both by direct toxic action and by the anæmic anoxia. In still others, and these are the rarest, the brunt of the attack is borne by the vascular endothelium. These develop an acute toxic purpura, but their marrow and liver are both affected to some extent.

It seems unlikely that the apparently selective effect on these different organs and tissues is due to differences in the type of the noxious agent. To dismiss the subject by assuming an inherent susceptibility of particular tissues to the toxic action of T.N.T. in different patients is to close the door to further inquiry. Acquired differences in tissue susceptibility is a more acceptable explanation, but as yet there are few if any clues as to the factors determining these variations.

FUTURE RESEARCH

Further inquiry into the problem of T.N.T. poisoning is undoubtedly called for. It is probably no exaggeration to say that in this war insufficient attention has been paid to the lessons learnt by bitter experience in our ordnance factories in the last war; but even when we are assured that all that was learnt in the last war is being applied fully and wisely, there is still obvious and urgent need for immediate and extensive investigation. Team work of whole-time investigators at or near the actual ordnance factories offers the greatest hope of successful research. These investigators should further have the benefit of the assured assistance of both medical and non-medical specialists, particularly physicists and biochemists, but the direction of the research should, I believe, be in the hands of a clinician. Part-time or financially starved research at this stage would be a disgrace to the country and a waste of effort on the part of the majority of those undertaking it.

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Section of Radiology

President—M. H. JURE, D.M.R.E.

[March 20, 1942]

DISCUSSION ON THE PLACE OF RADIOTHERAPY IN THE TREATMENT OF THYROTOXICOSIS

Dr. Laurence Martin: I have had the opportunity, while studying thyrotoxicosis in general, of following up a series of cases treated by X-rays at Addenbrooke's Hospital, Cambridge, by Dr. Ff. Roberts. The full results of this follow-up study have recently been published (Martin, 1942) and I shall only refer to some of the important points.

Reports from radiotherapists on X-ray therapy for thyrotoxicosis have tended to be over-enthusiastic, very technical, and often lacking in sufficient clinical details of the cases treated; surgeons have sometimes condemned X-ray therapy on insufficient grounds and over-emphasized the risk of scarring in the neck, while physicians—bewildered by the conflicting opinions—have fought shy of the treatment because they were not told exactly which types of thyrotoxicosis responded well, and which were unsuitable.

It seemed essential, therefore, for any follow-up study to aim at a precise description of the cases treated and a full discussion of the results obtained in the various types of thyrotoxicosis. In this way alone could the value and limitations of X-ray therapy be assessed. I was able to see 40 cases treated by Dr. Roberts in the years 1927-1938 inclusive, and two treated previously by Dr. Shillington Scales. 32 were females, 10 males, and the average age at time of treatment was 35 years, with extremes of 73 and 16 years. The follow-up interval varied from a maximum of fourteen years to a minimum of three years. The series consisted of the following types of case: Primary mild 5, primary moderate 22, primary severe 4, secondary mild 1, secondary moderate 1, secondary severe 5, and non-toxic goitre 4. The following grading of results was used at the follow-up:

Grade I.—Full ability to resume normal life or work without remaining symptoms.

Grade Ia.—As for Grade I but with some disability such as hypertension, diabetes or hypothyroidism not causing restriction of life or work.

Grade II.—Improved—but with some restriction of work or activity.

Grade III.—Invalidism.

The results obtained were as follows:

GRADE AT FOLLOW-UP.

| Type | I | Ia | II | III | Dead | Total |
|-------------------------|----|----|----|-----|------|-------|
| Primary, mild ... | 1 | 2 | 2 | 0 | 0 | 5 |
| Primary, moderate ... | 8 | 5 | 7 | 0 | 2 | 22 |
| Primary, severe ... | 3 | 0 | 1 | 0 | 0 | 4 |
| Secondary, mild ... | 0 | 0 | 1 | 0 | 0 | 1 |
| Secondary, moderate ... | 0 | 1 | 0 | 0 | 0 | 1 |
| Secondary, severe ... | 0 | 0 | 0 | 1 | 4 | 5 |
| Total (toxic cases) ... | 12 | 8 | 11 | 1 | 6 | 38 |
| Non-toxic goitre ... | 0 | 0 | 4 | 0 | 0 | 4 |
| Total (all cases) ... | 12 | 8 | 15 | 1 | 6 | 42 |

This shows that of 31 cases of primary thyrotoxicosis 19 or 61% were in Grades I or Ia and could be considered as cured of thyrotoxicosis although the 7 cases in Grade Ia had some other disability. 10 cases (32%) were improved but restricted in activity. 2 cases were dead—one of pneumonia a year after treatment and the other of rheumatic carditis, during the course of which she had developed thyrotoxicosis. Of the 7 cases of secondary thyrotoxicosis only one could be considered as cured, and she had hypothyroidism; 4 were dead, one was bedridden and one restricted in life.

The four cases of non-toxic goitre with neuroses or autonomic imbalance were not improved.

These results show clearly that X-ray therapy has no place in the treatment of secondary thyrotoxicosis or toxic nodular goitre. The melancholy results recorded here, when contrasted with the brilliant results of surgery in this type of thyrotoxicosis allow of no other conclusion. Admittedly 5 of the 7 secondary cases were severe and 4 had auricular fibrillation and heart failure, but they did not respond to X-ray therapy and 3 of them died after thyroidectomy undertaken as a desperate measure.

Mild and moderate cases of secondary thyrotoxicosis, however, are prone, untreated, to progress inexorably to thyrotoxic heart failure—although this may be a slow process—and I have been unable to find in the literature any unequivocal statement that X-ray therapy can either cure or prevent thyrotoxic heart failure or auricular fibrillation. Such patients therefore should not be lulled into false security by X-ray therapy when they cannot be guaranteed the same immunity from cardiac damage which is provided by thyroidectomy. Furthermore a patient with a nodular goitre may, at any time, get tracheal compression and urgent dyspnoea from the hæmorrhagic enlargement of a nodule, and there is no evidence that X-rays can protect from such a mechanical disaster. I consider, therefore, that cases of secondary thyrotoxicosis or toxic nodular goitre should be treated by surgery alone. This, in effect, means that the decision whether a case of thyrotoxicosis is primary or secondary is of fundamental importance. The decision may sometimes be clinically difficult or even impossible, and I think it is then better to advise surgery, thus removing any doubts about the future of the patient's heart. Another point is that relief of thyrotoxic symptoms in 19 of 31 (61%) primary cases, and improvement in a further 10 cases show that X-ray therapy can compare favourably with surgical results. There were only 4 severe primary cases, and although three died well and one was improved, the numbers do not justify any dogmatic statement. We know the dire peril in which such patients live—with the constant threat of crisis and cardiac or mental breakdown hanging over them—and the decision for X-rays or surgery must be entirely individual.

In the moderate primary cases—probably the commonest type seen—X-rays removed thyrotoxic symptoms in 13 of 22 cases (59%) and caused improvement in 7 (33%).

Autonomic imbalance.—It is with mild primary cases, borderline cases and cases of pure autonomic imbalance that I do not think X-rays can be expected to do very much when their nature is considered. If thyrotoxicosis be regarded as having two main components—namely thyroid dysfunction grafted on a basic constitutional nervous instability—then the mild and borderline cases are those with minimal thyroid dysfunction and maximal nervous instability, while true cases of autonomic imbalance have no thyroid over-activity at all. An attack on the thyroid by surgery or X-rays can only do the same thing—namely to remove the minimal thyroid component and leave untouched the nervous instability which is causing most of the symptoms.

It is true that some cases of pure autonomic imbalance improve with X-ray therapy, but we cannot exclude this as being the result of suggestion for these patients improve equally with medical treatment and more orthodox psychotherapy. I have no large series of cases of autonomic imbalance to substantiate this view, although it is supported in the literature, but I have reported on a small series (Martin, 1939). There were 13 cases, of whom 2 had been treated by X-rays and one by thyroidectomy without improvement, while 7 of the remaining 10 cases were doing well with psychotherapy and symptomatic medical treatment. I feel that they are best treated at present in this way until the nature of their disability is better understood.

Only 7 cases in this series were treated by both X-rays and surgery—3 of them, already mentioned, had thyroidectomy as a desperate measure with fatal outcome after failure to respond to X-rays. One patient who relapsed after a hemithyroidectomy did well with X-rays and the remaining 3 were really unsuitable, being patients with residual neuroses and anxiety states after operation.

Hypothyroidism developed in 6 (14%) of the 42 cases of this series. In 4 it could be directly attributed to the X-rays as it was associated with keloid scarring of the neck and came on early. These cases, however, were amongst the very earliest treated and no scarring has occurred since adjustments in Dr. Roberts' technique at that time. One patient developed myxoedema some years after the end of treatment and another had a striking phase of myxoedema lasting a year soon after treatment, only to be followed by a relapse of thyrotoxicosis for which he was again treated by X-rays. This last patient was the only one in the series who relapsed after apparent cure.

Progress.—I cannot speak of the progress during treatment of the cases in the follow-up series, but I have observed other patients during that period. Improvement seems generally to occur in the following sequence—decrease in nervousness and sweating, increase in strength and energy, gain in weight and decrease in appetite. The pulse-rate

drops steadily, but still retains its lability on emotion and exertion which is probably a manifestation of the underlying nervous instability and not of thyroid dysfunction. The goitres diminished in size rapidly but exophthalmos, as after thyroidectomy, was very variable in its disappearance and seldom did so completely. The patient's own estimate of improvement has been particularly reliable, and those who insisted that they were no better were usually very mild cases or examples of autonomic imbalance.

With Dr. Roberts' technique, treatment usually occupied an average of six months including periods of rest to avoid producing telangiectases or for observation, and this was regarded as a minimum period of incapacity. In general, normal life was resumed in twelve to eighteen months. It was very striking that improvement continued after cessation of X-ray therapy sometimes for a further six to nine months. It is evident that no definite rules can be laid down for rate of progress in each case.

Three cardinal points must be emphasized:

(i) Every case of thyrotoxicosis is an individual problem, and there is a place for medicine, surgery, X-rays and psychotherapy in treatment of the various types. There is no panacea for thyrotoxicosis and only by skilful decisions can the best results be obtained from the various forms of treatment.

(ii) No attempt has been made to compare the relative merits of X-rays or surgery in cases suitable for either method. The decision rests entirely on individual factors such as the surgical and radiotherapeutic facilities available, and the economic position and wishes of the patient.

(iii) Surgery and X-rays can only do the same thing by different methods—namely removal of the thyroid component of thyrotoxicosis—and a patient upon whom thyroidectomy would confer no benefit, would also derive none from X-ray therapy.

Suggested Indications

(i) Primary thyrotoxicosis of such a degree that severity does not demand early operation, nor its mildness suggest autonomic imbalance rather than true thyrotoxicosis.

(ii) Primary thyrotoxicosis in children and adolescents in whom one is reluctant to advise removal of a valuable endocrine gland during the growth period.

(iii) Cases of primary thyrotoxicosis in whom thyroidectomy has been followed by relapse.

(iv) Patients with primary thyrotoxicosis who refuse operation, or whose dread of it threatens mental breakdown.

Contra-indications

(i) Cases of secondary thyrotoxicosis or toxic nodular goitre.

(ii) Primary or secondary thyrotoxicosis with cardiac failure or auricular fibrillation (paroxysmal or established).

(iii) Where the goitre causes tracheal compression or displacement, and retrosternal goitre.

(iv) Where the diagnosis of thyrotoxicosis is doubtful.

(v) Where the fundamental decision cannot be made as to whether the thyrotoxicosis is primary or secondary.

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Mr. Frank Ellis: This paper is based on 91 cases referred to me for radiation treatment for thyrotoxicosis from 1931-41. Of these 3 were treated successfully by operation before radiotherapy was instituted and two were cases of carcinoma found at operation for thyrotoxicosis and treated by radium for recurrence in the scar. These cases are not included in the discussion. In addition to these 4 cases have not been followed up, leaving 82 cases to form the basis of this paper.

The patients were all referred through consulting surgeons or physicians, radiation being preferred to surgery because of the case being so slight or so severe that operation was not considered justifiable (although in one case operation was refused) or because, having had one or even two operations the patient still suffered from thyrotoxicosis.

Besides the 4 not followed up there were 9 patients who were not medically examined during the past month. The other 78 were all examined for this meeting by 2 colleagues and myself. The analyses included in this paper were made from the clinical notes on a special form completed as far as the facts would allow.

Of the 82 patients treated by radiation and followed up, 54 were classified as cases

of primary thyrotoxicosis and 28 as cases of secondary thyrotoxicosis. This division was found to be difficult. The primary cases were mostly young, with definite exophthalmos, no cardiac irregularity, not necessarily having lost much weight, but suffering greatly from nervous symptoms and having a uniform smooth goitre. The secondary cases were generally at the menopausal age, with less tendency to exophthalmos, higher incidence of cardiac irregularity, great loss of weight and irregular or slight goitre.

Cases free from thyrotoxicosis are said to be improved. Cases with slight symptoms of thyrotoxicosis are said to be improved.

To assess the place of radiotherapy in the treatment of thyrotoxicosis, the following points must be considered:

(1) The Influence of Radiotherapy on Thyrotoxicosis

This can be ascertained by assessing the results in cases treated by radiotherapy alone, or after surgery. In the group of cases of primary thyrotoxicosis 50 came under the heading of which 37 were cured and 13 were improved. In the group of secondary thyrotoxicosis were 21 cases of the 28 filling this condition and of these 10 were cured,

TABLE I.—CASES TREATED BY RADIOTHERAPY ALONE OR AFTER SURGERY.

| Primary thyrotoxicosis (54 cases). | | | | Secondary thyrotoxicosis (28 cases). | | | |
|------------------------------------|-------------|-----|-------|--------------------------------------|-------------------|-----|-------|
| 50 so treated | 37 cured | ... | = 74% | 21 so treated | 10 cured | ... | = 48% |
| | 13 improved | ... | = 26% | | 6 improved | ... | = 28% |
| | | | | | 3 unchanged | ... | = 14% |
| | | | | | 1 worse | ... | = 5% |
| | | | | | 1 died (cerebral) | ... | = 5% |

6 improved, 3 unchanged and one made worse, while one died after ten days' radium treatment of a cerebral embolus or thrombosis, having had a similar cerebral attack before admission to hospital. Thus in the primary group all were improved and 74% cured, while in the secondary group 48% were cured and 28% only improved. Therefore radiotherapy can cure or improve most cases of thyrotoxicosis, but is more effective in the primary than the secondary type.

TABLE II.—INFLUENCE OF RADIATION ON SYMPTOMS AND SIGNS.

| | Primary. | | | | Percentage | Secondary | | | | Percentage |
|----------------------|----------|----|----|----|------------|-----------|----|----|----|------------|
| | Before | SL | SV | SL | | Before | SL | SV | SL | |
| Palpitation | 48 | — | — | 8 | 93 | 18 | 1 | 4 | 5 | 65 |
| Nervousness | 48 | — | — | 11 | 88 | 16 | — | 2 | 4 | 75 |
| Sweating | 47 | — | — | 9 | 90 | 14 | 1 | 2 | 1 | 83 |
| Exophthalmos | 35 | 5 | — | 24 | 69 | 7 | 1 | 2 | 3 | 53 |
| Dyspnoea | 47 | — | — | 12 | 87 | 19 | — | 6 | 2 | 63 |
| Dysphagia | 13 | — | 1 | 3 | 81 | 2 | 1 | 1 | 1 | 40 |
| Sleep | 33 | — | 2 | 3 | 90 | 5 | 3 | 3 | 2 | 48 |
| Work | 44 | 5 | — | 7 | 93 | 16 | 1 | 3 | 5 | 67 |
| Menses | 12 | — | 3 | 1 | 71 | — | — | — | — | — |
| Diarrhoea | 8 | — | 6 | — | 100 | 4 | — | 0 | — | 100 |
| Cardiac irregularity | — | — | — | — | — | 6 | — | 7 | — | ~16 |

Slight = $\frac{1}{2}$ Severe, for assessment of percentages.

SV = Severe.

SL = Slight.

In connexion with this point I analysed the symptoms and signs with regard to the influence of radiotherapy. The figures are shown. The percentages are assessed by allowing a half each "slight" symptom or sign. Again the percentage "cure" of symptoms is high and better in primary than in secondary thyrotoxicosis, but in none of the 6 cases in the secondary group with cardiac irregularity was the irregularity cured and in one case auricular fibrillation developed after treatment.

(2) The Particular Factors Making for Success or Failure of Radiotherapy

This must be considered with regard to the patients and with regard to the treatment. It is intended here to deal with the former. The question of primary or secondary thyrotoxicosis has already been dealt with. The influence of sex and age has been investigated.

TABLE III.—CASES TREATED BY RADIOTHERAPY ALONE OR AFTER SURGERY.

| Primary thyrotoxicosis. | | | | Secondary thyrotoxicosis. | | | |
|-------------------------|----------|------------|--|---------------------------|---------------------------|-------------|--|
| Influence of Sex. | | | | Influence of Sex. | | | |
| 8 M. | 4 cured | 4 improved | | 6 M. | 3 cured | 1 improved | |
| 42 F. | 33 cured | 9 improved | | 15 F. | 7 cured | 2 unchanged | |
| | | | | | 1 unchanged | 5 improved | |
| | | | | | 1 worse | 1 worse | |
| | | | | | 1 died (cerebral embolus) | | |

No significant influence of sex.

In the primary group of 8 males 4 were cured and 4 improved, while of 42 females, 33 were cured and 9 improved.

In the secondary group of 6 males 3 were cured, 1 improved and 2 unchanged, while of 15 females 7 were cured, 5 improved, 1 unchanged, 1 worse and 1 died.

The differences are not significant.

The differences in results also according to age-groups are probably not significant.

Primary thyrotoxicosis.

| Age | Cases | Cured | Improved |
|----------|-------|-------|----------|
| Under 20 | 9 | 7 | 2 |
| 20-29 | 15 | 7 | 8 |
| 30-39 | 13 | 10 | 3 |
| 40-49 | 13 | 13 | |

Secondary thyrotoxicosis.

| Age | Cases | Cured | Died | Improved | No change |
|------------|-------|-------|----------------------|----------|-----------|
| 40-49 | 4 | 2 | 1 (cerebral embolus) | | 1 |
| 50-59 | 8 | 5 | | 2 | 1 |
| 60-69 | 9 | 3 | | 3 | 1 |
| and 2 died | | | | | |

In view of the small numbers, the only significant fact seems to be the increased cure rate in the older patients suffering from primary thyrotoxicosis. This high rate at least suggests that they have been assigned to the correct group.

It was thought possible that previous operation might influence results.

In the primary group 12 cases in which operations had failed showed a ratio of cured to improved of 6:6. With such small numbers this is not a significant difference from the ratio in the whole group and any lack of response may be due to the fact that the cases were particularly stubborn since surgery was unsuccessful. 2 cases in the secondary group were treated after operation and were both cured.

The severity of the thyrotoxicosis is obviously of considerable importance, but the records do not lend themselves easily to analysis on this point. My impression is, however, that the very severe cases are as likely to be completely cured as are the mild cases, but they may require more treatment.

The Results of Radiotherapy Compared with Surgery

Neglecting in this section the question of operative mortality, it is safe to say that not all cases operated upon were cured because 14 of the 82 cases referred and treated were patients who had had partial thyroidectomy performed (twice in two patients).

TABLE IV.—CASES OPERATED UPON: PARTIAL THYROIDECTOMY.

| Primary thyrotoxicosis. | | Secondary thyrotoxicosis. | |
|----------------------------------------------|--|----------------------------------------------|--|
| Before Radiotherapy: i.e. surgical failures. | | Before Radiotherapy: i.e. surgical failures. | |
| 10 | | 2 | |
| 54 | | 28 | |
| Improved by radiotherapy 4 | | Cured by radiotherapy 2 | |
| Cured by radiotherapy 6 | | | |
| After Radiotherapy: i.e. radiation failures. | | After Radiotherapy: i.e. radiation failures. | |
| 5 | | 7 | |
| 54 | | 28 | |
| Cured by operation 2 | | Cured by operation 1 | |
| Improved by operation 1 | | Improved by operation 6 | |
| Cured by further radiation 1 | | | |
| Improved by further radiation 1 | | | |

In the primary group there were 10 in whom operation failed and of these 6 were cured and 4 improved by operation. Of patients whom I referred back for operation because of the failure of radiotherapy 2 were still not cured by operation and further radiotherapy cured 1 and improved the other. 1 patient was submitted to surgical interference without my knowledge only four weeks after a course of radium. She subsequently developed myxœdema, but was cured of thyrotoxicosis. I do not class this as a failure of radiotherapy since sufficient time was not allowed.

In the secondary group only 2 cases were referred for radiotherapy after operation. Both were cured. 7 cases, however, were referred for operation after radiation had failed to effect a cure. Of these 6 were improved and 1 cured.

The figures are shown in the accompanying table. It is obvious that there are fewer surgical failures in the secondary group and more radiation failures than in the primary group. This suggests that operation is more definitely curative in the secondary group. This group is the one in which the symptoms and signs are more definitely those of uncomplicated hyperthyroidism. For instance, there is less exophthalmos. The corollary to this is that the so-called secondary thyrotoxicosis is really primary thyrotoxicosis and that the thyrotoxicosis of the so-called primary group is a secondary phenomenon.

There was a possibility that the cures of surgery were better in quality than those of radiotherapy and this question was tested roughly by comparing the averages of the pulse-rate, weight and blood-pressure of the operated cases and those treated purely by radiation.

No statistical significance is claimed for the figures because of the small numbers of estimates available of conditions before treatment. In no instance is the result of radiotherapy inferior to that of surgery.

It must be remembered, however, that a large proportion of these figures represent surgical failures, but they were the only figures available.

| | Pulse-rate | | Weight | | Blood-pressure | | | | Pulse pressure | |
|--------------------|------------|----|--------|------|----------------|----|----------|-----|----------------|----|
| | B | A | B | A | Diastolic | | Systolic | | B | A |
| <i>Primary :</i> | | | | | | | | | | |
| Surgery ... | 119 | 92 | 7-7 | 9-0 | — | 94 | — | 160 | — | 66 |
| No surgery ... | 125 | 84 | 7-11½ | 9-8 | 74 | 86 | 150 | 142 | 76 | 56 |
| <i>Secondary :</i> | | | | | | | | | | |
| Surgery ... | 110 | 85 | 7-4 | 9-0 | 97 | 94 | 193 | 170 | 94 | 76 |
| No surgery ... | 123 | 96 | 6-7 | 9-10 | 92 | 95 | 180 | 164 | 88 | 69 |

I was under the impression that the effect of radiotherapy might be to reduce the pulse pressure by raising the diastolic rather than lowering the systolic pressure. The above figures do not support such an impression but the point needs investigating.

It is important to decide at what period the patient commences to improve and when the improvement attained may be considered complete. This question is dealt with in the analysis of the effects of technique.

The Dangers of Radiotherapy Compared with those of Surgery

Danger of death is very remote as a direct result of radiotherapy. Most authors agree on this point, although Prüfer in 1931 reported 6 fatalities.

Of the patients concerned in this investigation 6 are dead. The only case in the primary group to die, did so five years after treatment from bronchopneumonia. She was free from thyrotoxic manifestations. Of the other 5, all in the secondary group, 2 died of myocarditis, one of diabetes mellitus, 1 of widespread metastases from carcinoma of the breast and 1 of cerebral embolus. The last was the only one in which treatment could be implicated as a possible cause and in this case it seems unlikely because she had suffered a similar attack at home before admission into hospital. It can definitely be accepted that the mortality of radiotherapy is less than that of surgery. This is partly because the acute crisis is a much more likely event following operation and as Joll remarks, there is no known panacea for the acute post-operative crisis. The other dangers of radiotherapy are, skin damage, myxœdema and cardiac degeneration due to delay in removing the toxic process, as a possible cause of avoidable damage to the heart muscle, but in this series of cases only 1 case has developed irregularity of the heart rhythm, whilst under observation. Against this, however, is the fact that none of the patients with cardiac irregularity was cured of it although this commonly happens after operation. Other complications such as respiratory infection and laryngitis have not occurred. Two patients who developed an acute crisis, both severe cases in the primary group, were controlled by iodine and intravenous saline. The operation is not rendered noticeably more difficult. 5 cases in the primary group and 7 in the secondary group operated on after radiotherapy all survived the operation and the wounds healed by first intention. The dangers of tetany and recurrent laryngeal nerve paralysis have not been mentioned in the literature as a possible result of radiotherapy and have certainly not occurred in this series. One other danger of operation to which radiotherapy is not liable is that of making further operation impossible, partial thyroidectomy makes a further operation much less possible.

One danger common to both radiation and surgery is that the underlying cause of thyrotoxicosis may remain untreated. In many cases neurosis bordering on psychosis has seemed to me to be the cause and should be treated.

The Effect of Differences in Technique

Several techniques have been used for X-ray treatment, but only one method for radium in this series.

The radium technique has been to apply radium on a wax collar at a distance of 4 cm. from the skin. The dose delivered at a depth of 2 cm. to the surface of the skin, treating an area of about 8 cm. square over each side of the thyroid, is of the order of 1,000 r in ten days. This course of treatment is repeated after about three months and again three months later in most cases. In some cases, fewer than three courses, and in some a fourth course of treatment, were used. For the treatment, the patients were admitted to hospital and the radium applied for twelve hours daily for ten days.

The X-ray techniques can be divided into four groups:

- A.—One dose of radiation of 400 r delivered to the whole of the thyroid, small fields being arranged to deliver the dose uniformly.
- B.—A dose of about 800 r delivered to the thyroid gland in one week.
- C.—A dose of about 1,200 r delivered to the thyroid gland in two weeks.
- D.—Weekly doses of about 250 r given for about eight to ten weeks.

200 kV. 1 mm. Cu. H.V.L. 1.5 cm.

In all these methods, the size of the field used was sufficient only to cover the gland. No attempt was made to use large fields of about 15 or 20 cm. in diameter such as have been used by various American authors.

The effects of the various methods of treatment in bringing about cure or marked improvement have been analysed. This analysis indicates no effect of age on the time of improvement of the patients' symptoms and signs and these figures are not included.

The relationship of type of treatment to the time of improvement is indicated in the accompanying tables. Adequate data were not available at the time of this analysis, but no selection was attempted, the figures being taken from notes made at the time the improvement was noted.

PRIMARY THYROTOXICOSIS—*Initial Improvement*

Of 46 out of 50 patients in whom such notes were available, 25 showed improvement within one month of treatment being started and 39 within three months. The proportions for radium and X-rays of early initial improvement are similar.

Final Improvement

The striking fact here is that nearly half the patients treated by X-rays were cured in less than three months, while the same proportion of patients treated by radium did not attain final improvement for one or two years and, similarly, those previously operated on showed late final improvement but early initial improvement. Most of these were treated by X-rays.

SECONDARY THYROTOXICOSIS—*Initial Improvement*

3 out of the 4 treated with X-rays showed initial improvement within one month, while only 3 out of 12 of the radium patients showed initial improvement in the first month. Of the 2 treated after operation, both show late initial improvement being treated with only one course of radium.

Final Improvement

The final improvement of the radium cases was again relatively late, but so was that of the cases treated with X-rays, although both of those previously operated on were cured within one year.

The early improvement of most of the cases makes radiotherapy in no way inferior to medical treatment and the long period of invalidism to which the latter condemns a patient is definitely avoided. The difference between the effects of radium and X-rays, however, suggests that the irradiation of the rest of the body may have a delaying effect on the ultimate result. When one remembers the generalized hyperplasia of lymphoid tissue which is characteristic of exophthalmic goitre these results suggest that this lymphoid tissue, sensitive as it is known to be to radiation, may be damaged by the radium and that its damage may result in a delay in the healing processes, whatever they are. The implication of this speculation is that the lymphoid reaction may be an attempt to deal with the cause or the effects of the toxic process.

This suggests that X-ray treatment is better than radium for thyrotoxicosis. There is an added advantage in X-ray treatment when one considers that the patients so treated were all treated as out-patients and did not require hospitalization. Moreover, although the radium cases all rested in bed during treatment two of them developed crises which were treated with intravenous saline and digitalis, and none of the X-ray patients were so affected.

The patients undergoing treatment are advised to rest, but are not kept in bed unless their general condition, or the treatment, make it necessary. The effect of radiation on the thyroid gland seems to be to cause involution. Three slides are shown in which radium was used before operation and one in which no radiation was used. The patients all had iodine, so that the difference cannot be said to be due to iodine.

In connexion with the question of iodine treatment as an adjuvant to radiotherapy I can only say that my impression, for which I have no proof, is that cases so treated at the time of radiation were longer in recovering from their thyrotoxic symptoms. This accords with the view of Jenkinson and Hunter (1938) and Williams (1932).

No adverse effect on the operation was caused by the radiation. In no case were the operation difficulties increased by the previous radiation and no operation mortality has occurred.

In the past ten years many papers have been published in the continental and especially in the American literature and from these it is obvious that results of the kind I have been discussing have been obtained for some time. This being so it is difficult to understand why in this country more radiotherapy has not been practised in this condition. It may be due to a mistrust of results which I hope this analysis will do something to dispel. The criteria of improvement should certainly be very rigid to satisfy us, but these criteria should be applied equally to both surgical and radiological cases.

The possibilities of radiotherapy do not necessarily end with the thyroid. Cases have been reported in which the pituitary was irradiated (Borak, 1935; Habs, 1936), and it is suggested that combined irradiation of the pituitary and the thyroid might improve the results, the latter author noting a more rapid improvement in the combined cases.

In two cases of this series the pituitary was irradiated and in one temporary improvement was noted, no other treatment being given. In the other, the irradiation of the pituitary was followed by a rapid disappearance of the remainder of the thyrotoxic symptoms persisting after treatment by a radium collar.

CONCLUSIONS

In routine treatments the conclusion may be drawn that radiation with X-rays (or a radium beam) to the thyroid is the treatment of choice in primary thyrotoxicosis, but that in secondary thyrotoxicosis operation is the treatment of choice, because although X-rays may be used to improve such cases, it cannot cure the cardiac irregularity and the delay in improvement relative to operative results might allow cardiac degeneration to become worse.

My thanks are due for the help of all the medical and clerical workers of the Sheffield Radium Centre, and especially to Dr. Brody and Dr. Shanks, without whose help I could not have done the necessary work. I am grateful to Dr. Hermitte for the histological slides and to Mr. Watson for preparing the other slides.

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[The foregoing papers were amongst those read at this meeting.]

Section of Psychiatry

President—A. A. W. PETRIE, M.D., F.R.C.P.

[October 14, 1941]

Reconstruction in Psychiatry (*Abridged*)

PRESIDENT'S ADDRESS

By A. A. W. PETRIE, M.D., F.R.C.P.

My predecessor in this Chair, Dr. C. P. Symonds, gave a reasoned statement in his Presidential Address why he believed that Neurology and Psychiatry would inevitably come together into one combined specialty, whose practitioners he designated as Neuro-Psychiatrists. I firmly believe in this, and in this paper I have reiterated such arguments.

The subject matter discussed has largely appeared in various documents such as the Feversham Report on the Voluntary Mental Health Services [1], the Report of the Committee on Mental Health Services, of the British Medical Association [2], and some of the points were considered in a departmental committee under the Earl of Radnor, the activities of which were terminated by the war. Various committees also considered medical education in relation to psychiatry and made reports. I have referred to voluntary sterilization which has been the subject of a blue book [3]. A brief combined re-statement of all these projected changes does not appear inopportune at the present time.

What will be the future of medical practice? Will the general practitioner and the consultant whose independence has been so valuable to the community become mere Civil Servants without proper power of expression and representation?

The future of Psychiatry is important both to the medical profession and to the community at large.

It is still a developing service made up of different components.

At present workers enter this field from a number of different aspects, with the result that their knowledge reflects their angle of incidence, and to this knowledge is generally added a number of prejudices which prevent a proper conception of the whole. I envisage a gradual merging of these components into one important and dominant group of medical opinion, influencing the profession and the nation.

The gap between the academic psychologist and the medical psychologist is to some extent bridged by the industrial and educational psychologist, to whom the subject of potential capacity means so much. The utilization of elementary psychological principles in advising suitable occupations has diminished the number of misfits and has resulted in increased output. Factory rules and regulations have been modified to the benefit of all concerned. Proneness to accident is another point on which the psychological worker may warn the industrialist, and so save trouble both to the employer and to the insurance company.

Closer contact between the psychiatrist and industry is necessary when considering the problem of absence from work, due to neurosis. These absences are described under many different headings, such as dyspepsia or bronchial asthma, according to which organ the patient primarily complains about. An understanding psychiatric physician would soon diagnose the essential condition and save many big industrial undertakings a large number of hours owing to absences.

The educational worker is undertaking most important work, and it has been suggested that the future university student can be predicted when at an elementary school. There is a danger of too great deductions being made from these tests. The emotional and volitional factors are not covered by the usual tests, and these factors are at least as important as the intellectual equipment, and although personality tests are being developed, these are apt to depend too much on the point of view of the investigator. Interviewing boards, to modify the results of a pure examination system, show the reaction against the errors which can be made by a slavish adherence to any mechanical form of test. The mistakes that a pure examination scheme can produce, is shown in the fallacies of an earlier civilization, namely, that of China where a too conventionalized examination system caused a stagnation which ultimately led to violent upheaval. A personal evaluating factor to counter-balance this can be supplied by the experienced physician. Recent personnel selection in the armed forces is developing on these lines. All psychiatrists come across the unstable, but highly intelligent person, who, exalted

beyond his capacity, causes trouble and who generally fails to develop into a personality capable of guiding others.

On the mental deficiency side, the training evolved, mainly from the educational side, has proved of great value. Careful and systematic teaching raises the whole standard of the individual, and in the higher grades, enables a practical system of resocialization to be attempted with some success. In the lower phases of mental deficiency there are many contacts with the neurologist and many syndromes have been described bearing on neurological work. Again, schizoid deterioration is not uncommon in the defective, indicating the need among such cases, for an individual with a knowledge of neuro-pathology and general psychiatric experience allied to a knowledge of mental deficiency.

The approach from the side of the neurosis has perhaps attracted the most interest as it involves the greatest number of potential patients, and touches general medicine at all its angles.

The psychology of many different physical diseases is attracting more attention and the need for teaching the medical student these aspects is slowly being appreciated. The present-day student is realizing that in a considerable proportion of his patients, the psychological factor is predominant, and its consideration is essential to a proper diagnosis and treatment of the cause.

These minor failures to adjust to the environment are receiving increasing notice, and opinion varies as to the relative importance of the constitutional and environmental factors. The former is stressed by those who tend to ignore the possibilities of treatment, while the latter is inclined to be over-emphasized by those who treat them. It is becoming apparent that organic upsets in the central nervous system can create or increase the tendency to the neurotic or the psychoneurotic reaction in those formerly normal or comparatively normal. The obvious instance is seen in the sequelæ of epidemic encephalitis, while the most evident analogous organic disorder in the psychoses is seen in general paralysis. In each case, the importance of an organic disturbance is apparent and it is here that the neuro-pathologist and neurologist can so suitably work in conjunction with a psychiatrist or better still, a neuro-psychiatrist.

The Basis of the Mental Health Services

Useful work is being done by the National Council for Mental Hygiene which has linked the voluntary psychiatric activities together, and brings these needs before the public. The work of the Feversham Committee was to inquire as to these activities and to co-ordinate and focus them into an effective whole. These proposals suggested the fusion of the National Council for Mental Hygiene, the Central Association for Mental Welfare, and the Child Guidance Council, into one body to be called the National Council for Mental Health for England and Wales. The Feversham Committee presented an excellent report which assumes the voluntary principle where possible, and suggests the linking up of all activities, for example, the co-operation between the education and mental health committees of County Councils, with, in some cases, joint committees, is an obvious necessity. The report quotes Professor Henderson as saying that we must talk of mental health and its maintenance, rather than mental disease and its cure. This is an essential outlook. The physical and mental health services should be closely united and this has recently been done in London—a progressive step.

MENTAL SERVICES AND PSYCHIATRIC HOSPITALS

A radical change of standpoint among public bodies is essential, and a well-ordinated scheme between the voluntary and State or municipal services is as important from the mental aspect as on the physical health side.

The public bodies should establish or reconstitute mental health committees, which will deal with all questions relating to mental health, in their area. Their function will be to further mental health, and only incidentally to deal with segregation.

The educational problem, including child guidance, as well as that for the various grades of mental deficiency, will need to be included in these schemes, and representatives should be appointed to confer and co-ordinate with the clinics held at voluntary hospitals and elsewhere.

The Joint Board should be formed of university or hospital authorities and the local authorities.

Should beds for mental cases be in a general hospital or in an associated clinic? The absence of facilities for exercise and adequate occupations, which is essentially a part of mental treatment, renders any small unit, without such amenities inadequate and structural modifications, if acute cases are taken, make such a unit difficult to obtain. Against this is the need felt in all large hospitals for some beds where cases taken from the other departments, e.g. maternity or medical, can come for mental observation. This

would save cases having their physical progress interrupted by being removed to quite another institution.

A larger clinic serving a group of hospitals is able to provide more beds, and allows a better classification, and, given a suitable location, may have space to allow proper exercise and occupational centres.

The obvious rejoinder is to move it to the country, or to set up its functions in the grounds of the mental hospital. Under present conditions, I believe such a unit is best maintained in juxtaposition to a general hospital, where every form of expert advice is readily obtainable. Given that the planning of the future transfers the great hospitals to the periphery of the cities, part of this objection would be met and the mental unit could move with the general hospital. There are two sources from which such units can develop: a centre for less acute cases receiving the psychoneurotics and mild psychoses on a voluntary basis, and a unit receiving the more acute psychoses, including those who need care and restraint. If the clinic for milder types is evolved as part of a general hospital, no further co-ordination will be necessary, but if it develops as a separate unit, its out-patient department both for children and adults, will need to be linked up with those of the neighbouring teaching hospital, and its facilities made available for students. Similarly, with in-patients, there will be no need to duplicate beds, if the unit is close to the teaching hospital.

As indicated, one source of development of such a clinic is as part of the university or teaching hospital; so far this has not occurred in this country, although there have been tentative attempts to establish such centres in connexion with teaching hospitals in London and the Midlands, but the war has stultified such efforts. Generally speaking, however, it is likely that the university clinics will tend to be devoted to the less acute kind of mental illness. The type of case received will inevitably largely depend on the director of the clinic, as he naturally attracts the type of case in which he is interested. One with a practice primarily concerned with the neuroses will receive many of this class and incipient psychoses may prevail more in other instances. Perhaps the most interesting and important from the point of view of development is the neuropsychiatric clinic, which may be a special ward or branch of a more general unit. Under Sir Frederick Mott, the Maudsley Hospital, when dealing with military cases during the 1914-18 war, combined all these functions with reasonable success, and under Professor Winkel the Utrecht clinic had a similar tendency. The Tavistock Clinic has also concentrated its activities on out-patient work with the psychoneuroses, and has combined teaching with its other activities.

The only really notable development of the clinic idea in England, has been the Maudsley Hospital, which represents a University Clinic maintained by a municipal authority. This was initiated, when a sum of money was left by that far-sighted benefactor, Henry Carr Maudsley, aided by Sir Frederick Mott, and their ideals were further developed by Edward Mapother. Despite the notable success attained, no municipality has so far attempted to repeat the venture.

Coming to the unit for the more acute type of case I believe the study of the early stages of the more acute syndromes offers an even greater scope for physiological and neuropsychiatric investigations. Every kind of clinical and metabolic observation at this stage is likely to be of value, but at present, little use is made of these facilities. In the larger cities these cases are generally received into observation wards, but the buildings and laboratory facilities are often ill-adapted for their purpose; the staff maintained is not necessarily expert and sometimes nurses, attendants, and doctors regard the work as troublesome and uninteresting.

No attempt has so far been made to change the observation ward into a psychiatric hospital for the more acute type, although the London observation wards have been combined, enlarged and improved. The utilization of such reception centres as teaching research units, has been little developed. The first steps, already taken in London, are to provide specially trained doctors and nurses, and this alteration alone has produced greatly improved conditions. After this, a removal to more suitable buildings should be merely a question of time.

Improvements in the observation wards may gradually transform them into psychiatric hospitals where real advancement in the specialty can be made. Then perhaps as the worth of this work is realized, enlightened municipalities will create suitable buildings.

In the smaller towns, the possibility of two psychiatric hospitals in one area hardly seems reasonable, and a fusion of all psychiatric clinics in one centre seems the only solution. Here again, the question as to the basis arises. Will developments come from the side of the key general hospital or will the municipal services gradually improve until the broader psychiatric needs of the neighbourhood are satisfied?

Mental Hospitals

No number of clinics will remove the need for treatment in the mental hospitals, owing to the fact that many cases take longer to recover than can be reasonably catered for in a clinic, with its limited number of beds. Further, it is impossible to forecast with certainty which cases will ultimately recover, and which may need continued care. Many potentially recoverable cases will be sent to the mental hospitals.

No one can deny that there is apprehension and a sense of inferiority at the suggestion of care at a mental hospital. The patient is considered to have disgraced himself, and reflexly, his relatives, by exhibiting abnormality. This is partly due to the mediæval attitude still felt towards any mental disturbance, where disorder of conduct is exhibited, and is based partly on the sound principle that stable stocks do not exhibit mental disorder, and that there must therefore be some weakness, degeneration or deteriorating of the stock, for such an abnormality to appear. Many of those who express an unnatural antipathy towards psychiatry and psychiatrists are sometimes exhibiting an over-reaction to a fear or knowledge of such instability in their own family group. Allowing for all these factors, a third reason, for prejudice against mental hospitals, remains the fact that segregation of the chronic patient is dealt with in the same institution as the treatment of the recoverable.

So far, the Board of Control has met the problem by inducing all authorities to build in the grounds of the mental hospital, admission units for recoverable cases, separated from the chronic wards, by means of separate entrances and similar devices and have thus endeavoured to dispel the prejudice. I believe that if recoverable units were situated on land separated from the mental hospital much prejudice would be avoided.

Buildings on nearby sites will cost little more than siting such buildings within the grounds of the mental hospital. The staffing of such units from the mental hospital will obviate much of the cost of establishing a new unit complete with personnel. This gives the further advantage that it will keep the medical men and nurses of the mental hospital in contact with the recent and recoverable cases which is so essential. Any type of staff, either medical or nursing, tends to deteriorate if only brought into contact with chronic patients. It must be admitted that the main mental hospital will become more depressing if recoveries and discharges are rare events, but the benefit to the recoverable patients, in treating them apart, seems to justify the experiment.

In public bodies who control many institutions, the question of the separate accommodation for the recoverable patients might, it is argued, be solved by the use of one hospital for all recoverable cases; against this is the problem of the medical and nursing staffs and also the fact that patients need to be treated somewhere near their own districts. Further, any unit of acute cases must be strictly limited in numbers, to allow of intensive care and supervision.

The social services include reports on admissions, with adjustments in their homes, help to those undergoing care, and resocialization of those fit for discharge. The object of every well-organized mental service should be to prevent or diminish the large and costly segregation problem. Some persons will need care for many years, but many, although not recovered, may be resocialized in from one year to eighteen months, while others gradually lose their acute symptoms often with an increasing degree of deterioration and dementia. Resocialization and partial resocialization should be tried, particularly among the older age-groups, by a systematic boarding-out, or prolonged trial system. Cases of doubtful stability which, if discharged forthwith, or after a short trial period, will inevitably relapse may be resocialized if dealt with slowly. Remunerative work where they may be economically self-supporting should be found. To a slight extent, this development touches the need for sterilization, but among the older age-groups, this will hardly be necessary.

The Mental After Care Association provides after care homes, but all large services should obviously have rehabilitation centres of their own, where discharges can be received, and suitable employment found for them and a home to live in.

If every quiescent patient was systematically dealt with on these lines, before he or she became institutionalized, a vast saving to the community would result. One trouble in trying to discharge such patients, who have been in for a period of time, is that they have become too lazy to face the struggle for existence. Unless urged by the desire for sexual gratification or alcoholic refreshment, they have no motive to re-face a world which has usually treated them badly.

The aggregation of these groups of institutionalized patients has been defended on the ground that by their work they help run the institution, and enable the others to be maintained at a cheaper rate. The patient whose work equals his cost of maintenance is rare, and in nearly all cases, the debit is definitely against the patient.

In such reconstituted mental health services an experienced adviser is essential. It is rare, however, to find in any one individual a knowledge of the neuroses, psychoses and mental deficiency and also of educational problems.

In the large areas, such services can be organized within present boundaries but unless the smaller areas are enlarged or co-operate together, proper services on these lines will not be possible.

London, Yorkshire and Lancashire, each form large grouped areas, where it should be easy to provide all mental facilities. In more sparsely populated districts, greater difficulties will arise. It is, however, possible to plan a regionalized system for England and Wales which would divide up the country into sections, each forming its own mental health group, complete with research and clinical centre, reception unit, social service and rehabilitation centre, not too far removed from local sub-sections, so as to enable the area to function progressively, as regards its mental health.

The cost of a well-organized mental health service will be repaid by diminishing the segregation problem, and by improving the mental and general health of the community. The outlay of psychiatric hospitals by the provision of early and suitable care, will be worth while, as cases will more readily come forward before the terminal stages, when treatment is of little avail.

The placing of the more recoverable types into buildings separated from the mental hospitals will give the recoverable mental patient a sense of being treated entirely under hospital conditions, and this, I believe, will alter the whole attitude, and dissolve much of the prejudice towards mental illness. It is only a step forward from the practice already followed in mental hospitals of keeping recoverable cases in admission units until discharge, or chronicity, supervenes.

Against this, it will be said that observation units are not necessary, and that the direct admission into the mental hospital is the obvious procedure. It is argued that by doing this, the whole status of the segregated chronic section can be sustained at a hospital level, whereas this actually prevents a true therapeutic attitude being taken towards the recoverable case. "Asylums" have been relabelled "Mental Hospitals" and now "Hospitals" but the same prejudice still largely persists. Why not change all this by altering the basis and completely separating treatment from segregation?

[Section on problems of prevention and treatment and a discussion on causation are omitted owing to paper shortage.]

Eugenic Factors

In mental disorder we are still seeking causes, while treatment especially among the functional psychoses is, despite some advances, unsatisfactory and largely empirical. One cause is heredity, but even here we have not reached a satisfactory basis, although investigations are slowly pointing a way. The two syndromes in which the heredity factor seems of greater importance are the manic-depressive group, and the schizophrenic and paranoid psychoses, and it is these that form the bulk of the cases of actual psychoses, with which we have to deal. If we are dealing with a simple Mendelian dominant, as in Huntington's chorea, the answer would be simple, namely, that it would be the duty of the State to stamp out the disorder by compulsory sterilization.

Unfortunately, these syndromes have not even simple recessive inheritance, although all figures indicate a definite and in some cases, a considerably higher proportion of abnormal inheritance, among those mentally disordered than among the normal population. The Brock Report states that inheritance is the commonest single cause of mental disorder, at the same time emphasizing the part played by environmental factors. In their recommendations, they suggest that, subject to the safeguards proposed, voluntary sterilization should be legalized in the case of (a) a person who is mentally defective, or who has suffered from mental disorder; (b) a person who suffers from or is believed to be a carrier of a grave physical disability, which has been shown to be transmissible; (c) a person who is believed to be likely to transmit mental disorder or defect.

This was presented in 1933, but the facts contained in it, although hotly debated, are incontrovertible, and society and the state are neglecting a primary duty to the health of the nation in shelving this obvious reform.

The fact that sterilization will only partly affect the problem, and that the disease may not manifest itself until after children have been born, and that the detection of latent carriers is difficult, should not deter from action. In a democracy, and until further knowledge is acquired, the first step must be on a voluntary basis, but even this should do something to prevent the spread of disease and degeneration among the healthy stocks.

Some allowance will have to be made for the fact that brilliant strains are sometimes

Mental Hospitals

No number of clinics will remove the need for treatment in the mental hospitals, owing to the fact that many cases take longer to recover than can be reasonably catered for in a clinic, with its limited number of beds. Further, it is impossible to forecast with certainty which cases will ultimately recover, and which may need continued care. Many potentially recoverable cases will be sent to the mental hospitals.

No one can deny that there is apprehension and a sense of inferiority at the suggestion of care at a mental hospital. The patient is considered to have disgraced himself, and reflexly, his relatives, by exhibiting abnormality. This is partly due to the mediæval attitude still felt towards any mental disturbance, where disorder of conduct is exhibited, and is based partly on the sound principle that stable stocks do not exhibit mental disorder, and that there must therefore be some weakness, degeneration or deteriorating of the stock, for such an abnormality to appear. Many of those who express an unnatural antipathy towards psychiatry and psychiatrists are sometimes exhibiting an over-reaction to a fear or knowledge of such instability in their own family group. Allowing for all these factors, a third reason, for prejudice against mental hospitals, remains the fact that segregation of the chronic patient is dealt with in the same institution as the treatment of the recoverable.

So far, the Board of Control has met the problem by inducing all authorities to build in the grounds of the mental hospital, admission units for recoverable cases, separated from the chronic wards, by means of separate entrances and similar devices and have thus endeavoured to dispel the prejudice. I believe that if recoverable units were situated on land separated from the mental hospital much prejudice would be avoided.

Buildings on nearby sites will cost little more than siting such buildings within the grounds of the mental hospital. The staffing of such units from the mental hospital will obviate much of the cost of establishing a new unit complete with personnel. This gives the further advantage that it will keep the medical men and nurses of the mental hospital in contact with the recent and recoverable cases which is so essential. Any type of staff, either medical or nursing, tends to deteriorate if only brought into contact with chronic patients. It must be admitted that the main mental hospital will become more depressing if recoveries and discharges are rare events, but the benefit to the recoverable patients, in treating them apart, seems to justify the experiment.

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At most hospitals it is customary to allocate clerks to the Psychological Out-patients Department, where a conception of the neuroses, and, to some extent, the incipient psychoses, is obtained. To demonstrate psychotherapy in front of students is difficult, as patients are more ready to display their bodies in front of others than the intimacies of their minds, but after the patient has explained his symptoms, it is then easy in his absence for the teacher to expound the underlying basis of any case which he has previously investigated. This teaching can be linked up with tuition on suitable ward cases presenting psychological symptoms.

The psychology of the child and its mother is of great importance to the student, and co-operation between the psychological department and the pediatrician is essential. Some organized assistance for dealing with children is necessary, and it is questionable how far such units should be in touch with the general hospital, or form special units such as Child Guidance Centres. In any case, some study of the mental development and behaviour of the normal child is an essential of medical education.

The demonstrations at special centres are probably best given by teachers who are expert at those centres, as they will be more familiar with the cases, always assuming that reasonably expert tuition is available. Such supplementary teachers could be appointed clinical psychiatrists, and could give the teaching at the observation wards, and at the mental hospitals and mental deficiency institutions. An extension of the resident clinical clerkships among senior students would often attract suitable recruits to this class of work. It may be objected that this programme will greatly enlarge the time given by the medical student to the study of psychological medicine, leading, for example, to the neglect of medicine and surgery. Actually, little more time would be needed, and the bulk of the suggestions as to supplementing the physical side by the study of the psychological aspects, is what all good physicians have done throughout the ages, whether it be called a good bedside manner or given a higher sounding name.

As a corollary to the training of the medical student the teaching of the student nurse needs similar guidance, and some elementary lectures in psychology would be of great help in developing their training on proper lines. A system of seconding general trained nurses for short periods, to various mental treatment centres, would greatly help in promoting the understanding of both the neurotic and psychotic cases. In particular, it would increase the supply of nurses for dealing with the milder type of case cared for at home.

TRAINING OF THE SPECIALIST

All those connected with the present diploma in psychological medicine are striving to raise the training, and standard of examination, and it can, generally speaking, be said that the quality of the candidates is rather holding back the endeavour. Most of these come from the mental institutions and require to obtain the diploma if any advancement is to be obtained.

At present the principal diplomas demand, in the first part, a knowledge of anatomy and physiology of the nervous system and psychology together with some practical knowledge of the neuro-histological methods and also of practical psychology. In the second part, apart from the papers, a clinical examination is demanded both in neurology and psychiatry, the latter including the neuroses, psychoses and mental deficiency. In the London area most candidates acquire much of their knowledge from courses conducted at the Maudsley Hospital, or at times at Bethlem Hospital. Few candidates have come up to these courses with experience of more than one type of institution.

The requirements of the examining boards are likely in the future to demand experience in the neuroses, the psychoses and mental deficiency, and child psychiatry. Apart from properly devised courses, it is difficult for candidates to obtain such knowledge.

Such are the present difficulties, and these are often reflected in the one-sided knowledge of the candidates. If it were possible to insist on a really high standard, the diploma would largely fulfil any specialist requirements. The existing diploma is not, however, attracting the prospective neurologist, or the psychotherapist, who feels his especial needs are not catered for.

Dealing first with the neuro-psychiatrist, he will be required to exhibit expert knowledge of organic neurology, and be expected to be able to investigate problems in neuro-psychiatry. If he is to be able to do this, he will require a high standard in neuropathology, unlikely to be acquired by less than six months' work in a research laboratory. He will also need to become familiar with all methods of investigation, and he will require to have a good acquaintanceship with the biochemical problems likely to affect his work.

Research scholarships should assist in obtaining these workers, and it is in their training that our future hopes lie. What is to be the future of such workers?

unstable, although strong counter considerations would be necessary to justify failing to maintain the health of the nation.

I was once told by an abnormal member of an artistic family that seven of his brothers and sisters were abnormal while two were geniuses. Two were certainly prominent figures in one of the great arts, and presumably the rest were liabilities to their family and the nation.

Where abnormals, generally of the neurotic type, realize their limitations, and are willing to undergo voluntary sterilization, it seems a tragedy to withhold this. Such people wish to preserve those following them from the subjective misery they themselves have suffered, and when one considers the sum total of misery caused to others, surely we should cease to hesitate.

The intricate problem of sterilization of the mentally defective also awaits attention.

Conversely, great blame attaches to the higher elements of population who voluntarily sterilize or partially sterilize themselves by having one or two, or at the most three children, alleging economic necessity. Taxation designed to counter this tendency is the obvious remedy.

Something more than mere replacement is needed by the higher types, and every form of pressure, social and economic, should be directed towards the production of large families.

The recent tendency to a release of the ban on marriage among women employees in public services, shows a belated recognition of the fact that artificially induced spinsterhood among competent citizens is against national interest.

TRAINING OF THE MEDICAL STUDENT

Efforts to increase the psychological knowledge of the medical student have largely been resisted, on the grounds that the curriculum is already more than overloaded. Psychology is best taught with physiology but if this cannot be arranged owing to the overloaded curriculum it might be studied with the preliminary sciences and some knowledge might even be taught at the school level, among the much wider range of subjects now permitted to qualify for the entrance to the universities.

The student should be taught systematically to study the psychological make-up of every patient that he sees. Sometimes this will enable him to diagnose the condition in question. In many cases it will aid in the treatment of the patient, and even at times when it may appear irrelevant, it will have helped him towards a knowledge of mankind in general, so essential to any form of medical practice. If every clerk and dresser were compelled to add a paragraph in their case-taking, regarding the psychology of their patients, much benefit would result both to the patients and students.

Apart from general medical and surgical cases, there are numerous ear, nose, throat and gynecological cases and other special departments' cases where an adjustment of the psychological condition is as important as treating the immediate symptoms for which the patient seeks relief.

A generation trained on these lines will be greatly helped in their approach to their patients, and it will be advantageous to every kind of specialist and prove of inestimable value to those in general practice.

Apart from this necessary training in sizing up the general mentality of the patient, the practitioner needs to know and understand the possible neurotic reactions of his patients, and this can be acquired in all departments apart from that specializing in psychological medicine. The anxiety states and their variations are of principal importance, but hysterical manifestations, especially those associated with somatic disease are also important.

For the rest much can be done by teaching the students at the observation ward or at psychiatric hospitals, developed from these wards. These are usually, in the great cities, quite close to the teaching hospitals and medical schools. Little time will thus be wasted in travelling to and fro. Here he will meet the type of case which needs quick decisions in practice and which may recover so rapidly, that more permanent care may be avoided.

Demonstrations on mental deficiency are, of course, given to students, enabling them to recognize types, and to judge what can be done in any particular case, such as the use of Child Guidance Clinics. Parents bringing children to doctors always hope for a more favourable verdict than the one which they have previously received, but are unwilling to accept as true.

A knowledge of the value of intelligence testing is now becoming more universal, but the recent developments of the Educational Services is still not appreciated by all practitioners.

The general control of all this training is probably best in the hands of one supervising instructor.

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Research scholarships should assist in obtaining these workers, and it is in their training that our future hopes lie. What is to be the future of such workers?

A few will become teaching neurologists with a more tolerant bias, and perhaps a greater knowledge of the psychiatric work which they are so often called upon to deal with in private practice. Some will become teaching and consulting psychiatrists, and it is to be hoped that in the future, both these groups will become neuro-psychiatrists. It is also to be hoped that a number of these will find their *métier* in investigation and become directors of neuro-psychiatric laboratories, with which every properly organized mental health service should be associated. The type of worker outlined will, of course, easily obtain the ordinary higher diplomas of M.D. and M.R.C.P. and presumably that of the diploma in psychological medicine. Perhaps granting a diploma with special knowledge in neuro-pathology might meet these requirements, rather than giving a special diploma in neuro-psychiatry.

A conceivable solution would be to start a college in neuro-psychiatry, with Fellows and Members, as with obstetricians and gynaecologists, although this is hardly a solution which commends itself. At one time the Royal College of Physicians allowed its members to pass an examination indicating their special competence in psychological medicine, and some revival and modification of this might meet the requirements for a higher diploma for specialists.

The question of an added qualification for the medical psychotherapist raises even more debatable issues, but even a diploma in psychological medicine with special knowledge of psychotherapy seems hardly feasible.

There are a number of different schools of psychotherapy, all tending to be mutually exclusive, and an attempt to devise a test of competence is likely to daunt the boldest. While everyone has absorbed the salient and most important ideas put forward by the leading schools of psychotherapeutic thought, there is considerable divergence of view, and standpoint, and those closely attracted to certain schools of thought have evolved minor divergences of their own. Apart from the aspect of neuro-psychiatry, there have been demands for special diplomas in mental deficiency, in psychotherapy and child guidance, and delinquency may also follow suit. Such a spate of diplomas is not, in my opinion, feasible, and the institution of psychotherapeutic diplomas would have the added danger that non-medical psychologists of academic standing may wish to obtain it.

Allied to this last question is that of spiritual guidance. There are signs that the church, which at one time was largely concerned with healing may wish to re-undertake a portion of this section of our work. The wiser councils on both sides would limit the work of the doctor to the person suffering from symptoms which can be described as disease, while the work of the priest would be limited to the disturbed but normal person. This, it can be understood, is a very fine line of distinction, in which overlapping can readily occur. When patients require in-patient treatment, it seems likely that the province of the doctor is involved, and even out-patient treatment, requiring regular seances associated with payment seems to come within the same category. Much though the medical psychologist might wish to guide the thought of the nation, and wisely though he would doubtless perform this function, he has to remember that other advisers exist whose standpoint may not always be the same as his own. The problem of assisting by giving suitable psychological instruction to the younger members of religious bodies is obviously the proper way in which co-operation should be given, and the effect of such teaching, judiciously given, should be to harmonize points of view and lessen mental stress and unnecessary repressions.

SUMMARY

I recommend the creation of mental health services throughout the country, embracing areas large enough to provide every facility, with the separation of all potentially recoverable mental patients from those who have become chronic. An effort should be made to reduce the segregation problem by attempting the resocialization of all possible mental and mentally defective persons, with safeguards against the spread of the disease or defect by procreation.

As a preliminary measure, I suggest the voluntary sterilization of all markedly neurotic or psychotic patients who desire this, in the belief that much unhappiness will be prevented thereby, both among the unstable and those who have to associate with them.

There should be improved facilities in the psychological education of the medical student and a fusion of the varied interests in psychiatry, so that all the elements might benefit by a comparatively common training, thus forming an important body of opinion, which would direct and guide the mental health of the whole country.

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Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

[April 24, 1942]

DISCUSSION ON THE FUTURE OF PUBLIC HEALTH NURSING

Miss M. E. Flambert (Deputy Chief Nursing Officer, Ministry of Health): To-day we find that most of our health visitors have qualified for this work by taking a three or four years' training in general nursing followed by six months' or a year's training in midwifery, as well as the health visitor's course; a total period of from four and a half to five and a half years.

I am purposely touching on the training of health visitors because, although it is generally agreed that the best basis for their future work is that of a general trained nurse, there is some doubt whether the general training, as it is to-day, is sufficiently comprehensive. In the many schemes for the revision of the general training which have been put forward, emphasis has been laid on the importance of including more preventive teaching in the early stages of hospital training, as well as more practical experience in the nursing of infectious diseases, mental patients and the home nursing of the sick. In fact there seems to be a universal acceptance of the view that nurses should be prepared to do preventive as well as curative work and that the basic training should include the fundamentals of public health nursing.

I suppose the nearest approach to a comprehensive form of service is being undertaken to-day by those Queen's nurses who are doing health visiting as well as district nursing and midwifery. These nurses are, for the most part, working in rural or semi-rural areas where it has become obvious that much time will be saved if one person can undertake all forms of nursing work, curative as well as preventive.

Many statutory duties dealing with maternity and child welfare and tuberculosis work are, in County areas, delegated to County and District Nursing Associations. These Associations are dependent to a large extent on voluntary contributions with the result that many rural areas are unable to afford the services of a fully qualified district nurse. Instead they train what are known as village nurse-midwives—girls who have been given a full midwifery training plus six months' experience in district nursing and perhaps three months' health work. These nurse-midwives undertake in most instances midwifery and district nursing and sometimes health visiting.

Dr. James Fenton in his Presidential Address to the Society of Medical Officers of Health in 1937 (M. Officer, 58, 99) said: "Nursing, in the broad interpretation of the word, is to-day a community service as is the profession of medicine and the two must supply what is required by modern enlightened opinion. This is a threefold requirement: (1) The curing of disease; (2) the prevention of disease; (3) the teaching of health to the healthy." I should like to see all public health nurses prepared in this way so that the district nurse, whose district may be so populous that she cannot undertake the work of a health visitor, will also waste no opportunity of impressing on her patients the value of maintaining health and preventing disease.

The opportunities for public health nursing should be much more widely advertised, by talks to girls of school leaving age, and to parents and teachers, so that girls who are interested in social work and education may be attracted at an early age to public health nursing. At present, the girl who enters hospital has her vision of the future coloured by the prospect of caring for the sick for the rest of her days and may know nothing of the other and wider fields of nursing work.

A longer qualifying period required of student health visitors has also been recommended; that is to say that the health visitor's certificate should not be awarded until the student has been given a longer period of training and an opportunity of demonstrating her suitability for the work, as well as her ability to teach. This will of course not be possible unless more financial assistance is forthcoming, either in the form of grants or bursaries.

The present training syllabus for health visitors is constantly under review. Any extensive alteration must wait for the revision of the basic training, as it may then be possible to include instruction in psychology and methods of teaching, in addition to a wider study of economic and social conditions. Social life in all its varying phases

and states is of course the background of a health visitor's work: she must have a thorough understanding of social problems, and of the numerous social services, both voluntary and official. There is a tremendous lot of work being done by unseen, unheard and often unpaid social workers in this country and I should like to see it recognized as playing an important part in our health services.

School nursing.—There are many trained nurses employed in school nursing work only, but in an increasing number of cases outside London the work of the health visitor and school nurse is being combined, so as to give continuity of supervision of a child's life from birth to 14 years of age. To-day a school nurse must be general trained and state registered but she is not required to be a qualified midwife or health visitor. This means that her status is often not so high as that of a health visitor. No one will deny the importance of the work done by these school nurses and that they have great opportunities for health education.

I should like to see all health teaching in schools done by expert public health nurses who have demonstrated their ability to teach and have been specially selected and trained for this work.

If a better type of girl is to be attracted to public health nursing, one way of doing this would be to create more posts for superintendents of public health nurses, and even assistant superintendents, and to have such posts filled by experienced public health nurses who are capable of undertaking health teaching in schools, and elsewhere, while at the same time keeping in touch with practical health work.

Midwives.—As a profession almost distinct from nursing, midwifery is marching ahead and with improved salaries and better conditions we can look forward to a service which has no equal the world over. If we are to attract a sufficient number of nurses to midwifery, their chances of promotion must be greater and one way of doing this would be to increase the number of posts for non-medical supervisors. Besides improving the promotional prospects of the profession such appointments would also provide a supervision which would be both acceptable and profitable to the midwives themselves. Many health visitors and midwives would like to see the combined posts for supervisors of midwives and superintendent health visitors abolished. These are certainly two very distinct branches of nursing work and it is possible that many excellent women may be excluded because they have not had the necessary experience in either one branch or the other.

Nurses in industry.—Industrial nursing is only in its infancy but there seems to be an increasing awareness on the part of factory inspectors, and managers, of the value of this work, as evidenced by the rapid rise in the number of nurses both trained and untrained who have taken posts in factories since the outbreak of war. The Factory Act of 1937 mentioned medical supervision for the first time and as the law stands to-day the person in charge of an ambulance room in a factory is required to be "A qualified nurse or other person trained in first aid". Unfortunately this provides a loop-hole in that these posts can be filled by lay men or women who have the necessary first-aid qualifications for dealing with minor injuries yet do not possess the wider knowledge of health and disease which so befits the trained nurse for this work.

Miss E. Cogswell Phillips, B.S., M.A., R.N. (Associate Chief Nurse, in charge of Public Health Nursing American Red Cross—Harvard Field Hospital Unit): American public health nursing dates back only about forty years. The formation of the National Organization for Public Health Nursing in 1912 was a great step forward, for the aim of its members has always been to influence and improve the actual performance of public health nursing services—those services which are increasingly considered to be essential to the further development of the public health movement as a whole.

At the beginning of the present century there were only 130 public health nurses in the United States employed by only 58 agencies. By 1920 the number had increased to 9,000 in more than 4,000 agencies. The last twenty years have seen an increase to nearly 24,000 nurses working in over 6,000 organizations. These increases are encouraging but the development has always been most uneven in the various sections of the country. Furthermore, the standard of one public health nurse to 2,000 population, as set forth by the American Public Health Association, would require 65,000 public health nurses, three times the number we now have. To date this standard has been approached by only one state, Connecticut, while in another state, Oklahoma, the ratio is only one public health nurse per 16,500 population. At the present time our federal government is attempting by means of publicity and subsidies to the states, to add 10,000 new public health nurses to the field next year.

"Study of Nursing and Nursing Education in the United States", commonly known as the Rockefeller or Goldmark Report published in 1923, had a great influence upon the basic preparation of the public health nurse since her fundamental education is exactly

the same as that of the nurse in any other field of activity. To-day the basic courses in our better schools of nursing comprise experience in medical, surgical, obstetric, gynecologic, psychiatric, and pædiatric nursing, including the care of children with orthopædic and cardiac conditions as well as the care of the well child through experience in nursery schools whenever possible. An attempt is made too, to develop in the student a deep appreciation of the social and health aspects of nursing, both physical and mental, through an integrated programme of study in the classroom, wards, and out-patient department. It is upon such a foundation that the specialization of public health nursing is built after the nurse leaves the hospital.

"The Report on Municipal Health Practice in Eighty-Three Cities", known as the Winslow Report published in 1924, had a direct influence on the standards of preparation of the public health nurse, since it began to define her functions and outline her part in public health programmes.

The lack of hospital facilities in many sections of our country, particularly in rural districts, makes it imperative that sufficient public health nurses be available everywhere, and that they give bedside nursing care in addition to assisting in the control of communicable disease and in health education. In total war this need is even more important. Because there were still many hundred localities that had no such service last year, the Emergency Health and Sanitation Act of 1941 provided for the appointment of public health nurses by the United States Public Health Service. These nurses are assigned to State departments of health, which in turn reassign them to local areas. By January 1942 over 500 nurses had been requested but only 151 had been appointed.

In July 1941 Congress appropriated \$1,250,000 (approximately £312,000) for the expansion of the number of all types of nursing personnel, and present indications are that for the fiscal year beginning next July, the amount will be \$2,000,000 (or about £500,000). About three-quarters of this fund is being devoted to the expansion of the basic undergraduate courses for all nurses, and the rest is being used for post-graduate preparation for the nursing specialties, of which public health nursing is one. A typical plan for the training of public health nurses is as follows:

Graduates of approved schools of nursing who qualify for admission to a university course are eligible. They must have had no previous preparation or experience in public health. Four months, or one university term, is spent in a combined programme of college and field work which covers the first stage of the regular preparation of the public health nurse. The lectures are held in the university, and field work is done under the direction of a well-supervised public health nursing organization. An allowance of \$450 (or about £112) to cover tuition and subsistence is made. In return for this the nurse agrees to serve for at least two years as a junior public health nurse in any agency in the country specified by the United States Public Health Service. She also promises to complete within the next five years at least a year of university study in public health nursing. All courses satisfactorily completed, including field work, carry university credit and are counted toward the major requirements for a degree in science.

In the United States there are four kinds of organizations under which the public health nurse works: the official or tax-supported health agencies, the non-official or voluntary agencies, boards of education and commercial companies, both industrial and insurance. All four types are found in most districts although the official organizations play a more universal role. Because of our governmental organizations there can never be a federal programme applicable to the country as a whole. Each state now has its department of health but the core of the public health system will probably always be the smaller local units, the towns and counties, or a combination of them. On January 1, 1941, approximately only 4% of the 23,533 public health nurses were employed in state agencies, while 41% were in local rural or urban health departments. Non-official local agencies employed 25% of the total, while boards of education and industrial companies each had about 15%. The most rapid recent expansion in the employment of public health nurses has been in the factories, the number having increased by well over one-third in the past five years.

An all-over community health plan necessitates joint planning by all the agencies concerned. The tax-supported agency with its health officer will be the leader, but the voluntary organization must participate in the planning as well as in the execution of the programme. Nor must we stop with the public health agencies; the hospitals and other social organizations must join in. It has been demonstrated repeatedly that through a combination of agencies a higher standard for all public health services can be assured, and that the cost to the community will be less.

Such a plan for the effective combination of organizations usually accepts the standard of not more than two health agencies administering public health nursing in an area, one under official auspices and the other privately financed and administered. This

division of responsibility recognizes the traditional limitations on the use of public money and leaves the private agency free to undertake new services that will round out the community programme and to train personnel in ways that the official agency may not be able to do.

Until recently it was customary for public health nurses, whether in the employ of official or voluntary organizations, to be attached to specialized health programmes, and it was not unusual for a single family to have the services of several. This system was wasteful of time, money and energy of both family and community, but even after the recognition of this fact it was continued because we felt that one nurse could not be a specialist in all phases of public health. Now, however, we see that the proper place for the specialist is in child hygiene, communicable disease, nutrition, tuberculosis, cancer and so forth is on the consultant or supervisory level, and that if the staff nurse has the benefit of their direction and experience she can give safer and more than adequate nursing care to the whole family. We are now beginning to treat the family as a real social unit for health work and find it is most successful from all points of view.

Our public health nurse works only under the direction of a physician. He may be the private doctor of the patient or he may be in the employ of the official health department, the hospital, school or factory, but he alone can be responsible for the care of the patient.

Public health nursing has been defined as "an organized service (not for profit) rendered by graduate nurses to the individual, the family and the community". Such a service includes the interpretation and the application of medical, nursing, sanitary and social procedures for the prevention of disease and the promotion of health. Often it involves the bedside care of the ill in their own homes. Primarily the public health nurse is an educator and this function of teaching underlies all of her activities in every phase of public health work in the home, the clinic, the factory and the school. Even when she is engaged in helping to evaluate a part of the public health programme which is directed towards a particular end, such as diphtheria immunization, or when she is working with physicians in the investigation of some public health problem involving a special situation, such as an epidemic, this teaching function remains of paramount importance.

In America the advanced preparation for public health nursing has always been through universities, but the National Organization for Public Health Nursing accredits such programmes of study. At the present time there are twenty-eight which meet with its approval. Many private scholarships, fellowships and loan funds have been set up during the past fifteen years to help nurses pay for their university education, and in addition in 1935, and again in 1941, federal funds were made available for this purpose. Employing agencies have been most generous in allowing members of their staffs leaves of absence to pursue full-time study. By January 1941 62% of all the supervising nurses in the country had completed one or more years of public health education, and over one-third of them held university degrees. The percentage among the staff nurses was not as high, only 21% having completed one or more years of advanced specialized study, and 19% possessing degrees. However, an additional 37% of them, and 26% of the supervisory group, were in the process of completing a year's study, so that the next time the data are compiled there should be a decided advancement.

Probably never has a profession developed more quickly than public health nursing. Many aspects of it are still in a state of flux, yet certain definite trends in its procedure and thought are emerging.

(1) Bedside nursing is becoming a part of many programmes where it has never been before. (2) The public health nurse now accompanies her verbal teaching by a great deal of practical demonstration, no longer does she "just talk". (3) The family is being regarded as a unit for health care and instruction, and the nurse who visits it does so in a generalized capacity, although her organization makes provision for her to have the advice and direction of many specialists. (4) The nurse in the school is working more with teachers than directly with children in order that a concept of healthful living may permeate throughout the entire school curriculum. (5) Those years of life which in the past have received far too little attention from health workers—the preschool period, adolescence and senescence—are being included in public health nursing programmes. (6) The care of the chronically ill, both adults and children, is being augmented and improved. (7) Emphasis is being placed on those aspects of the public health problem which involve a great degree of nursing skill, such as in the care of the premature, the child with cardiac disease and the patient with pneumonia. (8) Industry is utilizing the service of the public health nurse, not only in the factory but in the homes of its employees to care for their families as well.

To my mind even more encouraging signs are seen in the interest and help that the

leaders of public health nursing are giving in the recruitment of the right kind of girls to be students in our schools of nursing, and in the provision of richer undergraduate and post-graduate experiences.

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Miss I. Hawkins (New Zealand): As I have been away from New Zealand for five years I can only give you a few scattered pictures of public health life rather than details of training and development of the Service.

New Zealand is a very new country. We started with all your experience behind us. There could be few problems for us that you had not already faced and dealt with, and put on record in the history of nursing, and this springboard, together with the friendly co-operation and help given to us by almost every country in the world, and particularly by our mother country, has made our task a good deal easier.

In all New Zealand there are only about 3,000,000 people, which means that we have no real slums. There are the poorer areas attached to all big cities, and we have the ever-growing potential slums to fight against; but we did not start our conscious public health life with them.

Against that there is the problem of large areas with only a sprinkled population; the problem of how to cover those areas for public health services most economically and efficiently. There are many branches of public health nursing now, the district nurse, the school nurse, the industrial nurse, the infant welfare nurse, the tuberculosis nurse, &c., but it is not practicable to send four or five people long distances into the country to see two isolated families, when one nurse with a good all-round training and common sense could do what is required.

One specialized public health service of the country, a service that over a period of thirty-odd years has had a most dramatic influence on the health of the people, and has increased their expectation of life, is the Plunkett nursing service founded by Sir Truby King.

The service as it stands now consists of two branches; first the Karitane nurses who are trained to look after babies from two weeks to school age in the home; and second the Plunkett nurses, who having done their general training and usually midwifery as well, take a post-graduate course. There are, I think, about 10 Karitane hospitals scattered over the Dominion, but only one of these is a centre for the training of Plunkett nurses, and until the increase of population makes it inadequate, it is a good thing, because it does give a uniform standard of training. Of the Plunkett nurses, some return to their own jobs with the additional knowledge to help them; others are chosen to staff the Karitane hospitals, to teach mothercraft and train the Karitane nurses; others do district work, and here, though it would be a poor thing if a certain amount of individuality did not creep into the work of a district nurse, it is appreciated that the basic training of every Plunkett nurse in the Dominion is the same, and that a mother with a small baby moving from the north of the North Island to the south of the South Island can feel safely confident that she will get the same advice and guidance that she is used to.

The Karitane hospitals are not elaborately equipped with complicated apparatus and expensive labour-saving devices, but deliberately made to approximate the average home as closely and as attractively as possible, so that both the mother and the nurse can put into practice at home what they have learned working under the same conditions. No methods or equipment are used in hospital that cannot be translated to the poorest home.

An interesting factor in the development of public health nursing in New Zealand has been the native population. The Maoris are a very fine race both in physique and intelligence. They share our schools and universities and our hospitals. Before we came they lived an open-air life, killed their own food and were a strong, virile people. With civilization came new diseases to which they were very susceptible. Their teeth, once

perfect, began to decay. Most serious of all, tuberculosis spread among them, and the Maori population started to fall. Then we realized our responsibility and tried to teach them how to live healthily under our conditions, with the result that the Maori population to-day is increasing, and is increasingly fit. Public health nurses work among them who speak their language and know their customs, but it is extraordinarily difficult to teach hygiene to a superstitious people who may have a deeply religious reason for doing something most revoltingly unhygienic.

Many of them still look on hospitals with suspicion, as places to die in, and though in time results will be convincing enough to penetrate their reason, one cannot wait for that penetration before admitting a case of acute diphtheria. At one time if a Maori was going to die in hospital, say of tuberculosis, and the relatives were notified, they would come 10 to 30 strong to take her home over miles of rough country roads to die among her own people. In the same way, if a case of typhoid was to be admitted from a country district, one automatically got at least 20 beds ready. The patient would arrive closely surrounded by all his friends and relations, in a buggy or truck, and they sooner or later would probably all be admitted themselves.

This may sound foolish to you. The obvious criticism is: "Why did you let them take home a girl dying of tuberculosis to infect the rest of the family? Why not send an ambulance to take the case of typhoid into hospital?" The answer is that by ordering them to do something they could not understand, perhaps to violate a tribal custom, we should have frightened and antagonized them, thus undoing the education and progress of 20 years by one act. Surely it was better to let them play our game their way until we could convince them that our way was best, even at the risk of spreading infection. In each of these cases the public health nurse could be sent to follow up the contacts—and could do it. But if we had been highhanded she would have lost the right of entry into their homes, and with it the privilege of helping them. In time their mistrust and fear, which is only a fear of the unknown, will disappear completely.

Miss Eleanor Jones, N/S (15th Canadian General Hospital, R.C.A.M.C., C.A. Overseas): In Canada three types of training for public health nursing are offered by recognized universities. There is the one year post-graduate course, the five-year course, leading to a degree in science, and the three-year undergraduate course in public health nursing which is offered by the School of Nursing of the University of Toronto.

The School of Nursing was founded in 1933, under the directorship of Miss Kathleen Russell.

The purpose of this new course was to provide a direct training in public health nursing. Previously, the training had been divided into two distinct parts, the three years spent in the hospital training, and the one year post-graduate training in public health. This new course combines those two parts and fuses them together into one well integrated whole.

The qualifications for entrance to the course are: Completion of secondary school education with a good background in sciences, good health, and a minimum age of 19. In an attempt to attract a better type of student, preference is given to those over 20, and with further education.

The first three months of the first year are spent in the classroom and the laboratory, and include an extensive course in sciences. The fundamentals of preventive medicine are studied, and a forty-hour laboratory course in infection and immunity is given. The principles of nursing and of public health nursing are taught, and the student is given the opportunity of observing the health work done in the community.

With this foundation, the student proceeds to various hospitals for her general training in bedside nursing. Experience is acquired in those clinical services of most value to the future public health nurse, such as obstetrics, paediatrics, communicable diseases, psychiatry, and tuberculosis. Care is taken to protect the quality and quantity of work done in the hospitals, and constant supervision is given. Lectures and laboratory work continue throughout the hospital training, and the student nurse is constantly being brought in contact with the health work of the community through various agencies.

In the third year the senior course in public health nursing is given. This includes lectures and seminars covering such subjects as the principles and practice of public health nursing, the principles of teaching, social case work, and an intensive course in preventive medicine.

Approximately four months are devoted to field work with various public health agencies during this year. These include a municipal department of health, which does generalized public health work, a visiting nursing organization similar to the Queen's nurses, which does bedside nursing and health teaching, and the provincial Red Cross Society, which does generalized public health nursing in rural communities. Field

work is done with a family case work agency. The staff nurses of these organizations supervise and teach the student nurses.

The course is thirty-nine months long, and meets all the State-registration requirements. On graduation the nurses receive diplomas which qualify them to practise both general nursing and public health nursing. They write the State-registration examinations.

This course was started as an experiment, and some of the funds for financing it were given by the Rockefeller Foundation. Although six classes have graduated to date, whose members are holding responsible positions in various public health agencies throughout Canada, it must still be considered an experiment, as it is constantly changing to meet changing demands. It is too soon to predict just what effect it will have on the future of public health nursing education.

Dr. Nicolas Gebbie (M.O.H. City of Kingston-upon-Hull): In the systematic development of the public health service in this country the health visitor, the school nurse and the tuberculosis visitor have played an important part and are destined to make an equally important contribution in the future.

We shall all agree with Prof. Chas. McNeil when he says: "The potential of service that can be rendered to the people by trained health nurses is immense."

(1) *The public health nurse.*—The forerunner of the public health nurse as we know her to-day was the female sanitary inspector. In the days when environmental hygiene was the chief concern of the public health department and when little emphasis was placed on personal or individual hygiene the scope of activity of the female sanitary inspector was necessarily limited. Her pioneer work in the supervision of female outworkers and in the investigation of working conditions of women and girls in factories must always be recognized. It was not until measures of personal and social hygiene were begun, e.g. school medical work and maternity and child welfare, that the importance of nursing qualifications and experience was realized.

(2) *The work of the public health nurse.*—I need not recount the many and varied activities of the nurses in a modern public health department. The scope of the work of the nurses is ever widening and is likely to continue to do so, e.g. recent Scabies Order.

Home visiting is and must remain the most important duty of the public health nurse. In the early days these visits were resented by the mothers and it says much for the tact and ability of the pioneers that in a comparatively short time the house doors have been thrown wide open and the nurse has become a welcome visitor not only to the house but to the home.

The prams at the cottage doors in the summer sunshine are a lasting memorial to the work of the health visitors. The knock-knees and bow-legs of rickets are now practically limited to adults—unlucky enough to be born before the days of the health visitor.

The school nurse, too, relentlessly pursues her campaign to improve the cleanliness of the school children and has been affectionately dubbed "the nit nurse" in the process. Improvement in the standard of cleanliness of the school population has been most marked, but the revelations during the mass evacuation remind us that our task is not yet completed.

The tuberculosis visitor has also made her contribution to the betterment of the social welfare of her patients and their families. The part she has played in after-care work has to be experienced to be appreciated.

The midwife, as a municipal officer, is of much more recent date than her colleagues. When the bill which became the Midwives Act, 1936, was being considered by the House of Commons, the then Minister of Health stated: "The midwife in future will be a member of our organized service. She will be an integral part of the public health service and no longer a lone figure in the campaign against maternal mortality".

(3) *Training for public health work.*—Before commencing her specialized training the student public health nurse should have had a sound training in nursing. Her appreciation of the social and domestic repercussions of the commoner ailments, some of which may cause long periods of disability, will be heightened if her training has included the nursing of chronic cases.

The public health nurse, whether she be health visitor, school nurse, or tuberculosis visitor, is essentially a teacher. Her success in her profession will be measured by her skill as an educator of the public in hygiene. The student public health nurse should in my view, have reached a standard of education comparable with that demanded of entrants to the teaching profession in our elementary schools.

Scholarships should be available to permit of extra places for students who cannot afford to take their training. In such cases the capitation grant (£15) should remain.

The course of training should be of not less than twelve months' duration and should be eminently practical.

It should aim at preparation for interavailability. Interavailability is of great importance, and with properly qualified, trained and experienced nurses it works well in practice.

The visitor can maintain a closer personal touch with the people in her smaller area. She has more scope for her teaching activities and can render greater services to those under her care. The school child, the adolescent and the adult are just babies of a larger growth. Duplication of visits by different nurses to the homes is avoided. The health visitor, the school nurse and the tuberculosis nurse should be one and the same person and should possess the health visitors' certificate. What about the municipal midwife?

In "Public Health and Social Services" by Dr. Geffen and Mr. Farrer Brown emphasis is laid on the part to be played by the midwife as a member of the public health team. "If the midwife is to use to the best advantage of her patients her opportunities to act as an educator and adviser on health matters, she must know what are the various branches of the public health and social services, the facilities provided and the appropriate person or authority to approach in any particular case. It is with a view to equipping midwives to make the most of these opportunities that the Central Midwives Board has required in the latest rules that pupil midwives shall receive instruction regarding the public health and social services."

In the training of the public health nurse emphasis should be laid upon personal hygiene and social welfare. In the foreword to the book by the Ling Physical Education Association and entitled "A Modern Approach to Health Education" (published 1941 by the University of London Press) Dr. Gamlin, Chief Assistant School Medical Officer of Liverpool, writes: "The provision of more hospitals and clinics, the appointment of more doctors, nurses and health visitors would produce no spectacular improvements in the health and physical efficiency of our citizens until they recognize that the practice of individual hygiene is not only a duty but also a national necessity." The well-trained and experienced public health nurse will play a big part in securing the recognition by the people of the importance of personal hygiene.

(4) *What of the future.*—The public health nurse must continue to carry the teaching of positive health right into the homes of the people. She will be the adviser and educator upon general hygiene, both personal and environmental, upon mothercraft and baby care, upon the health of the school child, the adolescent, the adult and the aged. This war has brought about an increase in communal living but the pendulum will swing back towards the individual home as the unit and towards all that that means to the British people. The standard of cleanliness of our people must be raised and the public health nurse will, I believe, succeed in raising it. Public health nursing must be made attractive to the best in the nursing profession. The remuneration must be adequate to ensure comfort and happiness in her work and security and contentment in her retirement.

In the Report of the Joint Consultative Committee of Institutions recognized by the Minister of Health for the training of Health Visitors and of Organizations of Health Visitors, 1928-1937, there are the following conclusions:

"Of all the factors considered in this inquiry the salary scale is that to which most importance is attached by candidates for appointments. With few exceptions the number of applications received by individual local authorities in reply to their advertisements and the frequency with which they are obliged to advertise, varies directly with the salary scale offered. Where the minimum of the scale is more than £200 combined with a maximum of more than £250 there is no shortage of health visitors."

As regards age at retirement, 60 is old enough for anyone to be cycling round in all weathers.

The conditions of employment must be so arranged as to permit of ample relaxation and recreation and to include facilities for attendance at refresher courses, including instruction in the teaching of hygiene. The amenities and rewards of the public health nurse must be commensurate with the importance of her work to the nation.

Section of Surgery

President—E. ROCK CARLING, F.R.C.S.

[January 7, 1942, *continued*]

Endothoracic Sympathectomy

By Squadron Leader JOHN HUGHES, R.A.F.V.R.

Endoscopy has in recent times become not only a well established, but also an extremely valuable branch of Surgery. Though primarily used as a method of diagnosis, by improvements in equipment and technique, it is now also possible by this means to perform operative measures of considerable magnitude.

The thoracic sympathetic nervous system may reasonably be regarded as one of the more inaccessible parts of the body, but viewed from within the pleural cavity, the sympathetic chain and splanchnic nerves are only separated from this cavity by the parietal pleura. The object of this paper is to describe the successful resection of the stellate ganglion and splanchnic nerves by an endoscopic technique. It is not intended to assess the value or otherwise of this treatment for the clinical conditions described.

The possibility of section of the splanchnics and excision of the stellate ganglion, via the thoracoscope was suggested during operations on the lungs. In the majority of such cases the roots of the great splanchnic nerve could be clearly seen glistening through the pleura, thus giving a direct guide to the position of the nerve, the position of the stellate ganglion was seen during operations for internal pneumolysis and gave support to the suggestion that such an operative approach was feasible.

Trial was made on the cadaver, and it became obvious that the simplest method would be to work with two cannulae, one for the thoracoscope and the other for the passage of instruments. It was also obvious that it would be convenient to have two instruments that could be passed through the same cannula at the same time, one being a plain pair of forceps and the other a minute pair of scissors. These instruments were specially made, and proved sufficiently strong and satisfactory. Together with a small blunt hook, they are all that are required for use with the standard thoracoscope. It was also found on the cadaver, that the pleura could be picked up and cut with the forceps and scissors, thus exposing the underlying structures.

TECHNIQUE

Both for the excision of the stellate ganglion and section of the splanchnic nerves, the preliminary essential is a complete pneumothorax on the side of the operation, induced over a period of some days, and checked by radiography. The operation for resection of the splanchnics is performed with the patient prone, and for the excision of the ganglion in a half-sitting posture. The position of the lung during each operation can be thus foretold.

For the splanchnic resection the patient is placed face downwards on the operating table and the thoracic cannulae inserted under local anaesthesia between the 7th and 8th, and 8th and 9th ribs in the mid-axillary line. Using the upper cannula for viewing and the lower one for operating, the pleura is anaesthetized with 2% novocain by means of the long standard endoscopic syringe. An excellent view of the bodies of the vertebrae is obtained, and the diaphragm remains well down and the lung falls out of the way. The area of the parietal pleura which is infiltrated should cover the space of three vertebral bodies and also extend outwards to the necks of the ribs. It is interesting to note that the sensitivity of the pleura seems to diminish in a forward direction, so that while the pleura over the neck of the ribs is highly sensitive, the part which is reflected on to the mediastinum appears to be almost insensitive.

After infiltration, an assistant holds the viewing cannula and thoracoscope steady, so that both hands of the operator are free to manipulate the instruments through the operating cannula. The pleura is picked up at a convenient spot as low down as possible and cut with the scissors from the neck of the ribs to the front of the vertebræ. The pleura is elastic, and it is quite easy to pick it up and clean, without danger of damaging other structures.

The scissors are now removed, and the blunt hook inserted. With the forceps holding the pleura at the anterior end of the incision out of the way, the hook is passed towards that part of the vertebral body where it passes out of sight, and with a little gentle manipulation it is passed under the great splanchnic. The nerve is quite unmistakable when seen, as it can be drawn well forward and will spring back again like a piece of elastic. When seen through the thoracoscope it appears to be about the size of the median nerve at the wrist. The nerve is then grasped with the forceps and cut in two places as far apart as possible with the scissors. By gentle dissection at the side of the vertebral body, the sympathetic chain can be cut in a similar manner.

For resection of the stellate ganglion, the patient reclines on the table so that the chest is at an angle of 30° degrees with the horizontal. In this position the lung falls downwards and backwards, leaving a good space in the region of the upper ribs. The cannulae are inserted under local anæsthesia from the front this time, the viewing cannula between the 1st and 2nd ribs, and the operating cannula between the 2nd and 3rd, each being inserted about two inches outside the lateral border of the sternum. Using both the direct and oblique vision thorascopes, an excellent view of the dome of the pleura is obtained, and the line of the great vessels can be plainly seen, the veins, including the intercostals being particularly prominent. The pleura is infiltrated with local anæsthesia from above the neck of the 1st rib to the 3rd, and cut from below, upwards. The neck of the 1st and 2nd ribs are thus exposed. This area is then cleaned by careful dissection with the scissors and forceps, and the tissue removed kept for section. In one of the cases operated upon, the sympathetic ganglion was definitely seen, and its removal thus simplified; in the other it could not be identified with certainty, and a more extensive dissection had to be carried out. In both these operations as described above, the bleeding is negligible, and consists only of a very slight ooze from the minute vessels behind the pleura, which can be swabbed away by a small pledget of gauze held in the forceps.

Omnopon gr. $\frac{1}{3}$ and scopolamine gr. $\frac{1}{150}$ were used as premedication in four operations, the fifth receiving morphia gr. $\frac{1}{4}$ only.

Summary of cases.—The operation of sympathectomy by the endothoracic route was performed on four patients. In one case the procedure was carried out on both sides. It has thus been done on five occasions. Two of these cases had hypertension, in addition to which, one of them had coronary occlusion. One was a case of persistently painful amputation stump, and the other was a case of Raynaud's disease. The operations were all performed between 3.3.39 and 19.8.39. A short account of two cases is given.

Mrs. C., aged 39, admitted to Sheffield Royal Infirmary on 27.2.39 on account of hypertension. Blood-pressure 238/140. Induction of artificial pneumothorax on the right side was commenced on the day following admission, and was complete by 3.3.39. The right great splanchnic was resected by the method described above. On the same day blood-pressure half an hour after the operation was 140/108 rising to 210/140 on the following day. It remained at this level until her discharge from hospital on 8.3.39. She was readmitted to Sheffield Royal Infirmary on 20.3.39 for a corresponding operation on the left side. This was carried out on 21.3.39, part of the sympathetic chain being removed at the same time. The stellate ganglion was resected on the right side only: the patient gave an excellent clinical result, with alteration of thermal and other conditions in the arm associated with section of the sympathetic supply. On admission the blood-pressure was 230/150, and on discharge on 6.4.39 was 216/136. Sections of the tissue excised were reported upon as follows: "Large non-medullated nerve." In this case there were no pleural adhesions, both lungs collapsed well, and from a technical point of view it was most encouraging.

Mr. C., aged 51, admitted to Sheffield Royal Infirmary 16.7.39. The right arm had been amputated just below the shoulder-joint, nine years previously for osteomyelitis of humerus. Two years ago pain commenced in the stump. A terminal neuroma had been removed with partial relief of the symptoms, but these had again become severe during the last three months. On 18.7.39 the stump was injected with novocain and on 21.7.39 another terminal neuroma was dissected out, with but little relief. It was then suggested by Professor Ernest Finch that removal of the stellate ganglion might be beneficial.

Artificial pneumothorax was commenced on 9.8.39 and completed 18.8.39, when an excellent collapse was obtained. Endothoracic excision of the stellate ganglion was carried out on 19.8.39. The sympathetic chain in this case was identified behind the pleura. Histological examination of the tissue removed confirmed the presence of sympathetic ganglion cells.

There was immediate cessation of symptoms after the operation, and the patient left the hospital free from pain. It was interesting to note that while the novocain was active at the site of the operation, the patient exhibited contraction of the pupil on the corresponding side, which disappeared in about two hours, but there was no permanent inequality of the pupils.

By using this method it would incidentally be perfectly feasible to inject the sympathetic with alcohol under direct vision, and if the pleura were allowed to heal, before the pneumothorax absorbed, there seems to be no reason why the procedure should not be repeated indefinitely.

All the cases were operated upon at the Sheffield Royal Infirmary, and I wish to express my gratitude to the physicians and surgeons under whose care they were admitted.

[March 4, 1942]

DISCUSSION ON GROWTH AND NEW GROWTH

Mr. Harold Burrows: *The influence of œstrogens on cellular and organic growth.*—

When discussing stimulators and regulators of tissue growth, hormones derived from the testis and ovary are worth attention. Their composition is known, they are available in pure form for experiment, and examination of their activities has thrown light on some of the very many factors which govern cellular multiplication in vertebrates. Although the influence of these hormones is exerted mainly on the reproductive system, using this term in its widest sense, the nature and boundaries of their influence are not clearly marked.

The effects of œstrogen on the growth of tissues may be classified into three groups, namely: (1) Controlled growth; (2) uncontrolled growth or cancer; (3) imperfectly controlled growth. In the last group are included fibromyomata of the uterus, ovarian cysts, chromophobe swellings of the pituitary and some other innocent tumours.

For brevity the subject of imperfectly controlled growth will not receive detailed attention in the following remarks.

(1) CONTROLLED GROWTH.

(i) *Prevention and arrest of growth.*—Though a prominent function of œstrogen is to stimulate cellular multiplication, in certain tissues it can also exert the opposite effect. For example, œstrogen arrests the growth of epiphyseal cartilage, causing bony union between the epiphysis and shaft, which largely explains why women are smaller than men. The mammary ducts for their development, and perhaps for their existence, depend on œstrogen, yet under excessive doses the ducts will remain stunted. Another way in which œstrogen may hinder the growth of particular tissues is by checking the production of androgen and progesterone or by directly counteracting their growth-stimulating effects.

(ii) *Stimulation of growth.*—(a) *Cellular differentiation and function:* A feature of normal tissue development in response to œstrogen is its adaptation to a purpose, in attaining which cellular multiplication, differentiation and function follow each other in orderly succession and appropriate degree.

(b) *Specificity of action:* Œstrogens apparently are not general stimulators of cell proliferation. Their action is mainly specific and is confined almost entirely to structures concerned, directly or indirectly, with sexual reproduction. This specificity is independent of the sex of the individual; homologous structures in the two sexes respond alike. It may be remembered that the formation of œstrogen is not confined to the female; males also elaborate and utilize the hormone.

(c) *Responsive and irresponsive tissues:* From what has just been said about specific action, it follows that different cells and tissues vary in their capacity to respond to œstrogen. The degree and quality of this responsiveness is innate in each individual cell or group of cells, is unaffected by neighbouring cells, and is retained after transplantation into another part of the body or into another individual, whether of the same or of the opposite sex.

(d) *Gradients of reactivity:* The readiness with which different organs respond to œstrogen shows wide divergences, and the threshold of reactivity may vary through a large range within the extent of a single organ.

(e) *Reversible and irreversible effects:* Many of the normal effects of œstrogen are reversible and are maintained only while the hormone is supplied in adequate amount. Other effects are permanent, notably some of those seen in the course of embryological development. Mammary cancer is another kind of irreversible change induced by œstrogen.

(f) *Adaptational resistance to œstrogen:* With continued exposure to œstrogen the individual may develop some degree of resistance to its action. This resistance is not attributable to the formation of antihormones.

(g) *Inactivation and excretion:* The liver seems to be the chief inactivator of œstrogen the waste products of which are excreted in the urine and perhaps also in the bile; and hepatic disorder has been shown by experiment to accentuate the effects of artificially administered œstrogen.

(h) *Pituitary gonadal relationship:* The production of œstrogen in the body is normally controlled by the pituitary. Œstrogen inhibits the output of gonadotrophin by the pituitary and so automatically checks its own production. Other gonadal hormones, including testosterone, restrain the production of œstrogen by stopping the supply of gonadotrophin from the pituitary.

(i) *Mutual antagonisms between different gonadal hormones*: Either by a direct counteraction, or by indirect influence through the pituitary, the growth stimulating effect of oestrogen on certain tissues may be prevented by other gonadal hormones, notably by androgens.

(j) *Co-operation between gonadal hormones*: On the other hand the co-operation of hormones may be required for the full development of a particular tissue. The co-operation takes place in one of two ways: either the hormones act simultaneously, or an organ must first be subjected to one of the hormones before the other can be effective.

(k) *The effect of a given dose of oestrogen* in an individual will be governed by many circumstances other than those already mentioned, including species, age, season, general health, diet and so forth.

It will be unnecessary to discuss every one of these just now. Perhaps it may be worth while to give an example of variation of response in different species. Fibromyomata of the uterus are readily induced in guinea-pigs by oestrogens but not as a rule in rats and mice.

(2) UNCONTROLLED GROWTH

(i) *Non-specific neoplasia*.—Apparently oestrogens have some power to induce cancer in tissues which are beyond the range of their normal specific activities, for sarcomas occasionally arise in rats and mice at the sites of oestrogen injection.

(ii) *Specific neoplasia*.—Apart from these relatively unimportant instances the cancers induced by oestrogen are confined to those organs which are under the normal physiological influence of oestrogen. In this list may be included cancer of the breast, uterus, testis and bones. Among them, malignant tumours of the breast have been intensively studied in mice. The knowledge gained by this study may perhaps be applied to the investigation of other forms of cancer. An important fact concerning malignant disease of the breast is that, although a response to oestrogenic stimulation, it will not occur unless certain additional factors are present. Chief among these are two which are inherited, genic and non-genic.

(a) *The genic factor* is inherited as a Mendelian dominant, and may be defined loosely as a susceptibility to mammary cancer in the presence of the other two main factors.

(b) *The non-genic factor* is not carried by the chromosomes but is transmitted to the infant in the mother's milk. The factor can also be conveyed to the young by implantation of pieces of the mother's tissues. It seems probable that the transmissible agent is a virus; the liability to mammary cancer which it carries is handed on to subsequent generations and therefore, it appears, the agent must multiply in the body.

(c) *Subsidiary factors*.—Certain additional factors play a part in mammary carcinogenesis. These include diet and general health. By underfeeding mice on a diet that is adequate in quality, or by keeping mice on diets which are ample in quantity but deficient in certain qualities the incidence of mammary cancer can be reduced.

Perhaps some of the facts which have been mentioned and others concerned with the influence of oestrogen on tissue growth may be of interest in connexion with the stimulation of growth by carcinogens. If analogy may justify an opinion, it seems that the different forms of cancer which occur in the body might well be regarded as so many distinct disorders, each having its own special aetiology.

Another conclusion which seems justified is that tissue growth, whether controlled or uncontrolled, is regulated in vertebrates by a multiplicity of factors.

Dr. W. E. Gye: The capacity to grow and divide is a property of normal cells which lasts throughout life; an appropriate stimulus must be provided. Growth of normal cells and malignant new growth are different in a fundamental way; in malignant cells the stimulus to growth is within the cell itself and growth is not subject to the forces which control normal growth. These statements, drawn from the work of pathological study of human cancer and in particular of metastases, have been confirmed and amplified by forty years of experimental studies. Hence the popular attempts to cure cancer with extracts of glands and other tissues which are supposed to provide growth-controlling substances are based on misconceptions of the nature of cancer. There are, broadly speaking, two working hypotheses concerning the intracellular cause of the autonomous growth of cancer: (1) That the cancer cell is a mutant of the normal cell; (2) that the stimulus to growth in cancer is an intracellular virus. There is no factual evidence for the first hypothesis; the second hypothesis is supported by the fact that some new growths, epithelial and connective tissue tumours, are known to be caused by a virus.

Mr. L. R. Broster : Growth.—Tissue growth and repair is the foundation of surgery, and we have been inclined to rely too much on Ambroise Paré's dictum, "I dressed him and God healed him", without looking towards its more obscure origins. What the stimulus to grow is—we do not know. As one of the *basic instincts*, we have to accept it like an axiom of Euclid—that it is an inherent property of living matter, and that it originates within the organism.

Phasic.—Growth is a dynamic and variable affair, intensely active during embryonic life, it continues rapidly after birth, bursts into activity again at puberty with the development of the secondary sex characters, and, maintaining a uniform level after the second decade, becomes dormant with age. It is a curious biological anomaly that with the waning of this normal growth process should correspond the rising incidence of abnormal or neoplastic tissue growth. It would seem therefore that we must look for some common biological denominator which determines growth, and from my own clinical experience of abnormal growth, I am led to the belief that it is primarily of a *homogenetic nature*. This fortunately forms a line of study which is amenable to scientific investigation, and for this reason I propose to explore certain avenues of research which tend to confirm this assumption.

Adreno-genital syndrome.—Starting with our own work on the adreno-genital syndrome we have learnt that the masculinization of the female and the feminization of the male, are associated with a specific stain—the *Ponceau Fuchsin Stain*, discovered by Vines—in which the granules of adreno-cortical cells assume a vivid red colour, in contradistinction to the blue colour found in normal people. Backed by such a standard of comparison, my colleague, Dr. Jocelyn Patterson, has shown that both these conditions are associated with alterations in *steroid metabolism*. In virilism the urinary steroids are increased and in feminism they are decreased. By unilateral adrenalectomy the pre-operative level of steroid is diminished in both, and this is associated clinically by a tendency of these individuals to revert to the type of growth normal to their sex.

There are in both these conditions: (1) alterations in general skeletal growth and in special tissue growth involving mainly the secondary sex characters and cutaneous surfaces; and (2) disturbances in sex function, which somehow cannot be divorced from these growth factors.

Fœtal growth.—Now it is obviously an anomaly that such abnormal growth as we have discussed should break out for no apparent reason in persons who were of normal stature previously. Consequently we reverted to the study of the *fœtus*, and Vines was able to show that the same fuchsin stain is present in the cortical cells of the fetal adrenal in both sexes. It represents a transient "*male phase*" roughly between the 8th and 20th fœtal weeks, stronger, more marked, and of slightly longer duration in the male than in the female. It seems reasonable therefore to correlate this event with the future outcrop of adult virilism. This "*male phase*" seems to mark the active functional integration of the fœtal endocrine system.

Experimental embryology.—I must now take you into another channel—that fascinating work on experimental embryology carried out by Needham at Cambridge and by Ross Harrison and others at Yale. Briefly, if the left limb bud of a frog's embryo is amputated, and the right limb bud of another embryo is grafted on to it, the host embryo will force that alien right leg to develop into a perfectly normal left fore-limb. This will only happen up to a certain time, after which the host embryo loses this power, and the transplanted limb bud will grow according to its origin. Already at this early stage there is a diminution in the growth stimulus, but it shows that this growth-compelling power is inherent in the embryo, and that one group of cells possesses the power to regulate and determine the development of other groups of cells.

These workers conclude that the organizing ability is biochemical in nature—a *hormone* in essence, or a *morphogenetic hormone* allied to the steroids and vitamin D.

Early in 1936, I performed an adrenalectomy upon a woman (L. S.) who had had amenorrhœa for two years. She subsequently married, and we were fortunate in catching her by means of a positive Aschheim-Zondek test in the 8th week of pregnancy, when she contained excess of steroid. She was one of the exceptions to our series in that on the old biological test we could find no free male hormone either before or after her adrenalectomy.

It is obvious then that if the growth-organizing capacity of the embryo is due to a morphogenetic hormone of the steroid group, the pregnant mother at the time of the fœtal "*male phase*" contains excess of steroid.

With regard to *anti growth*, Thompson and his co-workers in this country, working originally on parathyroid substances, have induced retardative growth effects on cancerous

and somatic growth. More recently they have found growth-inhibiting substances in the urine which contain one or more steroids.

Further evidence of what we may call "steroid growth" comes from the experiments of Dean Lewis and Geschickter of Johns Hopkins. Briefly, from bio-assays made of adenoma of the breast they have recovered large amounts of œstrin, and by injecting the latter directly into the undeveloped virgin breast, they have succeeded in producing mammary hypertrophy. However in their bio-assays of carcinoma they have not succeeded in isolating hormone with such pronounced properties.

Fœtal integration. Intersexual growth.—If we accept the suggestion that the fœtal endocrine system bursts into functional activity about this time, perhaps we must modify our views upon chromosomal determination. It has long been known and has been stated by Crewe that the sex chromosome mechanism can be upset and the sex determined in other ways. It therefore seems probable that chromosome determination *per se* is shortlived and that its main functions, especially those of growth, are shunted on to the endocrine functions of several glands, for we get so many glimpses of polyglandular upset in the pictures of our patients.

The nearest approach we can get to this difficult problem is the *time table* of endocrine integration proposed by Vines. It is suggested that should a female directed embryo undergo an abnormally strong and long male phase, tissue growth can be so altered as to result in the many degrees of *intersexuality* which we have encountered clinically.

Pituitary growth.—So far we have considered growth in those cases where we have been able to apply a hormogenetic label; there is also growth of a similar kind in which no hormone is obtainable and for the want of better knowledge we must refer it to some pituitary thalamic mechanism. However, there is a definite hope that the two forms of control must meet on common ground, and the study of Cushing's syndrome vaguely suggests there may be some reversible dual form of control.

A short while ago we published an article on the differential diagnosis of basophilism (*Brit. M. J.*, 1940 (i), 425). The symptoms are the same, whether they be due to basophil adenoma of the pituitary, or hyperplasia or carcinoma of the adrenal cortex. So far no hormone has been recovered from those due to basophil adenoma, whereas steroid in the adrenal group is present in inordinate amount—one case of ours secreting the colossal total of 270 mg. *per diem*. Adrenalectomy cures the latter, whereas treatment of the pituitary lesion is still far from satisfactory. It is a curious anomaly that compared with virilism general growth in Cushing's syndrome is not so much affected whereas sex function is, and growth is directed more into the channels of a permanently pathological adiposity, which comes on quickly.

Carcinoma.—The subject of carcinoma arising from hormonal causes should be a stimulating incentive to future workers in this field. There is no doubt about the marked influence they exert on simple growth. Practically we must consider the subject still *sub judice*.

It is a curious biological paradox that the cells of carcinoma, in their planless riotous growth, should not only recapture the energy for growth displayed in embryonic life, but at the same time free themselves from the "organized growth direction" of their host. By killing their host and themselves they behave like parasites, but at the same time they retain their physiological function. This function disappears with the removal of the primary growth but reappears with the formation of secondary deposits, and the majority of these tumours possess strong endocrine properties. It seems justifiable to assert therefore that the study of malignant tumours is more likely to be enhanced on physiological lines than on the old anatomical ones.

Mr. P. B. Medawar: *Biological aspects of the tumour problem.*—The foundation of experimental cancer research is the belief that its various lines, like the lines of perspective, converge somewhere to a point. A tradition as old as cancer research itself has it that this point lies within the domain of biology, and that the tumour problem is in some vague but important way a biological one.

The simplest, oldest, and most persistent of biological theories about tumours may be expressed as follows: *tumours are essentially collections of cells which have escaped from the growth-controlling influences of the body.* This is perhaps no more than a formulation of the tumour problem in biological terms; a formulation which is not self-evident, and which, in all probability, is not correct.

(1) *The relative rate of growth.*—The most striking feature of malignant tissue is its relatively high rate of growth. It may grow quite slowly, and yet multiply itself faster

than the tissues of the remainder of the body. It is now widely agreed that a relatively high rate of growth is not (from the aetiological point of view) a critically important character of tumours; and it is worth while setting down the specific grounds upon which this general opinion is founded.

Organisms change in shape as they develop. Up to the time when the main organ systems of the embryo have been blocked out, these changes of shape are brought about by growth (in the conventional sense), than by the streaming movements of cells and the convolution and folding of the various epithelial layers of which the embryo is primarily composed. Thereafter, changes of shape are chiefly determined by the *differential growth rates* of the organs themselves. Clearly, there must be some order and coherence in the process by means of which a final characteristic shape is reached, and D'Arcy Thompson (1917) and Huxley (1932) have made fundamental contributions to our knowledge of it. As a general rule, the specific growth rate¹ of part of an organism bears a constant ratio, over significantly long periods, to the specific growth rate of the whole. If this ratio is greater than unity, we speak of a "positive allometry". Some organs, like the antlers of deer and the claw of the fiddler crab, have remarkably high ratios of relative growth. If these organisms grew very much larger than they do, mechanical and other factors would bring about, or at all events facilitate their destruction in the course of natural biological competition. Speaking of the fiddler crab, Huxley remarks that one whose body weighed 1 kg. would theoretically bear a claw weighing 10 kg.: "It is perhaps no coincidence that the largest fiddler crabs attain sizes far below those of many other Brachyura, and even far below those of other land or semi-land crabs." The moral to be drawn from this is that there is a point in the course of normal growth beyond which, while precisely the same "growth laws" are obeyed (indeed, *because* they are obeyed), growth becomes abnormal and deleterious. Factors similar to this are thought to have been partly responsible for the extinction of the giant armoured reptiles of the Mesozoic. But there is nothing *malignant* about this sort of growth—in the technical sense.

(2) *Senescence and the damping-down of growth rates.*—Part of the biological problem of cancer may be approached through that of senescence (cf. Cramer, 1932). Some tumours may be propagated indefinitely by cellular homoplastic grafts, an immortality which (in a rather modified form) they share with normal somatic cells and with societies of non-cellular organisms (see Jennings, 1939). We can express the problem of senescence, in so far as growth phenomena provide a picture of it, in the following more or less formal way. In a constant environment—an environment which has to be *kept* constant—growth proceeds by compound interest; either something near continuous compound interest, as in the case of colonies of yeast grown in a suitable perfusion apparatus (Richards, 1928); or discontinuous interest, as in the case of tissue cultures, which undergo a microcosmic cycle of growth and senescence in their culture cells until their medium is renewed and the cycle starts afresh. But in actual development, the rate of self-multiplication of tissue does not remain constant; it falls off throughout life. In technical language, the *specific acceleration of growth is always negative* (Medawar, 1941). The rate at which the rate of self-multiplication falls off was regarded by Charles Minor (1908) as a measure of the rate of ageing; and it is in this sense alone that I believe the problem of senescence to be related to that of cancer. If we can find out *why* the multiplication rate of tissue falls off in this way, we shall know in *what terms* to express the empirical fact that in normal development, growth does not proceed beyond a certain point. There are several ways in which we can measure this process of falling-off. For example: if we remove pieces of the heart of a chicken embryo at various stages in its development, we find that their power of resistance to growth inhibition falls off exponentially, in the way that heat is lost from a cooling body (Medawar, 1940). It takes about twice the strength of a standard inhibiting substance to hold the growth of six-day old tissue in check as it does to suppress growth from an eighteen-day old tissue. What happens in the meantime? Is the process irreversible; are the tissues losing a capacity for unlimited free growth which they only regain when they become malignant? Curiously enough, it has only quite recently been possible to give a clear-cut answer to this question. The answer is No. For if we *cultivate* tissue from an older embryo, its power of resistance to chemical inhibition rises sharply, and it returns in this respect at least to an earlier embryonic state. It has likewise been elegantly shown (Hoffman, Goldschmidt, and Doljanski, 1937) that tissues explanted from donors ranging from six days to a year in age do not differ appreciably in their *capacity* for growth; they merely take an increas-

¹ The *specific* growth rate is simply the growth rate divided by size; $1/W \, dW/dt$ instead of dW/dt .

ingly long time to set about it (Cohn and Murray, 1925; Suzuki, 1925; Goldschmidt, Hoffman, and Doljanski, 1937).

There is therefore nothing irreversible in the consequences of differentiation (in mesenchyme cells) in so far as they affect the capacity for growth. Is the falling off of multiplication-rate *in vivo* due to some "intrinsic" change in cells, or is it due to some externally imposed and actively maintained growth-inhibiting influence? Both factors may play a part. But there is certainly no simply-extractable contact hormone in adult tissue which directly restrains the growth of cells. Though it has been said (Walton, 1914; Heaton, 1926; Brues, Jackson, and Aub, 1936) that adult liver tissue contains an inhibitor for explanted mesenchyme cells, saline extracts of most adult tissues tend on the whole to stimulate growth (Trowell and Willmer, 1939; Hoffman, Tenenbaum, and Doljanski, 1939). Tissue-culture experiments on growth-inhibition are by no means easy to evaluate. Carrel and Ebeling (1921; 1923 *a, b*) maintain that serum contains a lipoidal (Baker and Carrel, 1925) inhibitory principle whose concentration increases with the age of the serum. It is difficult to attach much significance to these results. Finely emulsified lipoids, such as they used, may well inhibit tissue-culture growth for quite non-specific reasons (e.g. surface activity: see Katzenstein and Knake, 1931); and it is in any case likely, as colloid theory suggests, and as Mayer's experiments (1936) directly prove, that emulsified lipoids occlude the growth-stimulating proteins of embryo extract. In either case we are dealing with a colloid phenomenon of no special significance.

Nevertheless, it has been conclusively shown that the lag-period before outgrowth *in vitro* which (as we said above) chiefly distinguishes adult from embryonic tissue growth, can be reduced without killing the cells by careful digestion of the tissue with trypsin or papain (Simms and Stillman, 1937). The experiments indicating this effect were devised in such a way as to exclude the possibility that tryptic digestion merely liberates growth-stimulating peptones and polypeptides (Baker and Carrel, 1928, *a, b*); and they likewise exclude the possibility that it liberates permeability-increasing factors which stimulate leucocyte activity (Menkin, 1936; Duthie and Chain, 1939). It is likely, then, that there is a non-diffusible protein in the intercellular fluids of adults that discourages the inception of free growth. Tumour cells, it should be noted, seem to lack the growth-inertia of adult tissues, though they do not necessarily grow very rapidly (Doljanski and Hoffman, 1940).

Experimental results such as these have been used as evidence for the doctrine (see, for example, Murphy, 1936) that there is a dynamic balance of opposed growth-controlling influences in the body, and that in the induction of tumours, this balance is upset. I cannot understand this view. The evidence I have quoted above indicates that if such a balance exists, it is struck between more growth and less growth—not between malignant growth and normal. This is also, in the main, the conclusion we draw from studies on endocrine and other systemic factors influencing growth.

One alternative to the view that the growth of developing cells is immediately controlled from without is that they themselves undergo a gradual change in the type of metabolism associated with growth. I have discussed some aspects of this problem elsewhere (1940): it belongs essentially to the biochemical field.

(3) "*Regulation*" phenomena; over-compensation.—None of the evidence we have so far considered seems to be directly relevant to the tumour problem, although some of it purports to be. The stepping-stone between normal and malignant growth has been laid down in imagination but not in fact. We may therefore turn to a third phenomenon of natural growth that has found a place in cancer aetiology as the stepping-stone we are looking for.

It is true, as a general rule, that the organism is capable of making up for the consequences of normal or artificial growth inhibition by a sudden burst of recovery growth when the inhibitory stimulus is released. To distinguish this phenomenon from normal regeneration (to which it is obviously closely allied) and from compensatory hypertrophy in the usual sense, I shall call it *post-inhibition growth rebound*. Four examples will show how widespread a phenomenon it is. Spear (1928) has shown that if mitosis in tissue cultures is brought to a standstill by temporary cooling, the loss of growth time is made good by a vigorous rebound on restoring the tissue to the incubator. *Amblystoma* larvae, though not perhaps the larvae of all amphibia, behave in the same way when cooled for varying periods from 22° to 6° C. (Buchanan, 1938). In his quantitative studies on the rate of healing of wounds, de Noy (1936) has pointed out that retardation of growth by infection is, in favourable cases, very largely adjusted by the relatively vigorous growth that follows when sepsis is restored. This is what we should expect, since Przibram (1917) has shown that the rate of regeneration of lost tissue tends to be proportional to the amount of tissue that has been lost.

A somewhat similar phenomenon occurs on a larger scale than this. If the calorific intake of young rats and mice is reduced for not too long a time below the level required for normal growth, re-feeding brings about a spectacular burst of recovery growth that takes the experimental animals back to, or even beyond the size of their controls (Osborne and Mendel, 1916; Clarke and Smith, 1938; Jackson, 1939). These experiments should be more widely known than they are.

In each of these instances we are dealing with the same type of physiological reaction: a burst of growth following artificial suppression which makes good the loss of growth time. Students of growth are familiar with the theory (*see* Haddow, 1935, 1938¹) that there is a marked correlation between tumour formation and the consequences of growth inhibition. Yet I do not think that this is the stepping-stone we are looking for. Post-inhibition rebound is a very widespread phenomenon; malignant growth is not known to be one of its consequences. Unfortunately, we cannot explain it, even in the most general terms, for lack of what we may call an adequate kinetic picture of growth. It is worth while mentioning that inorganic systems can be made to show an exact analogy to the phenomenon of post-inhibition rebound; and it is in this connexion that I should like to explain why I believe that the doctrine of growth-controlling factors, and the belief that cells stop growing *because* something stops them, is not self-evident, even if it is true.

We are so deeply influenced by the spirit of Newton's First Law that we tend to think that whenever a *rate* falls off, something is actively suppressing it. This is true of rates of motion, but it is not true in quite the same sense of the rates of a type of change which we may call changes in *probability states*. The rate at which heat is lost from a cooling body is initially high, and falls off as its temperature approaches that of the environment. The rate at which the distribution of molecules in a closed diffusion system tends towards uniformity is likewise rapid at first, and slower and slower thereafter. In these cases, and in others similar to them, we are dealing with rates which fall off "of their own accord"; with systems that tend to a certain, most-probable, state at a rate which depends upon how far they have yet to go to reach it. We may look in vain for inhibitors and controllers; they are not there.

We can use one of A. J. Lotka's (1925) statistical models to illustrate the type of change I am referring to. Suppose we start with two urns, one containing 50 black balls and the other 50 white, and at regular intervals remove blindfold one ball from each urn to replace in the other. After the first exchange, we necessarily have 49 balls of one colour and 1 of the other in each urn; and at the second exchange it is most unlikely (2499:1) that we shall be lucky enough to restore the *status quo* by exchanging the one white ball now in the black urn with the black ball in the white urn. But as swapping proceeds, it becomes increasingly likely that we shall remove from the originally all-black urn one of the white balls that got there on a previous exchange, and vice versa; so that the rate at which the most-probable-state (25 balls of each colour in each urn) is reached tends to slow down after being comparatively rapid at first. Multiply the number of balls a billionfold, and we have a model of the process as it takes place on a molecular scale. It is easy to construct statistical models illustrating regeneration phenomena (e.g. why a regenerating part grows to its right size, and no further), post-inhibition rebound, Le Chatelier's Theorem, and so forth. The models do not "explain" the phenomena in question, but they do suggest a way in which we can think about them.

I do not know whether what I have called the "kinetic picture" of growth will be found to fall within the domain of statistical mechanics, and I am not trying to substitute Maxwell's Demon for the demon of cancer. It is simply a picture we should try and keep in mind when thinking of growth processes, lest we should come to regard the doctrine of growth-controlling factors as self-evident, which it certainly is not. Carcinogenesis is the inception of a heritable capacity for growing under conditions in which other cells do not do so. This does not imply that normal cells are those which are somehow held in check.

(No more does it imply that a gene-mutation has been at work. Inheritance between organisms and between cells related to each other by mitotic descent are quite distinct from one another. After histological determination in the embryo, the tissue-cells breed true: epidermal cells divide to give epidermal cells, polymorphs to give polymorphs. Even tissues cultured for thousands of cellular generations maintain a characteristic modification of their histological type. Tumour cells likewise breed true, *in vitro* as

¹ Haddow's theory is expressed in a form much less general than this; it is only the principle we are examining here.

in vivo. If we assume that tumour cells are genetically distinct from normal cells, we can hardly deny that skin cells are genetically distinct from those of the liver. But this violates the fundamental theorem of developmental mechanics, which is that the cells of an organism have the same chromosomal make-up. The evidence of experimental embryology (see Needham, 1936); striking instances of de-differentiation in tunicates accompanied by a conversion of one cell-type into another; endocrine sex-reversal; heteromorphosis in crustacea and insects—all these show that no genetic factor is directly involved in the assignment and maintenance of histological type in cellular heredity. That the gene-complex reacts in different ways to different environments is a different matter. The mutation theory will be widely accepted when evidence has been found for it, and when certain difficulties of a purely genetical nature, concerning the extraordinarily high mutation-rate it implies,¹ have been satisfactorily resolved.)

To sum up: the growth of somatic cells resembles, or can by artificial means be made to resemble the growth of tumours in certain definite ways, and these ways do not provide us with the information we need for making a critical distinction between normal and malignant growth. In particular, tumours are not merely cells which have escaped from the growth-controlling influences of the body; or which have an excessively high rate of relative growth; or which have acquired a sudden malignant access of energy as a result of prolonged inhibition of their growth. Tumours are not *merely* anything. At the same time it should be said that experiments of the type quoted in the second section of this paper have by no means exhausted their significance for tumour theory, even if the significance is only indirect, and even if they relate to the properties of tumours already formed rather than to the problem of tumour formation. To link up some hitherto uncorrelated lines of research, we might investigate the fact that tumours, with a low growth-inertia, take comparatively well as homoplastic grafts, while adult tissues, with a high growth inertia, do not.² It is known on the one hand that careful proteolytic digestion of tissue abolishes the growth inertia in question; and on the other hand there are indications that tissue-storage reduces the intensity of the homograft reaction (Sønders and Young, 1942). Does the storage of tissue abolish the lag-period of adult tissue growth, and can proteolytic digestion be used to mitigate the homograft reaction? If they do so, we shall be able to establish a set of correlations with more than a remote bearing on the tumour problem, and of some interest for general surgery. But as hypotheses like these can be checked by simple direct experiments, it is not worth while pursuing them further in theory.

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1 I am grateful to Mr. E. B. Ford for pointing this out to me.

2 The approach to the problem of tumour transplantation through that of surgical homoplastic grafting and of "individuality differentials" is largely due to Loeb, who has reviewed it (1930, 1937; see also Weglem, 1929). Murphy (1926) and others believe that the lymphocyte reaction of homoplastic grafting is of fundamental importance in determining the resistance of the recipient to transplanted tumours.

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Dr. F. Gordon Spear: This paper summarizes part of an investigation by Dr. A. Glücksmann and myself which is being made to determine the changes in cellular activity after graduated doses of irradiation, in the course of which a number of normal and malignant tissues have been examined histologically before and after exposure. Since changes in cellular activity, whether spontaneous or imposed, must precede alterations in the tissue as a whole these changes should have a prognostic value.

(1) *Observations on unirradiated normal tissue.*—As an example of a relatively simple tissue we may take a hanging drop culture of fibroblasts which presents a form of growth consisting only of proliferating or of potentially proliferating ("resting") cells. In such a culture successive cell divisions lead to the symmetric outgrowth of undifferentiated cells in all directions (fig. 1a).

In the organs and tissues of growing embryos, proliferation is associated sooner or later with differentiation but the two processes are separated either in time or in space. For example, in the mammalian eye (human and rat) a period of proliferative activity is followed by a differentiation period during which cell division is suspended (fig. 1b).

In the frog tadpole eye, on the other hand, the central parts are fully differentiated and functioning, while proliferation still continues in the peripheral region, i.e. the two processes are separated spatially. In this case, about half the number of daughter cells which result from cell divisions remain in the germinative zone for subsequent division, while the rest migrate away and differentiate (fig. 1c).

Other examples of the spatial separation of proliferation and differentiation are found in the intestinal crypts, the hair follicles, and the stratified epithelia of many adult animals.

In all these cases the processes of proliferation and differentiation are mutually antagonistic; the differentiating cell does not divide.

(2) *Observations on unirradiated malignant tissue.*—The carcinogenic changes brought about by painting mouse skin with benzpyrene include not only a stimulation of proliferative activity but also interference with the differentiation processes of repair. Loss of hair and the destruction of skin appendages caused by the initial paintings are followed by the appearance of abnormal hairs and the formation of sebaceous glands at abnormal sites. With continued painting the stimulation to proliferation is maintained, together with the disorganization of the processes of differentiation, leading to an abnormal regenerative activity in which the clear-cut antagonism between the processes of proliferation and differentiation is lost. The basic tumour cell, unlike the normal proliferative cell in the germinative zone, exhibits some degree of differentiation while retaining its proliferative capacity. The degree of differentiation is slight and it is still possible to speak of the potential dividing malignant cell as a resting, or (relatively) undifferentiated, cell by comparison with the more fully differentiated tumour cell which is no longer a potential mitotic cell. These terms are relative to whatever tissue they are applied, e.g. the "resting" cell of normal growth is itself more differentiated than a very primitive embryonic cell.

However the malignant cell may have originated, considered from the point of view of cellular reaction to abnormal stimuli, malignant growth does not appear as a mass of irregular and lawless cells entirely dissimilar in behaviour to the normal cells from which the tumour arose. In the majority of tumours it is quite easy to recognize areas of proliferative activity in the advancing edge of the tumour, and if these areas are examined by quantitative histological methods almost any tumour shows a surprising constancy in cell behaviour in each of these regions. The proportion of dividing (M), resting (R), differentiating (Df) and degenerate (Dg) cells determined for any one growing area of a tumour is typical for the other growing areas of the same tumour (see table).

TABLE SHOWING RESULTS OF QUANTITATIVE HISTOLOGICAL ANALYSIS OF DIFFERENT GROWING AREAS OF THE SAME TUMOUR.

Df differentiating cells, M mitotic cells, R resting cells, Dg degenerate cells

| Sample No. | Total counts: | | | | Percentage: | | | |
|------------|---------------|----|-----|----|-------------|---|----|----|
| | Df | M | R | Dg | Df | M | R | Dg |
| 1 | 7 | 14 | 259 | 25 | 2 | 5 | 85 | 8 |
| 2 | 11 | 13 | 388 | 32 | 2 | 3 | 88 | 7 |
| 3 | 8 | 7 | 286 | 24 | 2 | 2 | 89 | 7 |
| 4 | 11 | 10 | 213 | 24 | 4 | 4 | 83 | 9 |
| 5 | 7 | 10 | 218 | 28 | 3 | 4 | 82 | 11 |
| 6 | 8 | 9 | 234 | 22 | 3 | 3 | 86 | 8 |
| 7 | 8 | 7 | 204 | 25 | 3 | 3 | 84 | 10 |
| 8 | 10 | 8 | 219 | 27 | 4 | 3 | 83 | 10 |
| 9 | 9 | 9 | 212 | 19 | 4 | 4 | 84 | 8 |

The variation seen in other regions of the tumour is a result of the ageing of cells (advanced differentiation), death of unstable cells, and changes due to alterations in the circulation or in the tumour bed. Comparison of such parts shows no constancy either qualitative or quantitative.

When the growing areas of a series of growths are analysed quantitatively, different tumours, at present included within the same pathological classification, are found to vary in the proportion of differentiating cells which these areas contain, and also in the ratio of dividing to non-dividing cells. The relation between differentiation and proliferation can, indeed, be used as a basis for classifying tumours in terms of cellular behaviour, and such a classification is beginning to be of practical use.

In a rodent ulcer, for example, the active cells divide but do not differentiate (fig. 2a). In a keratinizing or a parakeratotic squamous-cell carcinoma on the other hand, the cells may either divide or differentiate (fig. 2b). The ratio of proliferating to differentiating cells varies from one type of tumour to another, and the tendency is for differentiation to be less advanced in the tumours which show the greater proliferative activity (fig. 2c).

The differentiation which occurs in a malignant cell is not of a normal type, and the cell, being unable to function, eventually degenerates, though a considerable time may elapse before it disintegrates. Many undifferentiated cells, on the other hand, survive only a relatively short time and break down in the process of mitosis. The rodent ulcer which appears to consist solely of potentially dividing cells is actually of low malignancy owing to the number of cells which degenerate through lack of nutrition or some other cause when division is attempted. A more malignant tumour is one whose cells exhibit a low degree of differentiation and survive without losing their capacity to proliferate. The parakeratotic basal-cell carcinoma is an example of this type of growth.

(3) *Observations on irradiated material.*—The significance of cellular activity in the youngest areas of both normal and malignant growth in determining the ultimate character of the tissue is emphasized after exposure to sublethal doses of radiation, which inhibit one form of activity without preventing some alternative behaviour.

At the lower dose levels (100 r—1,000 r) irradiation of a tissue culture with gamma or X-rays has the effect of delaying the onset of cell division with subsequent breakdown when mitosis is attempted. Thus a diminution in mitosis is followed by the appearance of degenerate cells in place of dividing cells, and the amount of degeneration is proportional to the number of premitotic cells present at the time of irradiation.

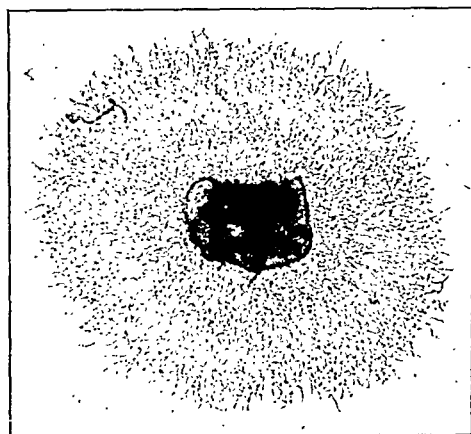
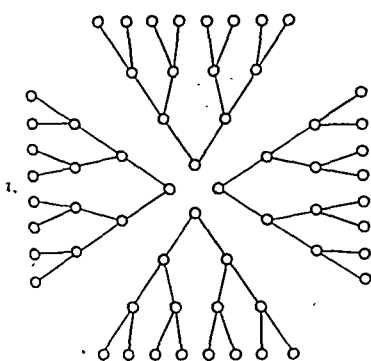
A similar result follows the irradiation of the rat eye in the proliferative period, with little or no effect (at this dose level) on the cells which have differentiated.

In the case of the tadpole eye, where proliferation and differentiation are occurring simultaneously, a cell prevented from entering division may be induced to differentiate instead. In these circumstances the diminution in mitosis is followed by a small amount of degeneration and an increased amount of differentiation which is the alternative to a cell attempting division and breaking down.

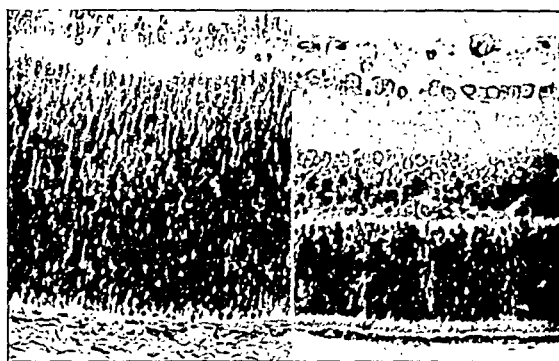
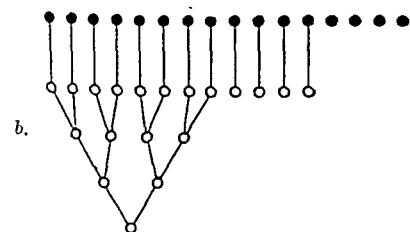
Malignant cells also vary in their response to radiation according to their activity at the time of exposure. After irradiation proliferating cells of a rodent ulcer break down in greater numbers, giving a wave of degeneration which replaces the mitotic activity characteristic of the growth.

In the case of the squamous-cell carcinoma, where the processes of proliferation and differentiation occur simultaneously, the diminution in mitosis is accompanied by increase in differentiation. Significantly enough, the parakeratotic basal-cell growth is the more difficult to deal with radiologically by present methods than either of the other two types of malignant growth. The inhibition of mitosis does not seem to induce the degree of differentiation necessary to exclude the possibility of subsequent mitotic activity.

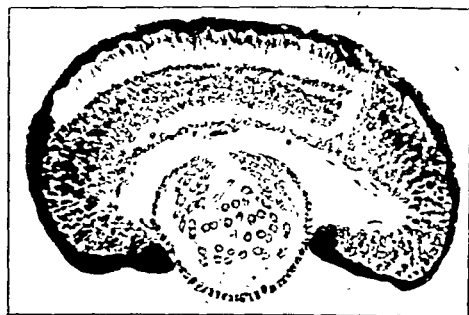
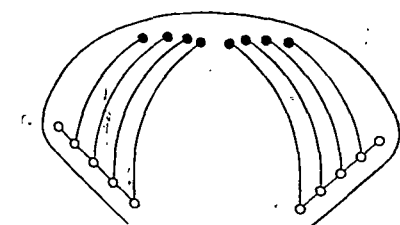
Observations on the limited amount of material so far examined suggest that any estimation of the character of a tissue, normal or malignant, or of its likely response



× 10



× 210



× 360

FIG. 1.—Growth and differentiation in some normal tissues shown diagrammatically (left) and photographically (right). a. Tissue culture: proliferation among undifferentiated cells. b. Rat eye: 2-day eye in stage of proliferation (on left); 10-day eye in stage of differentiation (on right). c. Frog tadpole eye: proliferation and differentiation occurring simultaneously.

○ = resting cell.

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● = differentiating cell.

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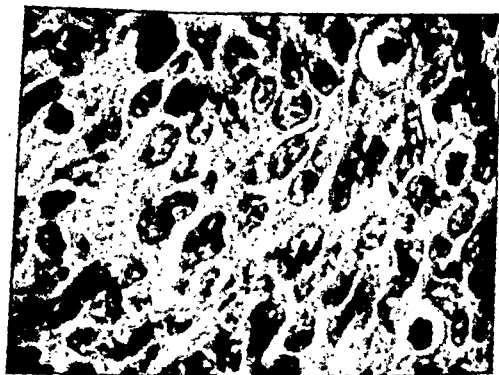
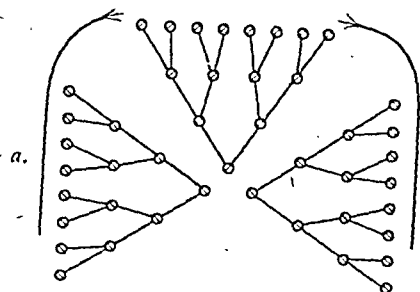
to radiation, or any other agent, must be made not in terms of proliferation or differentiation considered as isolated processes, but in terms of the quantitative relation of these two processes to one another in carefully selected young areas of the tissue.

Dr. Alex. Haddow: This discussion has so far dealt with one or other aspect of normal growth, as also with the processes of wound healing, regeneration and hypertrophy, and with the long-continued maintenance of non-malignant cells *in vitro*. The factors which normally restrain the potential growth-capacity of normal cells *in vivo* are in part associated with the degree of cellular differentiation, and in part with the density of the cell population. The importance of the latter condition is well shown in Wigglesworth's studies of the healing of wounds: migration of cells towards the wound defect produces a relative sparsity of cells at a distance, and this permits the division of the cells in that area to a point where the normal cell numbers are approximately restored. From these considerations it is clear that the growth of malignant cells is not strictly a new or unique character, but rather the permanent unmasking of a property possessed all along by their normal prototypes. The outstanding feature of the malignant cell lies rather in the manner in which its heightened growth-rate is persistently maintained, being only slightly affected by environmental factors which normally contribute to the restraint of cell-division. As Dr. Gye has said, the course of malignant proliferation is limited simply by the life of the host, and may be extended indefinitely by serial transplantation.

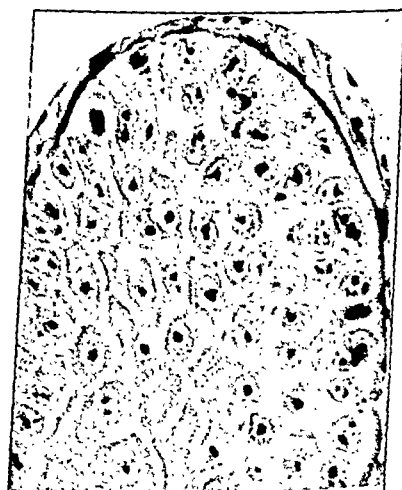
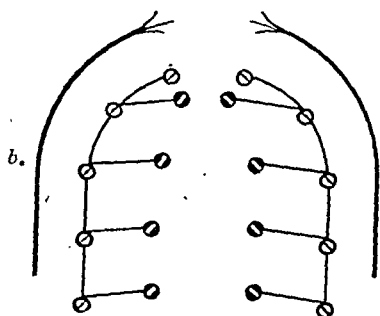
The change from the normal to the malignant state is essentially an irreversible alteration of the cell. Furthermore, when its induction is brought about by chemical means the malignant variant appears (1) not merely freed from the "normal" restraining influences, but also (2) independent of the continued presence of the carcinogen, and (3) resistant to the effects produced by the specific carcinogen on normal cells. These last effects, which it is presumed are the changes which instigate the malignant transformation, are essentially inhibitory in nature. Dr. Medawar, in quoting examples of the well-known phenomenon which may be called "post-inhibition rebound", has shown that this process can have little significance in the problem of the induction of cancer. The proposition would seem self-evident, since the great majority of agents which temporarily interfere with growth in such a way as to permit an early recovery or rebound—with or without compensation—are not carcinogenic. It has been reliably shown, on the other hand, that the carcinogenic hydrocarbons produce an interference with growth which is relatively *persistent*, so that the usual process of complete recovery is less easy and becomes progressively more difficult. It is in these circumstances, I would suggest, that the adaptation effected by the cell takes the form of a discontinuous and irreversible variation involving chiefly and usually a lowering of differentiation, and conferring automatically a proportionate and permanent enhancement of the rate of growth. These considerations depend for support on the substantial correlation which has been shown to exist between (1) the capacity to produce this type of growth-inhibitory effect, characteristic if not specific, and (2) the power to induce tumour-formation, in the carcinogenic hydrocarbons and groups of allied compounds, of which several hundreds have now been examined.

To many interesting chemical and biological relationships between carcinogenic and oestrogenic compounds may be added the growth-inhibitory and carcinogenic properties which have been ascribed to oestrogens, and certain of which have been alluded to by Mr. Burrows. In this connexion it may be significant that certain synthetic oestrogens (e.g. derivatives of triphenylethylene) may exhibit a structural or skeletal resemblance to cyclic compounds derived from 1:2-benzanthracene. The same type of approximate similarity of molecular arrangement (in this case to the synthetic oestrogen diethylstilboestrol and the carcinogenic hydrocarbons 3:4-benzpyrene and 1:2-dimethylchrysene) is shown by α -ethyl- β -sec-butylstilbene, a compound recently described by Dodds, Lawson and Williams as possessing slight carcinogenic activity in mice. Of related interest is our own finding that the polycyclic hydrocarbon 9-methyl-1:2-benzfluorene (synthesized by Dr. G. M. Badger) shows pronounced oestrogenic activity: the carcinogenic activity of this compound, if any, is not yet known.

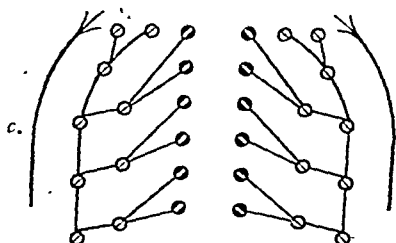
As to the means by which carcinogenic substances produce interference with growth, various suggestions have been made from time to time by different workers. I would like to draw attention to certain current researches reported by Rhoads and his group, from the Memorial Hospital (e.g. *J. Nutrition*, 1941, 21, Supp. 1, 14; *Bull. N.Y. Acad. Med.*, 1942, 18, 53-64; *Cancer Research*, 1942, 2, 1-10), which are of great interest in themselves, and may later prove to have an important bearing on this problem. In experiments involving a study of the metabolism of *p*-dimethylaminoazobenzene ("butter yellow"),



× 775



× 550



× 650

FIG. 2.—Growth and differentiation in some malignant tissues. *a.* Rodent ulcer: scattered mitotic figures among relatively undifferentiated cells. *b.* Squamous cell carcinoma: differentiating cells centrally with resting and dividing cells peripherally. *c.* Parakeratotic basal cell carcinoma: degree of differentiation much less marked.

⊙ = resting cell.

✂ = dividing cell.

◐ = differentiating cell.

The broken line represents the basement membrane.

Section of Neurology

President—GEORGE RIDDOCH, M.D.

[March 19, 1942]

DISCUSSION ON DIFFERENTIAL DIAGNOSIS AND TREATMENT OF POST-CONTUSIONAL STATES

Air Commodore C. P. Symonds: I shall assume that we are discussing the individual who has recovered from the acute stage of his head injury and is now in the chronic, or ambulant stage.

The problem of differential diagnosis and treatment in such cases may be divided into two. First there is the case in which the neurologist has seen the patient in the acute stage and has been able to observe subsequent progress. This is relatively simple. The second type of case is that in which the patient is first seen in the post-contusional or ambulant stage. (Ambulant, of course, must not be taken too literally. In many of these patients there are associated injuries which necessitate their being still in bed.) These cases are not only the most difficult, they are also the most common. Patients with head injuries are usually admitted to the nearest hospital and it is generally some time before the neurologist sees them. What, at this stage, is the general nature of the problem? It is that of a patient with a variety of subjective complaints, little in the way of abnormal physical signs, and, regrettably often, a quite inadequate record of the early stages of his illness.

Accurate diagnosis and correct treatment in such a case are of the utmost practical importance and the problem is one which demands a considerable expenditure of time if we are to arrive at any satisfactory solution. Here at once is a practical difficulty. Cases of this type should preferably be seen by appointment, or admitted to hospital for observation.

Reconstruction of the injury.—Our problem begins with the reconstruction of the story of the injury, often with scant information apart from what the patient can tell us. Fortunately the retrograde and post-traumatic amnesias can be estimated with rough accuracy at this stage, but it is important, if we are going to use these estimates, that we should standardize our end-points as far as possible. The man's last memory before the injury is usually a reliable point for the retrograde amnesia, but there are sometimes difficulties. Recollection is influenced by factors apart from the injury, such as the significance for the individual of events in the preceding period. Were they events worth remembering? Dull or exciting? Commonplace or unusual? The estimate of retrograde amnesia to be of real value should be accompanied by reference to these points. The estimate of post-traumatic amnesia needs similar details of circumstance. Moreover the observer must decide whether his end-point will be the first memory after the accident, or the beginning of continuous awareness. Often these correspond, but when they do not, when the first memory is, so to speak, an island, the beginning of continuous awareness of the surroundings is the safer guide. It would be convenient if all neurologists would agree to use this measure in stating the duration of post-traumatic amnesia.

In relation to the measurement of post-traumatic amnesia, it is important to know whether or not the patient has been given morphia, especially in cases in which the total duration is a matter of hours.

Type of headache.—We next want the story of the patient's symptoms up to the time of our examination, in relation not only to the sequence of time, but the sequence of events. We want to know, for example, not only whether he had headache and what kind of headache, but when he first had it, how often, and under what conditions it was worse or better. What was the effect upon it of having visitors, of sitting up, or first getting out of bed? All these questions apply equally to giddiness, and to the state of thought and feeling. If he was depressed or anxious, when and in relation to what circumstances, reflections or anticipations? What has his attitude of mind been towards the accident and its aftermath and what have been the stages in the development of his present attitude? This leads naturally to the analysis of his present complaints. Subjective symptoms are many and important.

The more experience I have of traumatic headache the more difficulty I have in dividing it into clinical types. There is, it is true, a localized variety, usually in the neighbourhood of the site of injury, intermittent, short-lived, sharp or throbbing, and related to physical effort or change of posture. This, when present, is highly characteristic of local injury, but it is uncommon in pure form. As another extreme example there is the continuous,

an azo dyestuff which Kinoshita had previously observed to cause liver cancer in rats. Rhoads and his co-workers found that the administration of this carcinogenic substance injured normal liver cells by interfering in some way with at least one enzyme system essential for their normal chemical and biological function, and that this interference was probably of aetiological importance in the malignant transformation. It was further suggested that, while the oxidation processes of the normal liver cell are extremely susceptible to such interference, the development of the mutation which characterizes the malignant liver tissue is marked by the presence of an oxidizing enzyme system no longer susceptible to the inhibitory effect. It is obvious that there is a striking parallelism between these biochemical findings and the views I have already suggested on the physiological side, namely, that the formation of a chemically induced malignant tumour is brought about by a rather characteristic interference with the growth-capacity of the corresponding normal cell, and that the resulting malignant cell is so altered as to be resistant, for a time at least, to the inhibitory activity of the compound which provoked its emergence.

Mental disability.—Complaints of mental disability are, as a rule, classifiable under two headings. The patient tells us that in certain respects he is unable to perceive, remember and think as quickly and clearly as he could before the injury; and he says that somehow he feels different. Both kinds of disturbance are usually present in the same case.

There are many variations of this central theme, some more characteristic of brain injury than others. On the intellectual side, inability to enjoy reading for lack of power to hold the thread of the story, difficulty in grasping the war news, forgetfulness of small things, are significant. In the sphere of feeling the most typical complaint is of loss of interest and liveliness, but nervousness, depression and irritability are common. There are also cases in which the mood is elevated and activity increased. This patient often complains of nothing. His beaming smile and confidence of his own fitness are disarming and, at the same time, significant.

It is generally recognized that the family and personal history are important in assessing disability and guiding treatment. It is essential before we sum up the case that we should have at our disposal the main facts of the pre-traumatic personality and intellectual level, and know whether beyond this there are possibilities of an inherited and latent disposition to mental disturbance of a kind which may be precipitated by injury. There is as a rule more to be gained from this source than from a protracted neurological overhaul and the time available for examination should be distributed in accordance with these practical values.

Neurological Examination

By the time our post-contusional patient is examined he is unlikely to show any abnormal physical signs. Nevertheless, routine examination may occasionally reveal something unexpected and important. Of such signs anosmia is the commonest. If this is complete it will have appeared in the patient's complaints as inability to smell and taste, but there are many cases in which it is not complete. Bilateral anosmia may be present in a man who is yet able to distinguish flavours reasonably well. Unilateral anosmia as a rule passes unnoticed by the patient until it is specially looked for. Our usual methods of testing are, of course, crude, but when there is inability to distinguish test odours, without local obstruction or inflammation to explain it, and with a previous history of normal capacity for smell, we may presume that the disability is the result of the injury.

There are two facts in this connexion which are perhaps not generally known. One is that traumatic anosmia is not uncommonly associated with occipital fracture. The other is that it may result from an injury without evidence of severe generalized cerebral disturbance.

Captain D. F., aged 27. On 20.11.41 at a sing-song he was attempting to seat himself on a mantelpiece when he slipped, falling on his back. He does not remember hitting his head, but probably lost consciousness momentarily. He got up, but felt dazed and went to bed. On waking next morning he had a "thick head", but no pain. He went on duty, which involved a long journey by car, during which he developed generalized throbbing headache, spreading down the back of his neck, nausea and eventually vomiting. He abandoned his journey and was admitted to hospital where meningitis was suspected. Lumbar puncture revealed a heavily blood-stained fluid with yellow supernatant fluid, and the diagnosis was altered to subarachnoid hæmorrhage. Headache and vomiting continued for three days, after which he rapidly improved. He found, however, that he had lost his sense of smell and could taste nothing in his food but sweet and bitter. He was transferred to a hospital for head injuries on 11.12.41 feeling well, save for occasional slight headache on reading. The positive findings were complete bilateral anosmia; a fine fissured fracture of the occipital bone in the mid-line running to the foramen magnum, and some low voltage 1 to 2 a second waves in both occipital regions in the E.E.G. There can, of course, be no doubt that the subarachnoid hæmorrhage was traumatic.

In a series of 1,020 cases of closed head injury, anosmia attributable to the injury was found in 76; bilateral in 62 and unilateral in 14. Of the bilateral cases 30 complained of their inability to smell and 16 of these also complained of inability to taste. Bilateral anosmia in this series was associated with X-ray evidence of fracture in 42 out of the 62 cases, the situation of the fracture being most frequently frontal (26 cases), and next most frequently occipital (12 cases), usually a fissured fracture of the occiput running into the foramen magnum.

A lesion of the optic nerve may have been missed if the field defect is small. Visual acuity therefore should always be examined. Lesions of the infra-orbital and supra-orbital nerves are also not uncommon in fractures involving the roof of the maxillary antrum, or frontal bone, and may be better evidence of fracture than X-rays.

Dysphasia, hemiparesis or sensory defect, if they exist at this stage, will almost always have been detected and should have been recorded in the earlier and grosser stage. Signs of slight pyramidal damage may sometimes be found when least expected. Homonymous visual field defects will occasionally be missed if the method of testing by confrontation is omitted. Perimetry seldom yields anything of value if confrontation tests carefully executed are negative.

These observations will serve to indicate that the neurological examination at this stage should be intelligently guided. To include examination for dysphasia or dyspraxia,

dull, generalized headache, unrelated to any circumstance. Either type may be encountered in individuals whose injuries have been of comparable severity, whose symptoms in other respects are the same, and who are apparently of the same constitution and disposition. Between these extremes there are all kinds of mixtures.

Knowledge of the circumstances which induce or relieve headache, and of the symptoms associated with it when it is present, is generally more helpful in differential diagnosis and treatment than the character of the headache. I would take as examples headache induced by continued though mild physical effort, such as walking, and associated with sensations of fatigue; headache similarly induced by continued mental effort and accompanied by a feeling of mental fatigue; headache induced by an unusual degree of stimulation of one of the special senses, such as noise or light; headache associated with mood disturbance, such as irritability or depression. Headache may be prominent after a brief amnesia. It may be absent after a prolonged amnesia.

If the amnesia has been prolonged it is useless to rely upon the patient's statement at a later date that he has never had any headache. It is not uncommon for a patient to have complained of severe headache during the amnesic period. It is therefore important that the observer of the earlier stages should record in the notes the presence, or absence, of headache during this phase. If a patient has reached the ambulant, or chronic, stage without headache it does not follow that he will continue to be immune. It is by no means uncommon to find a man beginning to complain of headache when, it may be several weeks after the injury, he is exposed to additional stress, and this is especially apt to occur when the exposure is sudden. It is important therefore when a patient has been headache-free up to the time of examination to know under what conditions of mental and physical stress this freedom has been preserved.

Dizziness.—A high proportion of patients in the stage under discussion complain of giddiness or dizziness. Of these a small proportion only describe true vertigo. Thus, out of 1,020 cases of closed head injury in which the symptom was inquired for, it was found present in 82. In 29 of these the vertigo was associated with deafness of middle or inner ear type, dating from the injury, and in 4 others there was a history of bleeding from the ear, or tinnitus in the early stage. This leaves 49 in which there was no evidence of aural damage. Two of these had damage to the 7th nerve, suggesting a fracture involving the petrous bone. In the remaining 47 cases there was no evidence pointing to the labyrinth as the probable site of injury. Of these 47 cases it is interesting to note that in 7 there was a history or presence of diplopia and in 2 others nystagmus was recorded in the early stages, symptoms indicating the probability of brain-stem injury.

There remain 38 cases, nearly half the total number with vertigo, in which no evidence either of aural or brain-stem lesion was forthcoming. We may, however, assume that vertigo in the true sense is evidence of damage to the vestibular sense organ or its central connexions. It is important evidence of organic damage and I suspect that if inquiry as to the presence or absence of deafness and tinnitus on the one hand, and diplopia and nystagmus on the other, were more rigorous in the early stages, we should have fewer cases in which corroborative evidence of labyrinthine or brain-stem injury is lacking.

Generally the complaint is not of true vertigo but of a transient disturbance of balance and often of the visual sense, experienced on stooping, or rather on rising from the stooping posture. This is probably due to a defect of vasomotor adjustment, and in the light of the recent experiments of Denny-Brown and Ritchie Russell (1941) may result from medullary concussion. It is a common constituent of the post-traumatic syndrome.

There are other varieties of dizziness which are less easily placed. I would draw attention in particular to one which is often described as a "black out". The onset is sudden, there is dimness of vision and a sense of insecurity of balance which may result in falling, without any description of true vertigo. Consciousness is often momentarily disturbed and may be lost. The main features of these attacks are syncopal rather than epileptic. Nevertheless, in some cases after repetition there is a transition into epilepsy.

An officer, aged 22, was injured in an accident on 7.10.39. Retrograde amnesia a minute or two, post-traumatic—forty-eight hours. He sustained a longitudinal fissured fracture of the left parietal bone and abrasions and contusions of the right chin, nose and left forehead. He found on recovering consciousness that he had anosmia and diplopia and suffered severely from headaches in the first three weeks. Within three months of his accident he was back on light duty with only occasional headache. The anosmia had persisted. The diplopia had recovered. Shortly after he returned to duty he began to have attacks, which he described as "muzziness", "you can't think as clearly as you would like, your hands get sort of clammy". It came on gradually and faded gradually after five to ten minutes. No spinning or sense of movement in space, and no feeling that he would lose consciousness at all. He paid little attention to these attacks, which were infrequent, but on 27.5.40 he had an attack beginning in this way in which he lost consciousness for five minutes.

There was no history of epilepsy in the family. He himself, as a child, after running on a hot day, had once fainted for a few minutes.

The E.E.G. was normal. He was retained in the Service in a restricted category and in January 1941 was admitted to another hospital on account of frequent attacks in the past three weeks of loss of consciousness. These would be preceded by a feeling of depression for one to three hours, together with drowsiness. He would then suddenly look very pale and become unconscious for several minutes.

The diagnosis made by experienced observers was that of epilepsy.

For convenience of description I suggest that it would be better to use the ordinary psychiatric headings with slight modification, e.g. Brain Injury with Intellectual Impairment, Brain Injury with Depression (or other affective disorder), Brain Injury with Hysteria, Brain Injury with Psychopathy.

Treatment

There should be no delay once the examination is completed, in giving explanation and reassurance. In giving this we had best be truthful. We shall then be obliged to admit that headache, dizziness, difficulty in concentration and feelings of nervousness and depression are often slow to disappear; but we can say, at the same time, that symptoms of this kind seldom prevent a man for long from returning to his usual occupation, provided that he is patient and will make the best of things. We can dispel fears of insanity, and it is surprising how common these are. We can, and should, do a great deal more than this, of course, in the way of psychotherapy, which has a place in the treatment of every case of this kind.

The ideal atmosphere for treatment at this stage is that of a convalescent hospital at which there are well-organized departments for occupational therapy, physical exercises and indoor and outdoor games. The daily routine should be planned with suitable spells of compulsory rest in the early stages, and a reasonable allowance of free time. For Service patients the problem of disposal naturally looms large and this has close relation to treatment. Men who are to be invalided should be separated, as soon as the decision is made, from those undergoing rehabilitation for return to duty. When there is a probability that a man's category will have to be reduced, the sooner the decision is made the better, in order that he may know what lies ahead of him.

There is much ground for optimism in the treatment of post-contusional states, especially in young people. We see many cases of complete recovery after a post-traumatic amnesia of many days, or even weeks, but we should be wrong to take these as our standard. If we do so we shall be promising the majority more than they will get, and asking of them more than they can give. The results often are disillusionment and resentment. These two symptoms are often prominent in the post-contusional state and hard to get rid of once they are set. They are symptoms therefore which need to be nipped in the bud. Early and accurate prognosis are indispensable if this is to be done. Ideally we should wish to be able to tell the patient that in so many days, weeks, or months, he will be symptom-free, or fit to return to his occupation; or if there is no such good prospect ahead, to prepare him to restrict his activities and make the best of his disability. In fact, we are, I submit, all too doubtful in many cases of what the future holds, and for this reason the tone of our encouragement is often a little flat, or it may be sharp. Inquiry into the factors which influence the prognosis of brain injury is therefore most desirable. It must be detailed and extensive. Long term follow-up is essential.

We are at present, I think, too much inclined to assess prognosis in terms of those facts which are most easily ascertained. We have learned to discount fracture to a great extent, but there is a tendency to lay too much stress on the presence and duration of traumatic amnesia. It is well known, of course, that a man may suffer a severe localized cerebral injury from a penetrating wound without any loss of consciousness. This is rare in cases of closed head injury, but does occur. A man may, for example, suffer a permanent and totally disabling aphasia from blunt injury without having lost his senses. In a case without focal symptoms, however, the absence of amnesia is generally good ground for a satisfactory prognosis. It is much harder to generalize with regard to the duration of amnesia, when present. In a series of Service patients with closed injuries, the numbers of patients with different durations of post-traumatic amnesia who were invalided or returned to duty have been tabulated. It must be observed that the cases providing this material were a selected group. Most of them had been transferred to a Head Injury Centre because they were doing badly. Moreover, the conditions of duty to which they had to return were exacting as compared with the more flexible conditions of civil life. This, however, for purposes of observation is an advantage, since it may be assumed that the conditions for every patient in the series were comparable, and that the man who relapsed did so because he could not carry the load as well as the others, not because he had to carry a heavier load.

| Post traumatic amnesia | I Number of cases | II Original disposal | | III Proportion of original number invalided later | IV Total invalided |
|------------------------|----------------------|-------------------------|-----------|------------------------------------------------------|-----------------------|
| | | Duty | Invalided | | |
| Less than 1 hour ... | 210 | 81% | 19% | 4% | 23% |
| From 1-24 hours ... | 302 | 78% | 22% | 7% | 29% |
| From 1-7 days ... | 216 | 71% | 29% | 9% | 38% |
| More than 7 days... | 143 | 63% | 37% | 11% | 48% |

perimetry or complete sensory investigation in the routine is a waste of time. On the other hand, failure to examine the sense of smell is a serious omission.

Psychiatric Examination

Intellectual impairment or personality disorder may be evident at the first examination and, if so, will of course be assessed against the estimate of the pre-traumatic state made from the history. The total situation must be taken into account, including the individual's adjustment, responsibilities, plans and ambitions before the accident and his reaction to the change in his environment and prospects following the injury. In many cases, however, a period of observation is essential before a just assessment can be made. This is equally true of civilian and Service patients. In the former the influence of over-anxious and over-sympathetic relatives, in the latter the inclination to exaggerate symptoms in order to evade unpleasant duties, may obscure the clinical picture at the first interview.

Special Investigations

X-rays.—By the time our hypothetical patient comes under neurological observation his skull will almost certainly have been X-rayed, but it is by no means so certain that the report he brings with him will be accurate. Common faults are inadequate pictures, misinterpretation and incomplete description. I suggest that radiologists should agree that when there is question of a fractured skull a standard series of pictures should be taken.

It is equally important that in X-ray reports the extent of the fracture should be described accurately, and with special care in the case of fractures running into, or close to, the accessory air sinuses, and that whenever there is any doubt as to interpretation this should be stated. The distinction between a small linear fracture and a vascular channel is notoriously difficult at times. In such cases the radiologist is perhaps unduly inclined to give the patient the benefit of the doubt, and it is not very uncommon to find a fracture reported when the final verdict decides that none exists.

Examination of the cerebrospinal fluid.—Lumbar puncture very seldom reveals any abnormality of pressure or constituents in the type of case under consideration. It may provoke severe headache in a patient who is on the mend and so impair confidence. It is therefore better omitted from the routine and reserved for the exceptional case in which there are clinical grounds for suspecting abnormalities of pressure.

Air encephalography.—What has been said about lumbar puncture applies with even more force to air injection, which should be reserved for cases in which there is gross evidence of organic cerebral damage from mental or physical examination. In such cases a lumbar or cisternal encephalogram may provide evidence which is of considerable value in pathological interpretation and assessment of prognosis.

Electro-encephalography.—The value of the E.E.G. in post-contusional states has been described so clearly and so recently by Denis Williams (1941) that I shall not recapitulate his findings. In about half the cases showing the chronic post-traumatic state he found an abnormal E.E.G. as compared with a figure of 8% by the same standards in a control group. The E.E.G. must now be regarded as an essential part of the special investigation in any case of severe or moderately severe head injury seen in the later stages. Its value will then be much enhanced if there is a record for comparison taken in the early stages.

Differential Diagnosis

Differential diagnosis in the stage which I am considering is seldom difficult if the record of the earlier stages is adequate.

As to the distinction between the physiogenic and the psychogenic factors in a given case, they appear in most cases so closely intertwined that to separate them is unnatural. I am thinking, of course, of the case in which there is no doubt that organic cerebral damage has occurred. That a man with a hurt brain should have a disturbed mind is to be expected. It is equally to be expected that this disturbance will affect his capacity for adjustment as a whole. What then follows must depend upon the psychological situations to which adjustment is called for. The disorder of function is related not merely to any set task of the moment, but a continuous series of adjustments. This is why our formal psychiatric tests are of relatively little value in assessing disability. We need to get inside the man as far as possible, looking back into his past and forward into his future. Even so, it is often impossible to measure disability except by putting a man to his old occupation for a continuous period of some weeks and seeing what transpires.

It will be understood from what I have said that I regard the practice of dividing the post-contusional cases into two groups, labelling the one organic and the other functional, or neurotic, as unprofitable and misleading.

importance than the duration of the amnesia. If the working rule provided by Cairns were followed there would be liability to error in two groups of cases. The first, which is numerically more important, comprises those in which, after a momentary loss of consciousness, there is mild confusion and automatism with amnesia, often for more than five minutes, and not infrequently for more than one hour, and with complete recovery in the course of a few days. I have seen many such cases in civil practice, and have no doubt that many such occur in the Services which are never seen at Head Injury Centres. The second, and smaller group, is represented by the man whose amnesia is of less than five minutes' duration (it may be nil), who suffers prolonged disability, possibly for several months, on account of localized headache, intellectual impairment, or personality change, probably as the effect of localized cerebral contusion. This group of cases Trotter rescued from the dumping ground of traumatic neurasthenia. It would be a retrograde step to put them back there, yet there is danger lest too close an adherence to the rule of the duration of the post-traumatic amnesia may lead to the assumption that disability beyond the limits of this rule must be psychogenic.

I am aware that I have discussed prognosis at some length, whereas it is not included in our title. I make no apology for this, for I believe that many of the symptoms which we have to treat arise from the uncertainty in the patient's mind about whether he will ever get rid of his headaches, or when he will get back to a normal existence. I hope that the material accumulated by the head injury centres in this country will enable us to get rid of some of the uncertainty in our own minds about the answers to these, and other questions.

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Dr. Aubrey Lewis : During the first few weeks after a head injury it will often be necessary to decide on the causes and pathology of the mental condition. That it is mainly exogenous will be concluded when there has been clear evidence of cerebral damage (I suppose that the term "contusion" is not going to be applied unless there is such evidence) and where some "organic" symptoms have followed close upon the injury. Direct visual evidence of cerebral damage, such as the surgeon may have, will of course often not be forthcoming, and the evidence will then be only of disturbed cerebral functions, among which consciousness will be the most important. The signs of a damaged brain, apart from the focal ones revealed by neurological examination, are much the same whatever the lesion: various degrees and combinations of impairment of memory, grasp, orientation, perception, thinking, affect and spontaneity. When an exogenous brain syndrome, following trauma, has been recognized, the question of aetiology has not been disposed of. The patient's delirium, for example, may be a straightforward delirium tremens far more dependent on his long-standing alcoholism than on his recent head injury; you can call that differential diagnosis, if you like, but it is better to consider it as aetiology since both the alcoholism and the head injury have probably contributed to it. Differential diagnosis is too prone to insist on absolute verdicts between more or less incompatible claims. There are several physical causes to be reckoned with, in acute post-traumatic mental states: alcohol, infections, presenile and senile conditions, cerebral vascular disease, G.P.I., tumours and epilepsy. Besides these diseases, which may occasionally play a large part in causing the acute mental disturbance following head injury, there are constitutional causes predisposing the patient to this or that type of disturbance. His fatuity and euphoria, for example, may be more eloquent of his hypomanic disposition than of a destructive lesion of his brain; his apathy and lack of initiative may be akin to the depression he formerly experienced after a bereavement or an attack of scarlet fever, and may have little to do with his frontal lobes; some patients exhibit schizophrenic syndromes as soon as they have recovered consciousness, others pass by stages through stupor and confusion into schizophrenia. It would be inappropriate to consider here how the catatonic stupor or excitement released by cerebral trauma may be distinguished from the exogenous traumatic syndrome coloured by schizophrenic trends. In either case, however, the previous history of the patient, and especially his personality may be an important guide.

So much for the early conditions, appearing during the first few weeks after injury. There is, of course, no clear distinction between early and late post-traumatic syndromes.

It is apparent from column II in this table that in the assessment of prognosis in a certain age-group and in relation to fairly well-standardized occupational demands, the duration of the post-traumatic amnesia is of value. The longer the duration of the amnesia, the less likely is it that the patient will attain a degree of recovery which justifies the decision to return him to duty. Column III brings out another point. The figures record the percentage of the total number of cases in each group in which a follow-up showed relapse and subsequent invaliding. They show that of the men who had been judged fit for duty after rehabilitation (which included heavy physical training), a proportion were unable to stand up to the demands of Service life and that liability to relapse, or falsification of a good prognosis, progressively increased with the duration of the amnesia. Putting it another way, in men who have apparently recovered from the effects of their head injury, the longer the duration of the amnesia, the greater is the probability that residual defects of cerebral function will be revealed by the crucial test of return to what is, for the Service patient, a normal mode of living.

Column IV shows the total percentages invalidated in each group, including the relapses. This reveals that if the duration of amnesia, without consideration of other factors, were to be taken as the sole criterion of prognosis, the expectation of successful return to duty for those with an amnesia of less than an hour is 77%, as compared with 52% for those with an amnesia longer than seven days.

From whatever angle these figures are viewed, therefore, the value of the duration of the post-traumatic amnesia as an index of prognosis is apparent. It is, however, equally apparent that the duration of amnesia is not the only factor which counts in prognosis. For example, if we were called upon to give an opinion upon the prospects of return to duty for a man who had recently emerged from a post-traumatic amnesia of ten days' duration, without examining the patient, and without reference to any other details of the case, on the basis of these figures, whichever way we decided the chances of our being right or wrong would be about equal. Taken by itself, therefore, the duration of the amnesia does not carry us very far on the road of prognosis. One patient with an amnesia of two or three weeks may be back at duty within four months of his injury, and succeed; another with an amnesia of less than one hour may not get back to duty at all, or having done so, may fail. If, therefore, we are going to make use of the post-traumatic amnesia as a yardstick by which to measure the severity of the injury in terms of prognosis, we should use it with a good deal of caution, and with a keen eye for all the other factors which may weigh the balance in one direction or the other.

Success in the treatment of closed head injuries—and I am thinking now especially of success as measured in terms of the shortest possible period of invalidism—has been hindered in the past by the traditional acceptance of fixed rules, such as that which imposed three weeks flat in bed for every patient with loss of consciousness, however brief, or that which necessitated so many weeks' absence from work after a fractured skull. It would be a great pity if, at this stage of our knowledge, we should enslave ourselves to fixed rules based upon the duration of post-traumatic amnesia. I have stated elsewhere (Symonds, 1941) reasons for supposing that the duration of the post-traumatic amnesia is mainly dependent upon a generalized disturbance of cerebral function, which is reversible. A long duration of amnesia, therefore, is compatible with complete—and rapid—recovery after clear consciousness is recovered. Inasmuch, however, as the duration of the amnesia is a measure of the severity of the generalized disturbance, it is also a measure of the severity of the blow. The greater the severity of the blow, the more likely it is to have caused local structural damage with long lasting or permanent effects, in addition to the generalized, reversible disturbance of function. It is to be expected, therefore, that symptoms of coarse cerebral damage will be observed more often after a long amnesia than after a short amnesia. During the period of clouded consciousness the most important of these symptoms, those indicating mental impairment, are masked. Therefore, it is not until some time after the patient has recovered clear consciousness that the extent of the more lasting effects of the injury can be gauged. It follows that the examination of the patient, and especially the examination of mental function, *after the period of amnesia is over*, is a truer guide to prognosis than the duration of the amnesia itself. These views have been confirmed by the impressions gained from the experience of the past two years, though they have yet to be subjected to the analysis of factual data collected from a large series of cases.

Meanwhile I deprecate the use of such a table as that proposed at a recent discussion before this Section (Cairns, 1942) in which the duration of the post-traumatic amnesia is set out in relation to the shortest time in which ability to carry out full work may be expected to return. For example, the first group taken is that in which the post-traumatic amnesia is from five minutes to one hour, the minimum period of disability for full work being stated as four to six weeks. Certain qualifications are made, but there is no mention of symptoms suggesting focal structural damage, which I believe are of greater

the cerebral damage is treating behaviour as though it were a neurological sign, constant and always referable to some local place of origin. If at the other extreme he concentrates on the environmental factors and the psychological reaction, he may be ignoring the most important aspect of that recent happening which has left its mark on the patient's brain, creating perhaps fairly rigid symptoms (like diplopia, dysphasia or even a headache) and making a pattern of behaviour from which he can now hardly depart.

If such a point of view is held, there is no sense in supposing that one must always decide whether late post-contusional syndrome is physiogenic or psychogenic. There will always be some of the latter in the causation, there may be quite a lot of the former. If one can appraise the physiogenic residue by neurological examination, or electroencephalography or other special device, of course one will do so. Even if it is extensive one will not treat its disturbing effects on function as irremediable; any more than one wants, after appraising the extensive psychogenic side, to psychoanalyse it or regard it as original sin. Before speaking of treatment, however, there is more to be said about diagnosis. Some of the symptoms may be obviously hysterical; sometimes the whole syndrome cries out hysteria. Even so it cannot safely be concluded that there was no structural damage to set these works going: there may have been. The headache, giddiness, lassitude, forgetfulness, insomnia, may differ in time of occurrence, degree and other characters from these symptoms as met with in the earlier stages of post-contusional disorder when the symptoms may be assumed to be chiefly due to structural damage. It would be misleading to infer that they are now, therefore, wholly a motivated construct; they may be hypochondriacal exaggeration of existing physiogenic symptoms. No one would deny that a person free from detectable neurotic predisposition may, after a head injury, become irritable, easily tired mentally and physically, depressive and apathetic, because of cerebral damage. But for one such easy case there are half a dozen or more, difficult ones, by no means clear-cut, and even in this easy case a little carelessness in the handling may result in prolonging or fixing the disability. Such carelessness arises from preoccupation with the antimony—physiogenic or psychogenic—which I have been deprecating for clinical purposes. Research into the problem is a rather different matter.

Assessment of physiogenic damage.—Goldstein and other workers have tried for a long time to use psychological tests to detect physiogenic damage to the brain. Pfeiffer's description of a long array of psychological tests occupied fifty pages of his monograph on mental disturbances after brain injuries in war, and in the twenty years since that was written, a great deal more has been done. It would therefore be impossible to review the matter in any detail. The bulk of recent work has been much influenced by Goldstein who emphasizes the disturbance in "categorical", i.e. conceptual thinking. Tests for conceptual thinking do not, however, cover the available methods. Babcock devised a method of detecting and measuring deterioration which relies on a discrepancy between present capacity and presumptive previous capacity estimated by a vocabulary test. Changes in personality due to head injury are referred by Goldstein to the conceptual difficulties, but may be studied in their own right, as by the Rorschach procedure. Disturbances of memory have been investigated, as in Zangwill's study of the Korsakow state.

The work done in this field by E. L. Trist and his co-workers at Mill Hill has taken account of most of these methods, and attempted to combine a number of different tests in a set that could be administered in a short time, say, half an hour, and would be clinically useful. No single test suffices to pick up deterioration because it is not a matter of independent functions such as memory, attention or intelligence which may each be separately affected. The tests used were modifications of the Shipley vocabulary, Dvoretzki pictures, Wechsler's similarities, a sorting test using shapes, Weigl's colour-form sorting test, and Kohs blocks: other tests such as Vigotsky blocks, Bolles' and Halstead's sorting methods were also examined. So long as the investigation was limited to persons with known cerebral damage, all was well: tests revealed the expected disturbance. This was true of the group of treated general paralytics who were selected as the most satisfactory analogue to the late post-contusional patient: the cerebral damage in them being certain but non-progressive, and often no longer clinically detectable. But when the same set of tests was administered to a group of neurotic subjects in whom there was no reason to suspect any structural damage, some of them behaved very much as did some of the patients with G.P.I.: the same was true of "normal" subjects tested, though this was less evident if one took the set of tests as a whole than if one looked at individual tests. To put a complicated matter briefly, it has become evident that when the diagnosis of organic damage is clinically doubtful, psychological tests cannot as yet be relied on to supply an unequivocal answer; there is no psychological Wassermann reaction here, no skin test, X-ray or blood picture to settle the vexed question. Because

But hitherto I have been considering those developing while the patient is still indisputably suffering from the effects of cerebral damage. If the evidence of damage has been slight, and the period of unconsciousness in particular quite brief, this stage is soon over, though it may be assumed that by a *contusion* something more than a mild or trifling concussion is meant.

The conditions seen during the later stage can be divided into three classes: (1) the semi-chronic or chronic organic syndrome (usually a Korsakow amnesic one or a dementia); (2) the semi-chronic or chronic insanity, usually a schizophrenia; and (3) that common, dubious, psychopathic condition—the bugbear of the clear-minded doctor and lawyer—post-traumatic neurosis and personality disorder. It includes the “minor contusion syndrome” of Symonds, the “psychasthenia” of Mapother, the “traumatic psychopathic constitution” of Ziehen, and the “post-traumatic personality disorder” of American writers. Uncertainty about it turns mainly on the question: Is it due to structural damage or is it psychogenic? The insistence upon this is understandable, but fallacious; understandable, because the somatic pathology of any disorder is of prime importance, and because so many social issues such as attributability and pension rights depend upon the answer to the question; fallacious because it ignores the real state of affairs at present, and asks us to say “Yes” or “No” to a question often unanswerable in that form.

The question presupposes that exclusively physiogenic and exclusively psychogenic cases can occur, and that every case will be at least predominantly psychogenic or predominantly physiogenic. To substantiate such a view, the criteria of psychogenesis or of physiogenesis must be clear and demonstrable. But they are not. Physical damage to the neuraxis can produce all sorts of mental symptoms, including neuroses and personality disorders: encephalitis lethargica is a convincing example of this obvious truth. The ordinary features of an exogenous mental syndrome may be totally lacking. It is therefore impossible to infer whether a mental syndrome is physiogenic or not by study of the symptoms presented in it. There is nothing characteristic of the syndromes thus produced by certain kinds of structural damage, to enable us even to group them together as of structural origin. Are we then to conclude that a condition is physiogenic whenever we can prove existent cerebral damage? Scarcely, because the brain may have adapted itself, as we know it can, to this lesion, and persisting disturbance of function may not then be attributable any longer directly to the tissue damage. Moreover in a large proportion of the cases in question there is no evidence of existent physical damage to the brain.

It could not be said that we are on safer ground in settling the criteria of psychogenesis. It is notoriously easy to find psychological causes if you look hard enough. You can find them in patients with tumours and disseminated sclerosis and carbon monoxide poisoning and all sorts of organic diseases. The adequacy of the psychological motives to account for the symptoms can be so much a matter of personal opinion—one doctor disagreeing entirely with another doctor—that it is hardly to be thought of as a useful criterion in any dubious case. For rather similar reasons the response to a change in the situation or to some psychological device or treatment can be deceptive and ambiguous: even a dramatic change can be produced, say, by hypnosis in the symptoms of a patient with disseminated sclerosis.

These arguments might perhaps be dismissed as casuistry since there are cases where no one is in doubt as to the mainly physiogenic or mainly psychogenic nature of a neurotic illness. But it is because of the lack of any unequivocal and agreed criteria of physiogenesis and psychogenesis that we are so often in a dilemma in diagnosing a patient with a post-confusional neurotic syndrome.

I believe that we have no unequivocal criteria, no final distinction, between physiogenic and psychogenic because the search implies a dualism which is not there. Focal brain damage may produce characteristic disturbances of function, usually seen as neurological signs: gross widespread brain damage may produce disturbances of function, recognizable and characteristic of exogenous mental syndromes (though even these may be closely mimicked by affective disorders and by schizophrenia); but less acute and coarse disturbance may produce nothing that could not also be produced in a man with an intact brain, exposed to stresses of another sort. The patient, as a wholly integrated human being, deals with what happens to him in ways that are determined by his hereditary endowment and previous experiences: if he sustains an injury to his head, his behaviour at any subsequent stage cannot be thought of as simply the sum of his normal functions plus the reduced or altered functions due to this destructive lesion. His behaviour at every stage is a reaction to an existing situation, in which his symptoms at the time, his financial, social, domestic and other difficulties are elements; the form of this reaction will obviously be determined by what has happened to him up to now. It is therefore in principle a plastic response, not a fixed one. The physician who concentrates on

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| 31 to 40 | 20 | 12 | Inert, without Initiative | 11 | 23 |
| 41 and over | 6 | 8 | Rebellious | 18 | 17 |
| <i>Service Patients Only</i> | | | Suspicious | 25 | 20 |
| Service Occupation—Skilled | 12 | 15 | Cyclothymic | 17 | 20 |
| N.C.O. | 6 | 3 | Schizoid | 17 | 20 |
| Category on Enlistment—Not A | 7 | 8 | Hysterical | 15 | 23 |
| Category on Discharge—E | 15 | 25 | Anxious | 30 | 44 |
| Annexure A | 15 | 9 | Hypochondriacal | 21 | 30 |
| Annexure B | 11 | 10 | Obsessional | 8 | 20 |
| <i>Civilian Occupation</i> | | | <i>History of Present Illness</i> | | |
| Professional or Administrative | 6 | 11 | Onset during Training | 11 | 15 |
| Skilled | 18 | 10 | Exposure to Enemy Attack: | | |
| <i>Earnings</i> | | | Severe | 20 | 8 |
| £3 and Under | 25 | 30 | Medium | 11 | 14 |
| £7 and Over | 5 | 2 | <i>Symptoms</i> | | |
| <i>Unemployment</i> | | | Somatic Anxiety | 25 | 28 |
| Much | 7 | 10 | Headache—Mild | 23 | 24 |
| Little | 10 | 17 | Headache—Severe | 25 | 6 |
| Work History: De-graded or unduly frequent changes | 9 | 5 | Dizziness | 27 | 21 |
| Duration of Stay—More than two months | 9 | 16 | Fatigue | 24 | 28 |
| <i>Family History</i> | | | Effort Intolerance | 21 | 18 |
| Psychosis | 7 | 14 | Dyspepsia | 7 | 14 |
| Neurosis, &c. | 22 | 24 | Fainting, &c. | 13 | 3 |
| <i>Personal History</i> | | | Pain | 7 | 29 |
| Upbringing other than by Parents | 1 | 5 | Severe Tremor | 3 | 6 |
| Upbringing Unsatisfactory | 9 | 15 | Stammer | 5 | 8 |
| <i>Education:</i> | | | Enuresis | 2 | 2 |
| Elementary—Poor | 12 | 18 | Sexual Anomalies | 2 | 5 |
| Secondary or Central | 3 | 8 | Anxiety—Mild | 21 | 15 |
| Higher | 2 | 1 | Anxiety—Moderate | 16 | 26 |
| Sex Anomalies | 6 | 12 | Anxiety—Severe | 8 | 8 |
| Social Activity—Much | 11 | 5 | Depression—Mild | 23 | 24 |
| " " —Little | 33 | 43 | Depression—Moderate | 15 | 16 |
| <i>Past Physical Health</i> | | | Depression—Severe | 4 | 1 |
| Medium | 18 | 27 | Apathy | 20 | 11 |
| Bad | 2 | 4 | Perplexity | 4 | 10 |
| Epilepsy | 1 | 0 | Hypochondriasis—Mild | 16 | 12 |
| Previous Organic Disease of Nervous System | 1 | 3 | Hypochondriasis—Moderate | 14 | 17 |
| Previous Organic Disease Elsewhere | 9 | 8 | Hypochondriasis—Severe | 0 | 4 |
| <i>Previous Mental Health</i> | | | Depersonalization | 1 | 1 |
| Symptoms in Childhood | 17 | 17 | Hysterical: | | |
| Predisposed in Adult Life | 18 | 28 | Motor | 4 | 3 |
| Definite Illness | 10 | 6 | Sensory | 16 | 12 |
| Previously Treated in Mental Hospital | 0 | 0 | Special Senses | 6 | 1 |
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| | | | Stress of Bombardment | 15 | 12 |
| | | | " Domestic, &c. | 19 | 14 |
| | | | " Separation, &c. | 24 | 32 |
| | | | <i>Treatment</i> | | |
| | | | Discussion, &c. | 39 | 34 |
| | | | Hypnosis, &c. | 7 | 5 |
| | | | Narcosis or Insulin | 1 | 2 |

Incidence of Head Injury in the Psychopathic Personality

This raises a further question—are people of psychopathic predisposition more likely to sustain a head injury than others? If gunshot wounds were the common form of head injury, the answer would certainly be "No". But while accidents on the roads remain so frequently responsible, a more careful answer is needed. I suppose everyone reading through a series of head injury records is struck by the way some of these patients seem to have been dogged by a malicious fate so that they have had two, three or even four head injuries in the course of five or ten years. It is less likely that one head injury predisposes you to have another than that some people are particularly prone to accidents because of some slowness of reaction in an emergency, defect of judgment, or other trait. Farmer and Chambers, in their industrial Health Research Board Report, No. 84, have shown convincingly that accident proneness is an important factor in motor drivers, leading to repeated accidents, and that experience in driving does not avail to alter the differences between those specially prone and others: moreover they found that certain psychological tests were done badly by those with a high average accident rate. It is not unlikely, therefore, that among those who sustain head injuries in road accidents there will be a higher than average proportion of predisposed, and perhaps neurotically unstable persons. This is not to deny that many who sustain head injuries have previously been well-adjusted, healthy people. It emphasizes, however, the need for looking into the previous personality of the man with a post-confusional syndrome and indeed paying at least as much attention to this as to the extent and persistence of cerebral damage.

the psychological tests do not settle the question of cerebral damage they are not to be thought useless. There are many cases in which the results of these tests are such as could only be yielded by cerebral disease; and in any case, besides the diagnostic issue it is often necessary to know exactly what functions are impaired and to what degree, so that suitable work and treatment may be offered the patient, and the rate of his improvement measured: for these purposes the psychological tests mentioned and others of a more special aptitude-measuring kind are indispensable. These tests, in short, are in the same relation to our routine clinical investigation of memory, grasp, &c., as the standardized intelligence test is to our impression of a man's intelligence: precautions are necessary in interpreting the standardized measure just as in interpreting our rough clinical findings. The validated set of tests, with its items systematically considered and checked, is a more precise instrument, and its findings on different occasions can safely be compared so that one can tell if the patient is improving in particular respects.

There are many other psychological tests, less concerned with the fundamental question of deterioration, that have been used in cases of brain injury. The most interesting and popular of these is the Rorschach inkblot—which is chiefly a personality test. The elaborate methods of interpretation and the obscure terminology of this test make it a rather esoteric affair, and I cannot pretend to be more than a proselyte at the outer gate. Piotrowski has laid down canons of diagnosis for organic cerebral disease by this method, and Harrower-Ericksen in Montreal has made easily understandable contributions to the same matter. It has been plain, however, in the work done by Miss Harvey, Trist and others at Mill Hill, as well as in Ross's paper, that what had been regarded as organic types of response to the test can occur in insane persons, hysterics and other psychopaths, and normal people of poor intelligence, without any organic cerebral affection. The position is, again, that these types of response occur commonly in organic disease but they cannot in a doubtful case, where there are psychiatric symptoms, be used as diagnostically decisive. For some aspects of the patient's personality, the Rorschach findings are illuminating, whatever the cerebral condition.

Survey of a Series of Cases

I have lately made a survey of a series of post-contusional states admitted to a neurosis centre. There were 64 patients in the series, all men, nine of them civilians and the rest soldiers. The form of the clinical syndrome displayed was diagnosed in the usual psychiatric terms, and a group of 64 patients taken for comparison from the other neurotic patients in the hospital. The selection of these was at random except that they were of the same sex, included the same number of civilians, and exhibited the clinical syndromes in the same proportion, as did the head injury cases. Thus there were 16 patients with conversion hysteria in each group, 2 patients with hysterical amnesia, 6 with a severe acute anxiety state, 14 with a chronic anxiety state, and so on. The number of cases, 64, may seem small, but the number of attributes in respect of which they were compared was nearly 150, and covered practically all the main points of psychiatric interest in each case. The main items are shown in Table I.

The points at which the two groups differed significantly (i.e. statistically so) were remarkably few. More men in the control group had been discharged Category E, had as adults shown signs of predisposition to mental disturbance, had been unsociable, weak and dependent, lacking in initiative, over-anxious, hypochondriacal or obsessional. More of them complained of pain (apart from headache) and anxiety symptoms; whereas the head injury cases included, as would be expected, more people who had been of stable, well-organized personality before their illness, and severe headache, fainting and irritability were commoner among them. But the differences in these respects were only on the margin of statistical significance, and it was evident that the head injury series was made up of very much the same sort of people (in family and personal history, intelligence, symptoms, response to treatment, and outcome) as the non-organic group.

It is clear that these post-contusional cases had been sent to a neurosis centre because some doctor thought they were of a particular type: they are not necessarily representative of the minor contusional syndrome, they will include a perhaps unduly high proportion of those whom the doctors referring them judge to have recovered from all physical effects of their trauma. They were, however, very good examples of the syndrome, clinically, and many of them had had very severe head injuries; where they differed from the average case, I think, was in the length of time that had elapsed since the injury, so that features of chronicity and habituation were prominent. However, the striking thing is that the long-standing, relatively intractable post-contusional syndrome is apt to occur in much the same person as develops a psychiatric syndrome in other circumstances without any brain injury at all.

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The points at which the two groups differed significantly (i.e. statistically so) were remarkably few. More men in the control group had been discharged Category E, had as adults shown signs of predisposition to mental disturbance, had been unsociable, weak and dependent, lacking in initiative, over-anxious, hypochondriacal or obsessional. More of them complained of pain (apart from headache) and anxiety symptoms; whereas the head injury cases included, as would be expected, more people who had been of stable, well-organized personality before their illness, and severe headache, fainting and irritability were commoner among them. But the differences in these respects were only on the margin of statistical significance, and it was evident that the head injury series was made up of very much the same sort of people (in family and personal history, intelligence, symptoms, response to treatment, and outcome) as the non-organic group.

It is clear that these post-contusional cases had been sent to a neurosis centre because some doctor thought they were of a particular type: they are not necessarily representative of the minor contusional syndrome, they will include a perhaps unduly high proportion of those whom the doctors referring them judge to have recovered from all physical effects of their trauma. They were, however, very good examples of the syndrome, clinically, and many of them had had very severe head injuries; where they differed from the average case, I think, was in the length of time that had elapsed since the injury, so that features of chronicity and habituation were prominent. However, the striking thing is that the long-standing, relatively intractable post-contusional syndrome is apt to occur in much the same person as develops a psychiatric syndrome in other circumstances without any brain injury at all.

should determine its treatment. More explicitly, one might say that after severe or moderate cerebral contusion, for a while the patient reacts as an average human being whose brain has been damaged at certain points, rather than as a particular human being who is in a particular fix. Later on the individual difficulties and reactions overshadow the general, more or less common, pattern of disturbed cerebral function. But what I said earlier indicates that this needs qualification, and that the influence of the patient's constitution, his past, and his present circumstances must be reckoned with from the outset. It is not possible to reckon with them to good purpose unless one has knowledge of them. This knowledge, which may be obtained sufficiently from a relative of the patient, is in many cases all that is necessary and practicable in the early stages after his injury, and may be all that is necessary throughout; but there are some in the early stages for whom it is not enough, and it is hardly ever enough in those later stages when the "minor contusional syndrome" has asserted itself ominously.

In other words, you are unlikely to succeed in getting rid of the patient's symptoms if you can only surmise what factors are producing these symptoms; and even if your surmise is correct you cannot always deal with these factors by environmental adjustments (through the social worker and the relatives) or by indirect methods only such as those listed under work, recreation and physiotherapy. Direct psychological treatment is called for—not, of course, invariably. I think there is a good deal of misunderstanding about this. Psychological treatment of any sort is good or bad according to its appropriateness in the particular case and the skill with which it is conducted; harmful probing is, of course, unskilful, as superficial dabbling can be, or crude ploughing and plugging. Psychological treatment will not consist in a choice between the extremes—psychoanalysis or a casual chat. As Colonel Cairns put it in that discussion "no attempts at rehabilitation are likely to be successful unless the patient's anxieties and fears are assuaged and unless he is helped through the phases of depression and the other disturbances of feeling that so often beset him during recovery from head injury". These affective disturbances may turn on responsibility for the accident in which others were injured. I lately saw an Army dispatch rider with an obstinate post-contusional syndrome including pronounced hysterical features in whom it required much finesse and persistence in delicate inquiry, before one learnt that he was in great financial difficulties, that these arose out of his attempt to contribute from his scanty Army pay to the support of the orphan of a man on another motor bicycle killed in the accident in which the patient as driver had had his head injured, and that this in turn was linked up with the censure pronounced on him for the accident by the Army authorities, which he considered unjust—they had degraded him—so that he had a strong hatred of his Army superiors and what they stood for. All this tangle had to be cleared up before he could improve. Some of it was cleared up by simple and obvious measures, some only by addressing oneself to the sources of his guilt, depression and resentment. I am not suggesting that in most post-traumatic syndromes one needs to behave as though the patient was one's oyster, but that it is equally senseless to assume he is a clam. At all events, oyster or clam, he will be better for tactful discussion of "what is on his mind"; this will vary so much from patient to patient that general inferences, e.g. about the effects of industrial injuries and the Workmen's Compensation Act can have only partial validity for any individual. To avoid misunderstanding, I should add that I believe social factors to be more important than individual propensities in keeping these residual neuroses going—Dr. Russell Brain's figures illustrated this—and that social adjustment, like social measures of prevention, is the essential preliminary to any treatment and itself a more effective means of treatment than psychotherapy alone can be; but psychotherapy, however brief and simple, or however recondite, should never be conducted as a thing apart from social adjustment, occupation, and the other features now recognized to be indispensable for restoring, as for maintaining mental health. The trained psychiatric social worker is often the person who does most, by direct action, to bring about the patient's recovery from a post-contusional syndrome; but, for this, she needs the guidance of the doctor in touch with the patient's emotional and private problems, and the doctor will not be able to give it who relies solely on a well-ordered, progressively adjusted hospital routine of physiotherapy, occupation and other pursuits to do everything for the patient.

Occupation.—Here too the ground was so admirably covered in the previous discussion that there is no need for restatement. I would only stress that it is profitless to let a man do trivial or absurdly easy work at a stage of his illness when neither intellectual nor affective disturbance prevents him doing something more like the jobs of ordinary life. Occupational therapists sometimes allow the principles suitable for treatment of inert melancholics and semi-stuporose or preoccupied schizophrenics to operate in a different type of illness, so that it is thought a triumph if the post-traumatic patient labours dully and steadily at some dreary repetitive job, or now and again tackles in spurts

The situation arising out of the accident has also to be considered, if the true ends of diagnosis are to be served, for this may be the most potent of all the causes of the post-contusional syndrome. I said earlier that the patient has to react at any stage to the existing situation. I would like to stress that this situation cannot be reduced to a few salient features, any more than personality and behaviour can. To single out the compensation side of it, or the chance it offers of escaping with honour from disagreeable duty, is to overlook a great deal. Often the desire to obtain money is construed by the doctor as the main motive in the patient's continued illness when cerebral damage no longer suffices to account for his symptoms. By no means all the non-physiogenic post-contusional syndromes are hysterical—depression and anxiety are conspicuous: nor are hysterics who claim compensation actuated only by this in the production of symptoms. Loss of employment, insecurity, and many other forms of social pressure are at work, not to speak of the hypochondriacal, anxious and other latent trends now set in motion by the severe threat to his life, his reason or his health which the patient believes he has sustained. His symptoms themselves form part of the situation he must cope with. We can see this easily enough in a man with dysphasia or squint—he obviously has to adapt to the disability—but we tend to overlook it when his symptoms are more of the psychological kind.

So much for the diagnosis of these common and often difficult cases—not so much diagnosis, I suggest, as appraisal of multiple causes, the doctor taking care neither to hunt the snark of physiogenesis to death, nor perfervidly to track the red herring of moral obliquity ("gold digging", "scrimshanking") to its lair.

Treatment

Treatment is more preventive than actual. The damage done by ill-advised treatment in some of these men could not be put right by a demigod. I would mention only what seem the essentials of preventive treatment: (1) To decide early what plan to adopt and, as far as possible, to adhere to it. (2) To let the patient know, as soon as may be, that he will, or will not, have such and such residual disability which will clear up, and that he need fear only so much incapacity, or none at all eventually. (3) To see that misguided relatives or friends do not tell him a highly coloured story of the accident, but that it is explained to him soberly and with due allowance for his amnesia and other symptoms. (4) Not to prolong the period of rest and inactivity, but to institute early some mild work or interests, no more exacting than his state warrants, and gradually to increase both the opportunity for activities and the incentive, taking care on the other hand to avoid overtaxing him to the point where frustration and "catastrophe reaction" could lead to an exaggerated concern over his disability. (5) To help him in any financial, legal or domestic embarrassments to which the accident has conduced: a skilled social worker is here most valuable. (6) To do everything possible to bring the phase of special examinations to an end, except in so far as they are necessary for assessing progress or deciding on special methods of treatment or disposal. I do not here refer to regular definite investigations (whether physiological like the E.E.G. or psychological, like the set of tests mentioned earlier) but to the repetition of X-rays, lumbar punctures, and other procedures which give the patient the impression that the doctors are not sure about him, that they cannot decide whether his brain, or his mind, is seriously damaged; worst of all is it when he passes from hospital to hospital, each repeating the investigations and perhaps reversing the diagnosis or the treatment advised at the previous one. These and other precepts are obvious enough, but they have often been flouted, heedlessly and harmfully, by the time the patient reaches the neurosis centre, at any rate, and I suppose head injury centres could tell the same tale. Much of this over-investigation must arise as I have said from mistaken concern over the question whether the illness is physiogenic or psychogenic—a question that is often the parent of muddle, though intended to bring light and clarity.

Rehabilitation

This subject was discussed three months ago before the Section.¹ A few points, however, call for brief reference. It was said in that discussion that rehabilitation will include occupation (diversional, constructive, and useful to the hospital), physiotherapy, and intellectual and recreational pursuits. There is no mention in this list of any special care for the patient's individual psychological problems. It is obvious that work and physical activities, games and reading all exert some psychological effect, that in this indeed their chief efficacy may lie; but the patient is an individual and unless his private difficulties and attitudes are given sufficient attention, the rest of the valuable routine may not avail to make him well. At different periods after his injury the importance of these psychological problems varies; the later it is, the more they control the illness and

Section of Medicine

President—GEOFFREY MARSHALL, O.B.E., M.D.

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DISCUSSION ON MINERAL AND VITAMIN REQUIREMENTS IN
RELATION TO WAR-TIME DIETARY

Dr. R. A. McCance and Dr. E. M. Widdowson: The diet of this country has been considerably modified by the war, but our nett requirements for the various dietary essentials have not been altered. To maintain ourselves in perfect health we have to absorb just as much calcium, phosphorus and other mineral elements as we had to in 1939. Nevertheless, most of us are eating foods to-day which never graced our tables in the piping days of peace. There has, moreover, been an alteration in the way in which foodstuffs are allocated. Now there is a system of planned food distribution, and the diets of rich and poor are tending to conform more and more to a common pattern. The milk which had begun to find its way in large quantities into the milk-bars has been diverted from them to provide all school children, rich and poor alike, with an extra third of a pint a day. It is therefore necessary to ask ourselves whether these changes in supply and distribution have materially affected our intake of minerals.

From individual dietary surveys carried out before the war, it has been calculated that the present rations of milk and cheese provide men, women and children with as much calcium as they were in the habit of obtaining from those two foods before the war. People are, moreover, probably eating more bread and potatoes to make up for the shortage of fats and sugar, and there is, therefore, no reason to suppose that calcium intakes have appreciably decreased provided that individuals are taking the whole of their rations. The same conclusion may be drawn about iron. The iron gained by the change from white to national wheatmeal bread fully compensates for the iron lost because of the restriction of meat.

The question as to whether these two minerals are absorbed as readily from a war-time as they were from a peace-time diet must now be considered. During the first five months of the war McCance and Widdowson (1941) carried out an experimental study of rationing, and the results suggested that calcium and magnesium were much less freely absorbed from the experimental than they had been from the pre-war diets. Although most foods in the experimental diet were severely restricted, potatoes and bread, made from 92% extraction flour, were allowed in unlimited quantities. Metabolism experiments were next carried out on eight subjects over a period of nine months, and the effects of white and brown bread on mineral absorption were compared (McCance and Widdowson, 1942a, b; Widdowson and McCance, 1942). Forty to fifty per cent. of the calories in these experimental diets came from the cereal undergoing investigation. It was found that the subjects, each and all, absorbed calcium and magnesium very much better from the white than the brown bread diets. Iron was also better absorbed. Brown bread evidently interfered in some way with mineral absorption. The first explanation suggested for these results was the laxative action of the brown bread, which was sufficient to increase the weight of the stools by two to three times. The second explanation was that the phytic acid in brown bread might have precipitated the calcium and the iron in the intestine and so prevented their absorption. To understand why phytic acid was considered as the noxious agent, it is necessary to go back to the work which was done by Mellanby soon after the conclusion of the last war. Mellanby stressed at that time that cereal diets tended to promote rickets in growing puppies and that while brown flour was worse, in this respect, than white, oatmeal was the worst of all. At that time Mellanby did not succeed in isolating the rachitogenic factor, which he referred to tentatively as a toxamine. It was not until 1934 that Bruce and Callow, working with rats, suggested that the toxamine might be inositol hexaphosphoric acid, a compound already well known for some twenty years, and present in quite large amounts in the outer parts of the cereal grains. Its rachitogenic action was attributed to the fact that its phosphorus was not so freely utilized as inorganic phosphorus. But while admittedly rickets in the rat is generally brought about by phosphorus deficiency, the disease in dogs and men is due to a failure to absorb enough calcium. Nevertheless, since inositol hexaphosphoric acid has very insoluble calcium, magnesium and iron salts, it was evident that it might precipitate these metals in the intestine and so promote rickets by preventing calcium absorption. In 1939, Harrison and Mellanby showed that the addition of sodium phytate to puppies' diets did in fact lead to rickets. No one, however, had shown directly that phytic acid affected

a task well below his actual powers. The patient is thought to be doing excellently when he applies himself assiduously to painting butterflies on glass or disfiguring wood with poker work mottoes. Many men with late post-contusional symptoms are content to loiter along in these pointless activities, which are as demoralizing as idleness. It is necessary at all stages to have the man doing something worth while; not of course flying too high and becoming upset or dispirited at failure, but not, on the other hand, accepting resignedly a low level of work, and aiming only at what would be exclusively leisure pursuits for him, e.g. rugmaking or raffia work. What I am advocating was tersely put by Dr. Brain when he said "occupational therapy should merge into therapeutic occupation"—but in some hospitals the rule that any occupation is better than no occupation seems still to be the high-water mark of aspiration.

There is no need to discuss at length the question of physiotherapy or of treatment of special disabilities such as dysphasia and epilepsy. As for the "intellectual and recreational pursuits", there is much to be said for making these unobtrusively part of the therapeutic plan. It would, of course, be foolish to tell the patient what he is to read, and to try to control every detail of his day; but if he is to have, let us say, as a soldier, some educational lectures while in hospital, let these have a bearing on real problems, stimulating his interest, but also providing incentives to counteract those which are perpetuating his symptoms. To illustrate this from actual experience would take too much time. There is, however, one still experimental instance of this which shows how education as commonly conceived and education as part of the psychological treatment can be combined: the doctor collects his patients in a group, talks to them a little about some familiar difficulty or misconception that often crops up when he is examining them alone or inquiring about their notion of their illness, and then invites them to ask questions. By this means prejudices and wrong attitudes can be to some extent corrected and the doctor's time economized. One of my colleagues, Dr. Jones, has used this method as an adjunct to treatment of a rather similar group (patients with cardio-respiratory neuroses) and has found it useful and economical. None of these methods is sufficient by itself.

The "demoralization", or psychopathic change in personality, that may follow brain injury, especially in children, is a more difficult business, though at bottom it is the same problem. I cannot say that I have seen outstanding success in the treatment of genuine instances of this; spurious instances are of course common and may do well. It is like the post-encephalitic behaviour disorders: you can palliate by training, but that is all.

A few words are necessary about the later forms of post-traumatic insanity. They have an incidence of nineteen per hundred thousand of the male population in the corresponding ages. The rate rises from 10 in the 20-29 age-group, to 16 in the 30-39 group, 20 in the 40-49 group, and 25 in the 50-59, 60-69 groups. This increased incidence as age advances cannot be accounted for by an increase in accidents sustained, but must be construed as another instance of how the ageing process is itself, with its reduced functions and loss of resilience, a very prominent cause of these traumatic psychoses. In short it is more an involutional or presenile disorder here than a traumatic one, and the age distribution very similar to that of presenile and other degenerative organic psychoses. Mayer-Gross and Feuchtwanger have dealt very fully with a series of post-traumatic schizophrenias, showing how diverse the factors and course can be. The persistent amnesia or Korsakow states and dementias are seldom uncomplicated by alcoholism, atheroma, senile or other somatic disease. It will depend on these other factors—constitutional or morbid—and not upon the injury itself whether the psychosis follows hard upon the accident or there is an intervening period of apparent recovery. This explains I think Mapother's observation that where schizophrenia or a paranoid syndrome supervenes after an interval of normality, the prognosis is worse.

The late post-contusional states are an exemplar of what social and preventive medicine means in the neuro-psychiatric field. The best the doctor can do may fall short because adverse genetic and social forces are at work which he cannot remove, and the effects of which he can only lessen. The least of his obstacles will often be the residual physical damage, and here it is therapeutically and clinically better to look to what is intact or repairable, to the undamaged tissues and the unimpaired and compensating functions, rather than to allow the irremediable structural damage to set a limit in advance to what may be worked for or attained.

The answer of course is that it is now possible to demonstrate conclusively that ill-effects do arise from partial deficiencies—or sub-clinical deficiencies would perhaps be the better term. Much of the evidence of the reality of sub-clinical deficiencies is quite recent—some of the most striking is only a few months, or even weeks, old.

The evidence for sub-clinical deficiencies: general.—The work to which I refer relates especially to the *specific* effects of partial deficiencies of particular vitamins. Sub-clinical deficiency of a more general type was well recognized before the war. The classical work of this type was that by Corry Mann; children having extra milk acquired a better state of nutrition than the supposed "normals", as shown by their improved gains in weight and height, by increased high spirits (naughtiness), glossier hair, better finger nails, &c. The same conclusion, that is that the average is frequently not the normal, has been proved repeatedly by similar trials all over the world: whether by the supply of extra milk, by the provision of Oslo breakfasts, by the beneficial results of supplementary feeding during maternity, and perhaps most strikingly by the demonstration that the wide gap between social groups (physique, morbidity) could be narrowed or bridged by equalizing the feeding. It is important to recognize that in such trials the evidence consists of statistical differences in the groups as a whole; there is not necessarily any obvious improvement which can be detected in each individual on superficial clinical examination.

Effects of undernutrition in specific factors.—Turning to specific dietary essentials, an excellent example of a sub-clinical deficiency is seen in anæmia. Helen Mackay proved that a large proportion of young working-class children and mothers had a moderate degree of anæmia—so moderate in fact that it would usually escape notice at medical inspection. Thus the first reaction of the clinician tended to be: "if the child looks healthy and has no evidence of suffering any disability therefrom, surely such a degree of anæmia must be 'normal' or 'physiological' or of no consequence." But what Mackay proved was that the provision of extra iron as well as raising the hæmoglobin levels reduced the morbidity rates in the group—there was therefore a true sub-clinical deficiency.

The effects of sub-clinical deficiencies of vitamins may be instanced by consideration of two of them, vitamin B₁ and vitamin C. The specific results of mild hypovitaminosis as first observed in experimental animals agree very well with those which can be detected in human subjects and indeed have furnished valuable clues as to what to expect.

Sub-clinical deficiency of vitamin B₁.—In young animals, growth is sub-optimal in the absence of sufficient vitamin B₁. A relatively small dose of vitamin B₁ will prevent symptoms of beri-beri; a somewhat larger dose will do no more than merely maintain constant weight; a still larger dose will procure mediocre gains in weight; and increasing doses can still be shown to have some appreciable effect in further improving the weight-gains up to quite high levels of intake—although to all intents and purposes the animal appears normal on inspection without such increased intakes. Three well-defined specific effects of "sub-clinical deficiency" of vitamin B₁ in animals are (1) sub-maximal growth rates, (2) gastro-intestinal hypotonia, (3) a latent metabolic defect (in carbohydrate metabolism). There is now enough knowledge available to make it clear that the same three phenomena are seen in man. (1) From America and Canada we have evidence, in numerous publications, that babies and children given extra vitamin B₁ improved their weight gains from "average" to a truer "normal". (2) The weight of evidence supports the contention that constipation was lessened in those groups of children having the additional vitamin B₁. (3) Finally, my own work and that of my colleagues has proved that tests for the latent defect in carbohydrate metabolism may indicate low levels of intake in man even when there are no symptoms of the more advanced deficiency.

Sub-clinical deficiency of vitamin C.—In young guinea-pigs an intake of vitamin C sufficient to prevent scurvy or obvious illness is not necessarily sufficient—

- (1) to promote maximal gains in weight.
- (2) to permit adequate formation of new tissues—e.g. (a) in production of bones and teeth and (b) in regeneration and the healing of wounds,
- (3) to promote fullest resistance to infection.

As an instance of the third factor my colleague Dr. Kodicek and Dr. P. D. S. Murray have found that guinea-pigs given experimental fractures develop infections at the site of the fracture when the diet is partially deficient in vitamin C, i.e. containing enough to prevent scurvy but less than the true optimum. Recent observations prove that the same three "sub-clinical" effects can be detected in man, viz. sub-optimal growth, poor healing of wounds and diminished resistance to infection.

Likelihood of partial deficiency of vitamin C in war-time.—The League of Nations' standard for the requirement is 30 mg. per day. Does it matter if people receive less than this amount, provided they do not develop scurvy, or provided they do not receive less than the amount recognized as adequate to prevent scurvy (about 15 mg. per day)? Let us weigh up the arguments for and against.

the absorption of calcium, and no previous experiments had been done on man. Some commercial phytin was therefore purchased, and converted to sodium phytate, and this was added to the dough in the preparation of white bread so that the bread contained as much phytic acid as the brown bread previously investigated. Metabolism experiments were then carried out as before, on diets in which this bread contributed 40% to 50% of the total calories. Absorptions of calcium and magnesium were depressed even more than they had been on the brown bread diets, and some subjects excreted more calcium in the faeces than they took in the food. Phytic acid was therefore definitely incriminated as the noxious agent in brown bread.

Metabolism experiments showed that vitamin D in physiological doses did little or nothing to improve the absorption of calcium by adults. The addition of enough calcium carbonate to the bread, however, effectively precipitated all the phytic acid and so left the calcium in the remainder of the diet available for absorption. This was the cheapest and simplest way of correcting the bad effects of brown bread upon the absorption of calcium. Fortification of the food would be the only way to cope with an iron deficiency due to phytic acid. Recently it has been found possible to study the absorption of calcium from a "dephytinized" bread. It was found impracticable to "dephytinize" wholemeal flour and to retain its palatable properties. Bran, however, could be dephytinized enzymatically, and the product has been used to reconstitute a flour with all the laxative properties of brown flour but containing no phytic acid. Metabolism experiments have been carried out with bread made from this flour and these experiments have clearly shown that although the hydrolysis of the phytic acid in brown bread does improve the absorption of calcium, it does not make the absorption as good as it is from white bread unless the inorganic phosphates which are formed by the hydrolysis of phytic acid are also removed. This "dephytinizing" process is quite impracticable on a commercial scale for the whole country, but small quantities of patent foods for children or invalids could undoubtedly be produced.

The Government has now introduced a flour containing 85% of the original wheat. This will bake a loaf with properties intermediate between those of white bread and bread made from 92% extraction flour.

There is evidence that diets tended to be short of calcium before the war. They may or may not contain as much calcium to-day, but what they do contain is probably not so well absorbed. It is suggested therefore that war-time bread should be fortified with calcium.

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Professor R. A. Peters said in extension of Dr. McCance's remarks about the need for fortification of 85% extraction flour with Ca, and in view of the considerable disturbance in the Press about the addition of calcium carbonate, it was worth recording that for the last thirteen months he and his family (four to six persons) had lived on home-baked 85% wheatmeal bread to which an addition of CaCO_3 (in the form of creta preparata B.P.) had been made to the maximum amount recommended of 14 oz. per 280 lb. flour, or about 0.5 g. Ca per 1 lb. bread. As would be expected, not only had no ill-effects whatever been found, but health had been excellent. In addition radiograms (exhibited) of the eldest member of 53, by Dr. Kemp of the Radcliffe Infirmary, showed no evidence of stone in the kidney or even of any calcification of the femoral artery. Though this was evidence from only one family, it did nevertheless show that for over thirteen months it was possible for a subject of this age to live on the small chalk addition without harm, and it could be produced as evidence against the critics.

Dr. Leslie Harris: *Partial deficiencies*.—My own investigations during the war have been concerned with tests on groups of people to determine their levels of nutrition in various specific dietary essentials. The object has been to ascertain whether their nutritional status has deteriorated or not as a result of the war—in other words whether they are up to or below given standards of nutritional adequacy, and if below such standards to measure quantitatively how much below.

The saturation test as a quantitative index.—One method which I have particularly used is the so-called "saturation test", applied for example to vitamin C (although equally applicable to vitamin B₁, or to the P.P. factor, &c.). This test enables us to express on a quantitative scale the degree of the deficit: i.e. just how far below the accepted standard of intake the subject has sunk.

The reality of partial deficiencies.—A fundamental issue has to be faced at the start. There are still those who say that scurvy is not much seen to-day, and that beri-beri is almost unknown in England; so that there is no need to determine levels of nutrition.

tokens of falling levels—this can be done by examination of representative groups of the population.

Saturation tests for vitamin C.—The principle of the "urinary saturation" method for vitamin C, to which I alluded at the outset, is as follows: Standardized test doses of vitamin C are given daily under controlled conditions, and the number of days is counted until the subject is approaching his plateau of excretion. With children who have been receiving the reputed requirement of vitamin C (30-50 mg. per day¹), saturation, so defined, is attained on the first or second day. With developed scurvy seven to ten days of such dosing are needed (this being a measure of the extent of unsaturation of the tissues). An intermediate number of days needed to saturate denotes an intermediate level of nutrition, in proportion—as has been proved by control tests on groups of subjects receiving various graded intakes. Thus we have satisfactory standards of reference. Fortunately, the amount of "scatter" in the responses of different subjects receiving the same known intake is relatively small, and less than might perhaps have been expected. To remove a misapprehension which still seems to persist, it is necessary to emphasize that we are not concerned with "saturation" or "unsaturation" as such—e.g. whether saturation is desirable or otherwise—but with a quantitative measurement of the degree of saturation, merely as an indication of the level of the past intake.

Results with vitamin C.—Our tests during the war (*Lancet*, 1942 (i), 642) have shown one thing in common with those before the war. Children at a well-conducted residential institution were consistently at higher levels of vitamin C than those from poor working-class homes. However as a result of the war both groups had sunk to considerably lower levels than the corresponding groups examined at the same season before the war. This conclusion applies to the results after the winter, and it can undoubtedly be attributed to the relative shortage of fresh fruits and other carriers of vitamin C during the winter in war time. (It may be noted that potatoes are the principal source of vitamin C in winter, and about 11 oz. of them daily are needed to provide only one-half, 15 mg., of the League of Nations requirement of the vitamin, 30 mg.) Perhaps the most striking result was this seasonal variation. After the summer all groups of children examined were at a reasonably high level. After the winter, however, most of the poor working-class children examined during the war needed four days and upward to saturate, whereas before the war the great majority of such children have been saturated within two to three days. Such results emphasize the need for care in providing vitamin C during the winter months. They amply justify the decision of the Government to set aside supplies of vitamin C for young children many of whom, as we have demonstrated, may be severely below standard in the absence of such precautions.

Vitamin A.—My colleagues and I have been specially interested in the dark-adaptation test for detecting sub-clinical deficiency of vitamin A. The method can be made sufficiently specific for vitamin A by the simple expedient of ascertaining whether each subject fails to improve in absence of vitamin A but responds after it has been administered in large quantities. Experimentally it has been repeatedly proved that individuals deprived of vitamin A show a progressive fall in their dark adaptation curves and improve when vitamin A is restored to their diet: we therefore have here an excellent example of a sub-clinical deficiency—the individual is free from clinical signs and symptoms and the presence of an undoubted defect is only discovered on laboratory examination.

Some years before the war my collaborators and I found a fairly high incidence of dysadaptation due specifically to low intake of vitamin A in certain poor districts in London and Cambridge, and this was correlated with a low consumption of milk among the children found to be deficient. The position is undoubtedly better now, notwithstanding the war, for very much more milk is drunk in schools than was then, margarine is fortified with vitamin A, and—a surprising enough observation—raw carrots in cellophane wrappings are supplied by tuck shops and relished by the children in the absence of the wonted sweets and chocolates. My colleague Dr. John Yudkin who has been carrying out nutritional surveys on factory workers and school children during the war has found low adaptation in 9% of children at an urban institution, 15% among urban children from poor homes and 18% among some village children. He has confirmed that the majority of such subnormal children responded after vitamin A. Among medical students and nurses 6% were low in adaptation, judged by arbitrary standards and a higher proportion among factory workers.

An alternative method of detecting deficiency of vitamin A, suggested by Kruse, is to examine for ocular changes (in the conjunctivæ) by means of a slit-lamp: as Kruse admits, however, these ocular lesions are not the sole, first or most important abnormality. Moreover the impression of my colleagues is that the changes detected by slit-lamp seem also less frequent and less severe in children than in adults.

¹ For alternative estimates of the requirement see *Lancet*, 1942 (i), 645.

(a) *Con.*—Zilva (*Biochem. J.*, 1941, 35, 1240) says: "As far as the civilian population of this country, leading a normal life, is concerned, the natural supply of vitamin C during the greater part of the year is so superabundant that, even allowing the widest margin for destruction in the cooking and the preparation of the food, the intake is more than adequate to supply the vitamin C requirements." Fox and his colleagues (*Brit. M. J.*, 1940 (ii), 143) found that among miners receiving 12 to 25 mg. of ascorbic acid daily only 12 out of a total of 950 developed scurvy; one man only in a group of 950 labourers receiving 40 mg. of added vitamin C developed symptoms suggestive of mild scurvy—the general health of the two groups was not otherwise different. In the experiment of Crandon, in which a human volunteer was given scurvy experimentally, the deleterious effect on wound healing was not apparent until a relatively late stage in the development of deficiency and the subject did not become infected (Crandon *et al.*, *New England J. Med.*, 1940, 223, 353).

(b) *Pro.*—Dealing first with the observations just referred to, I would argue that the mere absence of infection in certain cases or groups does not prove that subjects low in vitamin C do not in fact have a lowered state of resistance to infection. It is not to be supposed that all subjects on diets low in vitamin C are necessarily bound to become infected—it must obviously depend on the nature of the organisms to which they happen to be exposed and on various local conditions. In my own case, when I was restricted to a scurvy-producing diet I did indeed develop an infection (whooping-cough) followed by prolonged infection of the respiratory system which delayed my recovery. My own single positive experience admittedly proves little or no more than one other worker's negative experience—what seems significant however is that the balance of the evidence, in recent large-scale investigations, does support the view that the addition of extra vitamin C to war-time diets has increased resistance to infection, has promoted healing of wounds and has improved weight and height gains in children. This new literature is so important that it is worth summarizing it:

Effect of extra vitamin C on resistance to infection, wound healing, and physique.—

(1) In Germany, 1,600,000 children were given 50 mg. of vitamin C daily in addition to the ordinary war-time diet: the children having the extra vitamin C showed improved annual gains in weights and heights and a diminished rate of incidence of infections (*Bull. War Med.*, 1941, 2, 6; see also *Ernährung*, 1941, 6, 289).

(2) In Britain, 1,500 adolescent naval trainees took part in a similar experiment. The duration of tonsillitis (although not its incidence) was lowered in those having the extra vitamin C, and complications were lessened. 16 cases of rheumatic fever and 17 of pneumonia occurred in the control group, no single case in the supplemented group (Glazebrook and Thomson, *J. Hyg.*, 1942, 42, 1).

(3) The later work of Crandon and his associates contradicts an interpretation put by some workers on his earlier study of experimental scurvy. It establishes the importance of adequate intake of vitamin C for the healing of surgical wounds. "A study of pre-operative diet and pre-operative plasma vitamin-C levels of patients having operations upon the biliary tract was made. Those with poor intake or low levels or both had a higher percentage of post-operative herniae than those with better levels" (Lund and Crandon, *Ann. Surg.*, 1941, 114, 776). The investigations of Hunt, the experience of the surgeons at St. Bartholomew's Hospital, and much other evidence all confirm the relation between a sub-optimal intake of vitamin C and the poor healing of wounds.

(4) Similarly, it has been reported that the addition of vitamin C has aided the healing of the alveolus and gingiva after dental operation (Campbell and Cook, *Brit. Dent. J.*, 1942, 72, 6) and has reduced the incidence of some types of gingivitis (Roff and Glazebrook, *J. Roy. Nav. M. Serv.*, 1939, 25, 340; *Brit. Dent. J.*, 1940, 68, 135; Campbell and Cook, *Brit. M. J.*, 1941 (i), 360).

(5) Indirectly, the increased incidence of infantile scurvy (e.g. Paterson and Daynes, *Brit. M. J.*, 1941 (ii), 787) and of scorbutic gingivitis (reported by several observers) supports our contention: because, for every such case of developed deficiency disease, there must be others of less advanced, or sub-clinical deficiency.

Need for tests to measure levels of nutrition.—If we are satisfied, then, that there is such a thing as a partial deficiency—and facts similar to those just summarized could be cited for the other vitamins—we may next consider how it is to be detected. Two facts emphasize the urgent need for such tests. First, the onset of advanced deficiency may be disconcertingly abrupt: for example a subject with a low prothrombin figure due to hypovitaminosis-K may be free from hæmorrhage, but the moment this value falls below a certain critical threshold hæmorrhages will suddenly supervene. It is of supreme value therefore to be forewarned. Similarly, the transition from a state of sub-clinical deficiency of vitamin C to actual scurvy may be precipitous. Secondly, it is important to be able to follow the effects of restrictions in the national diet and keep a watchful eye on any

year, the largest group was the 90-95% division. I have no reports of an increase in the Hb. compared with pre-war in any section of the community.

Sir John Boyd Orr's survey of 1935-6 showed that the poorest 10% of the community averaged 8 mg. of Fe as the daily intake, and that 50% of the community had an intake of less than the 11.5 mg., a figure taken as the requisite minimum. Since then, rationing and restrictions have made alterations in the nation's diet. The poorest to-day can scarcely afford to buy their share of the rationed animal protein food. It is possible that the limitation of sugar increases their consumption of flour and potatoes, which helps to increase their mineral intake. The richest section is restricted in variety, but can still find sufficient unrationed foods to maintain a diet of high mineral content.

In August 1941 I made a careful survey of prices, which I sent to the Ministry of Food, showing that for an active man a complete optimum diet with reasonable variety could not be purchased for less than 14s. a week, and this amount was spent only by Boyd Orr's most wealthy 10%.

A similar diet for a sedentary worker costs 11s. a week, an amount spent by one-third of the community in 1935-6. Wages, and the prices of foods have risen since then.

Now the rationed foods—meat, bacon, eggs, cheese—contain about 25.6 mg. Fe in the week's ration, of which only part is available. Authorities vary on this question—the available Fe being given as from 7 to 14 mg., i.e. 1 to 2 mg. per day. In 1935-6 the poorer half of the community obtained half of their 10 mg. Fe per day from bread and flour, potatoes, and legumes. Apart from the poorest 10% it is unlikely that these people will eat much greater bulk of these foods, unless they are doing harder work.

The rationing of meat, bacon, eggs and cheese reduces their intake of Fe from 3 mg. to 1 (or from 6 mg. to 2 if one takes the higher values quoted above), so that there is a theoretical expectation of an increasing anaemia among the poorer half of the nation.

The problem arises as to how to increase the Fe of their diets.

If the Fe of the whole wheat berry is available for absorption, the increase due to the substitution of 85% extn. meal for 75% (i.e. white flour) would increase the Fe of the diets of the great majority by 2.5 a day. (I take the figure of 8 mg. Fe per lb. of 85% extn. bread, as given me by Professor Drummond.) This would bring the total Fe to over 10 mg. a day.

| | |
|---------------------------------------------|------------------------|
| From rationed foods | 1.0 mg. |
| bread and flour | 5.5 mg. |
| potatoes and legumes (Boyd Orr) | 2.0 mg. |
| points rations, unrationed meats, fish, &c. | 2.0 mg. |
| | <hr/> 10.5 mg. per day |
| Substituting white bread for 85% | 8.0 |

Before I consider the availability of the Fe of bread, I should like to clear the air with regard to the "man v. cow" problem, in the light of what I have learned from agricultural and milling experts.

From the point of animal feeding stuffs, it is necessary to estimate the effect of raising the extraction of wheatmeal to 85%.

As we all know the wheat berry consists of a central endosperm (or white flour), occupying 75% of the whole berry, and containing a small amount of Fe. Outside this is the germ layer—2% of the berry. This in turn is surrounded by fine bran, called weatings, and outside all is the coarse bran. Germ, weatings and bran are all rich in iron.

Coarse bran is a complete, balanced food for the milch cow. The weatings, however, are not, and are fed to pigs and poultry, which have not the necessary apparatus to make use of bran, on account of its roughage.

As the coarse bran of cattle feed is not used for the manufacture of 85% meal, the substitution of the latter for white flour will not affect our milk supply. But the use of some of the weatings in the new flour will deprive pigs and poultry of a part of their present food supply. Even the entire removal of bacon and eggs from the nation's rations (and this, of course, is far from the case) would cause a loss of Fe of less than ½ a mg. per day.

Looked at from the point of view of mineral constituents, the innovation of 85% extraction bread increases the Fe of the average diet by about 2.5 mg. per day, without disturbing the supply of Ca or P which comes from milk. Superficially then, from this standpoint, the change in bread is all gain.

It is now essential to consider fully the rather startling report of Widdowson and McCance (*Lancet*, 1942 (i), 588), suggesting that the higher extraction meals are a poorer source of available Fe than is white flour. From the data obtained from their experiments their conclusions appear just, but they cut so firmly across one's general ideas that one scrutinizes them with a mind even more critical than usual.

Partial deficiency of riboflavin.—About 4% of the school children so far examined at Cambridge were found on examination of the cornea by slip-lamp to have pathological vascularization at the limbus—the sign used by Sydenstricker and others to indicate pre-clinical deficiency of riboflavin.

Hæmoglobin.—In the experience of my colleagues, anæmia (hæmoglobin value of under 80%) was found among factory workers (Birmingham district) in 20% of the women and 5% of the men.

B vitamins.—A saturation test, somewhat similar in principle to that used for vitamin C, is available for vitamin B₁ (Wang and Harris, *Biochem. J.*, 1939, 33, 1356; Wang and Yudkin, *ibid.*, 1940, 34, 343) and also for nicotinamide (Harris and Raymond, *Biochem. J.*, 1939, 33, 2037; Kodicek and Wang, *Nature*, London, 1941, 148, 23). For vitamin B₁₂ a special carbohydrate-tolerance test is also a possibility. The substitution of flour of 85% extraction for white flour has, however, largely removed the likelihood of serious deficiency of both these factors. As Kodicek (*Lancet*, 1942 (i), 380) pointed out the previous war diet with white bread was dangerously near the margin of adequacy for nicotinic acid, and it may be remarked in this connexion that there was the suggestion of an increased incidence of pellagra in N. Ireland (Deeny, *Brit. M. J.*, 1942 (i), 147).

Conclusion.—Recent work has made it abundantly plain that ill-effects result from partial deficiencies even when signs and symptoms of frank deficiency disease are absent. Methods are now available for assessing the levels of nutrition in specific dietary essentials. Results obtained in surveys, using certain of these methods, are described in this contribution. The need for such surveys is particularly apparent with the restrictions in diet imposed by the war.

Dr. H. M. Sinclair reminded the meeting that the discussion was on "Mineral and Vitamin Requirements" and not on the incidence of malnutrition in the population or the reality of partial deficiencies.

Dr. Harris had quoted the excellent work of Fox which, however, was not wholly negative. Fox had 950 natives in each group, one group getting 12.25 mg. vitamin C *per diem* and the other an additional daily 40 mg. Apart from the incidence of scurvy, there was no significant difference between the two groups: weight, general health, physical efficiency, resistance to infection, healing of wounds and fractures were all studied, and the additional vitamin C failed to have any effect upon them.

Iron deficiency was one of the most prevalent deficiencies in the present diet and the Blood Transfusion Service enhanced it: we took blood, kept the plasma and threw away the cells (or, when we were more sensible, we used them for garden manure). Viewed academically, the cells, supplemented perhaps with dried blood from slaughter-houses and a few iron railings, might be added to the nation's bread, but some would regard that as cannibalism. From the practical point of view there was an overwhelming case for adding to bread traces of iron salts that were available to the body.

Dr. A. P. Meiklejohn: It would be interesting to hear Dr. McCance's opinion on the advisability of adding traces of iron salts to the bread.

Would Dr. Harris be prepared to give a more exact definition of "subclinical" deficiency? An individual who excretes only a small amount of a given vitamin, but subsequently, after receiving large doses of the vitamin, has a greater excretion, was not necessarily suffering previously from an impairment of health through lack of this vitamin. Even if he says that he feels better after receiving the vitamin, the possibility of improvement through suggestion must be kept in mind. The results of the "test-dose" method of assessing subclinical deficiency need to be correlated with the actual state of health as assessed by clinical methods.

Dr. R. A. Murray Scott: From the clinical point of view anæmia is the only mineral deficiency likely to be evident. Unfortunately we find no large-scale statistics to show whether anæmia is more prevalent to-day than pre-war, yet what reports are available show a present-day tendency to increase in anæmia.

There are descriptions in the literature of increased anæmia among small groups of pregnant women and nurses. Medical and obstetrical opinion in my part of the country (and in some other parts) quite definitely assert increasing anæmia and more Fe used by doctors and chemists: a survey of miners being conducted in Yorkshire at the present time shows no gross anæmia, but examination of their wives brings to light a considerable reduction of N. Hb. among the latter. The reason being their menstrual loss, and the fact that the miner frequently eats the whole of the animal protein food in the ration of the family.

Leeds second year medical students have for several years estimated their own Hb. When the results are divided into groups covering 5% divisions, the numerically largest group has been in the 100-105% Hb. division, for the six years prior to this war. Last

year, the largest group was the 90-95% division. I have no reports of an increase in the Hb. compared with pre-war in any section of the community.

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| | | | | | 10.5 mg. per day |
| Substituting white-bread for 85% | ... | ... | ... | ... | 8.0 |

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One could have wished for more clinical data—evidence of lack of energy or fall in Hb. when the subjects were on the brown bread diet, evidence as to whether they showed a tendency to an increase or decrease in the absorption of Fe on the third week of brown bread diet compared with the first week; i.e. evidence of the possible acclimatization to brown bread; evidence as to whether any of them was an habitual eater of brown bread prior to the experiment, how many of the subjects had loose stools on brown bread, how much Ca their diets contained and whether there was enough to supply their needs of this metal and in addition precipitate any phytic acid in the brown bread (for we cannot allow Mellanby's dogs to get rickets and Widdowson and McCance's subjects to become anæmic at the expense of the same phytic acid); evidence as to the age and state of physical health of the 8 subjects of experiment.

This problem of availability of mineral constituents of higher extraction meals is of particular importance in view of recent work on the value of the components of the vitamin B complex, which must be kept firmly in mind. (Meiklejohn, *New England J. Med.*, 1940, 223, 265, 224, 420; Williams, Mason, Wilder and Smith (Mayo), *Arch. Int. Med.*, 1940, 66, 785; &c.)

One must not forget the other side of the picture (here I confess I am being quite non-scientific)—those country folk the world over who eat wholemeals of one sort or another, the schools which supply only whole wheatmeal, and those who from preference or persuasion eat little other cereals, and are usually very healthy, vigorous, non-anæmic persons.

Let me give an illustration from my own household. Two adults—one of each sex, both aged 41 years—both from preference eat wholemeal almost entirely. They occasionally eat a little national bread. Both eat porridge regularly—another source of phytic acid. Subject A eats all vegetables in fair quantity, thus finding a source of additional Fe and Ca. Subject B has average menstruation, eats little potato and less green and root vegetables. Of other foods both are small eaters, the rationing of meat scarcely alters their diets. I find in their diets no other source of Ca or other mineral to precipitate phytic acid except those provided by an average war-time diet. Both subjects are extremely active persons. Yet on May 23, 1942, Subject A had an Hb. of 100%, Subject B 102%. How does their Fe escape the clutches of the phytic acid?

This is a matter not only for speculation, but for examination. Our ancestors thrived on wholemeal, and some of us obviously can thrive on wholemeal. When given a diet of white flour for some years, does our metabolism lose its ability to deal with phytic acid? On presenting it once again with wholemeal does it in time resume a lost function? In other words, does phytic acid always steal minerals from the diet, or are there other circumstances under which the P of phytic acid becomes precipitated? To conclude:

- (1) There is evidence of anemia in this country—probably greater than pre-war.
- (2) Most diets contain less total Fe than pre-war.
- (3) It is important to solve the question of how to increase the available Fe in the nation's diet, preferably through the medium of bread as the staple food of most diets.

I suggest:

- (1) If the medical profession and scientists are in future to advise on (and I hope direct) the health of the nation, an Annual survey of Food, Health and Income is imperative.
- (2) Experiments should be done to find what salt of Fe can be added to bread to increase the available Fe without interfering with other qualities. I have not succeeded in finding a description of such work in connexion with the enriched flour used in U.S.A.
- (3) The very interesting experiment of Widdowson and McCance should be repeated on a larger number of persons with full clinical control.

Dr. Geoffrey Evans reported his experience of a variety of deficiency states, and spoke of glossitis in which some cases were relieved by nicotinic acid: of cheilosis and several cases of acne rosacea much improved, and indeed on occasion apparently cured, by the administration of riboflavin: of vitamin B₁ deficiency shown in loss of appetite and gastro-intestinal disturbance: of intellectual lethargy shown in mental fatigue and lack of power of concentration, as well as cases of neuritis, in which last also it seemed that vitamin B₁ given in larger doses had contributed to recovery. Dr. Evans also spoke of the relation of vitamin C deficiency to gingivitis. He expressed the opinion that vitamin D deficiency was on occasion responsible for muscular hypotonia, in terms of which he explained certain cases of so-called rheumatism in the back. In relation to vitamin D deficiency he mentioned a family in which there seemed to be evidence of failure of vitamin D assimilation or utilization in spite of adequate supply, in which family there was evidence of this deficiency in two generations. He particularly emphasized the frequency with which in his practice he found a subnormal hæmoglobin content of the blood. Dr. Evans concluded that in his experience minor deficiency disease is of quite frequent occurrence.

and that so far as vitamin supply is concerned, because of the variety of deficiency, for the improvement of their health the population should be made food conscious rather than vitamin conscious.

Professor J. R. Marrack said that in March and April he had estimated the ascorbic acid in a number of meals, as served, both in school canteens and in British restaurants in Hertfordshire and in Bermondsey, Islington and Leytonstone. He had found no evidence of excessive destruction of ascorbic acid in the preparation and cooking of the vegetables. Taking average helpings, the total ascorbic acid supplied by one-third of the meals was under 10 mg.; one-third supplied over 20 mg.

He had made saturation tests on boys at three schools. At School A the average amount of ascorbic acid in a dinner was 8 mg.; one-third of the boys were saturated on the third day of the test. At School B, average ascorbic acid 16 mg.; two boys out of three saturated on the third day of the test. At School C, average ascorbic acid 24 mg.; one-third of boys saturated on first day, two-thirds on the third day. Except for one boy at School C (saturated on first day) who ate raw vegetables at home, the boys depended almost wholly on their dinner for their supply of ascorbic acid. All three schools had garden plots, School A grew a large amount of potatoes, which were plentiful and cheap, and did not produce enough green vegetables which were scarce and dear. School B grew a better choice of vegetables and supplied a raw vegetable meal (ascorbic acid content 21 mg.) once a week. School C grew abundant early green vegetables. The choice of vegetables grown in the garden plot determined the amount of ascorbic acid in the school dinners and the state of nutrition of the pupils in respect to vitamin C.

Too much faith should not be placed in the use of raw vegetables. The weight of vegetable, that would be regarded as an ordinary helping when cooked, looks an enormous amount when raw. In the first week of April he had estimated the amount of ascorbic acid in the plasma of girls at an Institution in which raw vegetables were served. In all the concentration was under 0.3 mg. per 100 ml.

Lieut. W. R. G. Atkins, F.R.S., asked Dr. Leslie Harris for what period it was possible to detect previous saturation with vitamin C and stated that forty men who had had large doses of the vitamin in January, up to saturation in the majority, had been examined again four months later. Many were then indistinguishable from their fellows who had never been dosed. The men of the group as a whole were only slightly higher in vitamin C than those who had received no added vitamin. Thus the administration of extra vitamin appeared to result in an increased utilization or excretion, or in both.

In the discussion on mineral requirements Lieut. W. R. G. Atkins drew attention to the importance of foods derived from the sea, since the latter was a reservoir for many or all the trace elements. Some, however, such as iron, were precipitated, so that sea water was richer in copper than in iron. Copper indeed was a constituent of the respiratory pigment of the crustacea, as was vanadium in the holothurians.

Mr. A. L. Bacharach thought the only possible explanation of Fox's results was that both groups of natives were suffering from multiple deficiencies; in that event, it was quite likely that the only demonstrable effect of ascorbic acid would be to prevent frank scurvy, which it seemed to have done in a significant number of treated cases. The effect of multiple deficiencies in preventing an individual vitamin from exhibiting its normal function was nothing new, as, for example, had been shown in the well-known Peterhead investigation. Unsaturation with vitamin C was, as Dr. Harris had pointed out, an indication of the state of nutrition of groups, and not of any individual, at a given time. It could, however, be an indication of the level of nutrition of an individual if, as a result of repeated observations, it were found to be chronic. It seemed impossible to avoid the implication that the permanently unsaturated individual must be nearer the scorbutic border-line than the permanently saturated one, even though the latter were actually receiving more than was necessary for the prevention of any specific sub-scorbutic manifestation.

Dr. McCance (in reply) stated that there was some evidence that hæmoglobin levels were lower now than in 1938, but that he felt more careful investigations should be made before any dogmatic statements were made. He thought that very small amounts of iron could be added to bread quite satisfactorily, unless the addition catalysed the destruction of any of the vitamins. Iron could also be added to table and cooking salt, and this might be a more practical way of increasing the nation's iron intake. Apropos of his experiments with Dr. Widdowson which had been discussed by Dr. Murray Scott, Dr. McCance pointed out that although there had been the fullest possible clinical control, there were no clinical data to give. He himself and his subjects had been very well all through the nine months of experimentation. There was no evidence of "acclimatization" to brown bread, nor would he expect to find any. Dr. Scott would find answers to his other queries in *J. Physiol.*, 1942, 101, 44.

Dr. Harris (in reply) said that Dr. Sinclair had questioned whether in this discussion, dealing with "Mineral and Vitamin Requirement in Relation to War-Time Dietary", a contribution on partial deficiencies and their incidence was in place. He maintained—in agreement with the Committee, who had invited him to speak on that particular topic—that it certainly was. The evidence which he had tried to present proved that various groups of apparently normal subjects when given, for example, extra vitamin C in addition to their war-time diet benefited, on balance, in such ways as improved physique, diminished incidence of infection and better healing of wounds. How, then, could one pretend adequately to discuss vitamin requirements in relation to war-time dietary without a consideration of the fact that an intake which was sufficient to prevent deficiency disease was not always sufficient to prevent these sub-clinical defects, or without noting that the minimal requirement was therefore very different from the optimal requirement, and hence without some attempt to estimate whether war-time dietaries did or not meet the reputed requirements for optimal nutrition (i.e. by measurement of incidence of undernutrition).

Dealing with resistance to infection, Dr. Harris said that the balance of evidence indicated that it was liable to diminish when the intake of vitamin C was low. This of course did not mean that every such person, or every such group would *always* become infected—it would depend on the extent to which they were exposed to infection and other limiting factors. Hence the negative experience in Fox's test, to which Dr. Sinclair had once again alluded, was not conclusive. A negative result in a trial of this kind might mean very little, whereas a positive result was of more significance. Similarly for healing of wounds and the other effects mentioned—taking into account all the published data, the balance of evidence was for a positive effect.

In reply to Dr. Meiklejohn's request for a definition, Dr. Harris said that a group of children had sub-clinical deficiency if, although apparently normal at inspection, it could be shown statistically that after a period on an increased vitamin intake they improved as a group (e.g. in yearly gains in weight and height, morbidity rate, and in the other effects mentioned) as compared with controls kept on the unsupplemented diet. There would not necessarily be a detectable improvement in each individual. Numerous trials had now proved that such sub-clinical deficiencies did in fact exist.

Dr. Meiklejohn had missed the point in asking whether there was really any need for saturation. Dr. Harris had never claimed that there was. As he had been at pains to emphasize in his paper, the object of the saturation test was to determine quantitatively the relative position of the subject or group in relation to any accepted standard of intake. Control tests had shown that with various graded levels of intake of the vitamin there resulted corresponding graded levels in the degree of saturation, as measured by the number of days needed to saturate. Hence from the result of the saturation test one could say whether a given subject had a level above or below that corresponding, for example, with the L.O.N. standard (30 mg.). If some alternative standard of intake were preferred, the results of the saturation test were still applicable, just as a series of hæmoglobin estimations did not lose their significance whether one preferred to refer them to a standard of 100% or of 95%. The chain of arguments connecting saturation tests with clinical findings was as follows: It was known that a dose of about 15 mg. of ascorbic acid was just sufficient to prevent actual symptoms of scurvy in most people; allowing a small margin, the L.O.N. had fixed the daily requirement as 30 mg.; according to some U.S.A. authorities the true optimum was probably 50-75 mg.; health had improved in trials in which ordinary, marginal intakes of vitamin C as consumed in the war-time diets had been supplemented with additional vitamin; and, finally, the purpose of saturation tests was to enable the observer to determine the relative position of the subject or group within such scales of intake. The question of whether or not a person felt better after a test dose did not of course arise, and the supposed analogy with the excretion of a drug like bromide after medication was entirely false.

In reply to Dr. Atkins, Dr. Harris said that vitamin C was not stored in the body to furnish a reserve to any considerable extent. In this respect vitamin C (and the other water-soluble vitamins) stood in contrast with the fat-soluble vitamins. The length of time taken for scurvy to develop in guinea-pigs, and apparently also in man, was not considerably increased even if the subject was given a great surplus of the vitamin, instead of his normal allowance of it, for a time before going on the deficient diet. Any surplus thus given was mostly excreted. Thus in the saturation test one was not concerned with the body's "reserves" so much as with the "degree of saturation" of the tissues. In other words the effect of a massive over-dosing was not lasting, and the result of the saturation-test indicated the average level of intake during the few months prior to the test.

Section of Comparative Medicine

President—G. DUNLOP-MARTIN, M.R.C.V.S.

[February 18, 1942]

DISCUSSION ON THE CONTROL OF THE DISEASES OF CATTLE INIMICAL TO MAN

Mastitis and Streptococcal Infections

[For previous Discussions see PROCEEDINGS, 35, 115, 469, 478]

Dr. A. W. Stableforth discussed mastitis with reference to a series of tables under the following headings: the various kinds of mastitis, the importance of mastitis from a national viewpoint, the financial loss, the methods of diagnosis, sites in which *Str. agalactiae* is found and methods of infection and the various methods of prevention, control and treatment. The three more important tables are reproduced.

TABLE I

| The kinds of mastitis | The bacteria concerned | The outstanding features |
|------------------------------------|-------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------|
| (1) Common chronic strep. mastitis | <i>Str. agalactiae</i> (<i>Str. mastitidis</i> cont. Group B (Lancefield) Mastitis Group I) | Widespread, incidence 35% of milking cows; many carriers or mild cases, but causes more clinical mastitis and loss of milk than any other |
| (2) More acute strep. mastitis | <i>Str. dysgalactiae</i> (Mastitis Group II) | Sporadic, acute or subacute, few carriers, serious in some herds |
| " " | <i>Str. uberis</i> (Mastitis Group III) | Sporadic, usually subacute, some carriers |
| " " | <i>Str. pyogenes</i> equi ("Animal pyogenes" Group C (Lancefield)) | Uncommon, acute, carriers rare |
| " " | <i>Str. pyogenes</i> (Group A (Lancefield)) | Rare, acute, carriers rare |
| (3) "Summer mastitis" | <i>C. pyogenes</i> | Heifers and dry cows, acute, purulent toxic, very serious in some herds and years |
| (4) Staphylococcus mastitis | <i>Staph. aureus</i> | Usually mild, chronic, fairly widespread; but may be acute, toxic and gangrenous |
| (5) Coliform mastitis | Various coliforms | Rare, acute, toxic |
| (6) Tuberculous mastitis | <i>M. tuberculosis</i> | Nearly always "cold," chronic and focal |
| (7) Other forms | <i>F. necrophorus</i> , <i>pasteurella</i> , &c. | Rare |

TABLE II.—WHY MASTITIS IS IMPORTANT FROM A NATIONAL VIEWPOINT

- (1) Occasional loss of the cow or more frequently loss of one or more quarters or the production of unsaleable milk.
- (2) Increased herd wastage.
- (3) Lowered milk yield even in non-clinical cases.
- (4) Changes in milk quality: decreased fat, casein and sugar, increased alkalinity and salt content which may interfere with cheese-making.
- (5) Mastitis may be caused by human streptococci and the milk be dangerous to human beings drinking it.

TABLE III.—THE LOSS FROM MASTITIS.

- (1) Comparison of 86 opposite infected and non-infected quarters (*Str. agalactiae*).
Milk yield of infected, 21% lower
Butter fat of infected, 25% lower
- (2) Comparison of yield of approximately equal numbers of affected and non-affected over 373 lactations in 3 herds.
Corrected for calvings, dry period, month of calving, &c.
Affected gave 10.8, 10.5 and 19.5% less milk
Take loss at 12% and incidence of mastitis infection at only 25%.
Cows in England and Wales = 3,200,000
25% affected = 800,000
Average yield = 450 galls.
12% loss = 54 galls.
Total loss = 43,200,000 galls. at 1/6 gall. = £3,240,000.
(N.B.—In these herds nearly all mastitis was of the common chronic type met with in most herds and there was no outbreak.)

The Control of Str. agalactiæ Mastitis

Str. agalactiæ mastitis is a chronic disease in which a large proportion of infections remain latent and despite the low morbidity rate, it is responsible for more clinical cases than any other and for a loss of milk greater than all other forms of mastitis.

As far back as 1887 the organism now generally known as *Str. agalactiæ* was labelled *Str. mastiditis contagiosa*; and it is to be presumed therefore that it was regarded as contagious and infected cows isolated. Later, when it was recognized that many cows showed slight changes in the milk at an early stage, various indirect tests were made use of to detect carriers: from 1906 onwards the sediment test, from about 1919 the reaction test and cell count and, a little later, the chloride test, catalase test and certain others. In 1933 evidence was brought forward in this country which appeared to show that the disease could be eradicated within a reasonable time by three-monthly cultural milk tests and segregation or sale of the infected animals. In one herd no *Str. agalactiæ* had then been found for three and a half years and none were in fact found during the succeeding six years whilst promising results were being obtained in other herds. Reports suggesting that the disease might be controlled by similar methods but with the aid of chemotherapy appeared from abroad at about the same time and others affirming eradication have since appeared. Meanwhile we had continued our attempts to eradicate the disease in other herds and, finding that new infections continued to appear, introduced a liquid enrichment medium. In this way a certain number of additional infected cows were found. It was also found, however, that these additional new infections, undetected by the plate method, continued to be found by the enrichment method and, secondly, that many of the new infections so found were never detected again or only infrequently and, moreover, were seldom followed by clinical symptoms or more marked infection. In 1935, therefore, we reverted to the plate method alone. This method though scientifically unsatisfying in view of the findings already mentioned seems of value because its use has resulted in the reduction of clinical *Str. agalactiæ* mastitis to negligible amounts, i.e. it appears to have a real practical value. Since 1935, many other reports have appeared showing that the more searching the methods and the more frequently they are used, the greater the number of infections detected. It has also been shown that *Str. agalactiæ* can be resident on the skin of the udder and milker's hands. Further that an infection can be detected in a higher percentage of freshly calved heifers than was at first believed (percentage is probably about 10). All of this raises seriously the question whether regular cultural examinations are worth while. When eradication within a reasonable time could be held out as a probable result the cost of regular cultural examinations was clearly justified. With the knowledge now before us, however, the question is more debatable and in present circumstances we have to realize that as part of a national scheme regular cultural examination is quite impracticable.

The Parasitic Standing of Str. agalactiæ

The position as already referred to also raises another question of some importance, viz., the parasitic standing of *Str. agalactiæ* and, associated with this, the host-parasite relationship in *Str. agalactiæ* infections and the importance of those factors previously generally regarded as predisposing or secondary factors. There are available, certain facts bearing on this question.

In a large proportion of clinical cases of mastitis *Str. agalactiæ* is the only organism to be found and it is present in large numbers. It is known to ferment lactose and clot milk and mastitis secretions are often acid though secretions less altered are relatively alkaline; the alkalinity may be, however, the result of a compensatory mechanism for the injection of acid solutions into the udder is quickly followed by an alkaline reaction. *Str. agalactiæ* has not been found in the bovine except in the udder or places closely associated with it, e.g. the skin of the udder, milkers' hands, bedding; and, in one laboratory, in a small percentage of faeces. Attempts to set up infection experimentally (in adults) have, with one exception, only been successful when the route was the teat or teat canal. On the other hand heifers may be found to be infected at the beginning of their first lactation: the exact route of infection is not known but infection probably occurs in calfhood. In self-contained infected herds all or nearly all of the infecting strains usually belong to the same serological type and subtype and even show the same minor cross reactions, and this serological herd similarity has also been established in certain outbreaks amongst heifers, or cows which were previously negative, i.e. there is evidence that a given type spreads in the herd. The incidence of both *Str. agalactiæ* infections and clinical symptoms increases with age. On the other hand, it is well known that of two herds with an equally high incidence of the same type of *Str. agalactiæ*, one

may have practically no clinical mastitis, whilst the other has a great deal, and in two herds which are similar as regards incidence one may suddenly show an outbreak of clinical symptoms. Sometimes this is referable to some outside factor such as the introduction of a milking machine. Sometimes nothing can be found to account for it. Again, although on the whole, clinical symptoms of a minor or major nature increase with the age of the infection, many cows can carry a heavy infection for years without showing symptoms of a marked nature and indeed often without showing any symptoms at all, whilst other animals in the same herd and carrying the same numbers and type of streptococci show marked symptoms.

What are the reasons for these paradoxes? Are they really dependent on the factors already well recognized, such as inefficient milking and management in its broadest sense and on injury, or is there some other factor, virus, physiological or hormonal which decides whether the streptococci shall exert their potential pathogenic effects?

There is much to be learnt about *Str. agalactiae* mastitis and indeed mastitis in general and certain aspects stand out for early study.

More figures are required for the incidence of the various kinds of clinical mastitis.

The study of the treatment of mastitis should include mastitis other than *Str. agalactiae* mastitis.

A careful comparison might usefully be made of the value of control by (a) hygienic measures alone, (b) hygienic measures plus simple indirect tests and (c) hygienic measures plus regular cultural examinations.

Finally, an intensive study of the unknown factors in the pathogenesis of the disease is urgently needed.¹

Dr. V. D. Allison: Bovine mastitis is predominantly an infection due to streptococci, with staphylococci next in order of importance, and infections caused by streptococci and staphylococci are problems in both human and veterinary medicine. Milk-borne streptococcal outbreaks in human beings are caused by strains of streptococci belonging to Lancefield's group A, *Str. pyogenes*, while streptococcal mastitis in cows is pre-eminently the result of infection with streptococci belonging to Lancefield's group B. Streptococcal mastitis does not bear the same importance in relation to public health and the practice of preventive medicine as that shown by infections such as tuberculosis and brucellosis.

The distribution of groups of hæmolytic streptococci from various sources in human beings in the absence of disease is shown in Table I, and shows that group B streptococci are infrequently found in human beings. Table II, reproduced from a paper by Hare

TABLE I.—GROUP DISTRIBUTION OF HÆMOLYTIC STREPTOCOCCI ISOLATED FROM HUMAN BEINGS IN THE ABSENCE OF DISEASE

| Source | No. of strains | Group distribution per cent. | | | | | | | | | | Authors |
|--------------------|----------------|------------------------------|------|-----|------|-----|------------|------|------------|----|-----|--------------------------------|
| | | A | B | C | D | E | F | G | H | K | ? | |
| Nose and Throat | 100* | 63 | 5 | 15 | — | — | — | 13 | — | — | 4 | Hare (1935) Coffey (1937-8) |
| | 50† 45 | 11.1 | — | 20† | — | — | 24 42.2 | — | 50 26.7 | 16 | 10 | |
| Fæces | 109 | — | 5.5 | 0.9 | 84.4 | 0.9 | — | 8.3 | — | — | — | Smith and Sherman (1938) |
| Vagina ante-partum | 11 | — | 45.5 | 9.1 | — | — | — | 45.5 | — | — | — | Lancefield and Hare (1935) |
| Vagina post-partum | 66 | 1.5 | 39.4 | 7.5 | 39.4 | — | 3.0 | 4.5 | — | — | 4.5 | |

* Producing soluble hæmolysin. † Not producing soluble hæmolysin. ‡ Includes group G strains.

TABLE II.—GROUPS TO WHICH STRAINS OF HÆMOLYTIC STREPTOCOCCI FROM INFECTIONS IN HUMAN BEINGS BELONG.*

| Source | Disease | No. of strains examined | Streptococcal group | | | | | | | | | |
|------------|-----------------|-------------------------|---------------------|---|----|---|---|---|---|---|---|---|
| | | | A | B | C | D | E | F | G | H | K | ? |
| Throat | Scarlet fever | 117 | 115 | — | — | — | — | — | — | — | — | 2 |
| | Tonsillitis | 77 | 73 | — | 3 | — | — | — | 1 | — | — | — |
| | Ac. rheumatism | 9 | 9 | — | — | — | — | — | — | — | — | — |
| Skin | Erysipelas | 23 | 23 | — | 5 | — | — | — | — | — | — | — |
| Uterus | Puerperal fever | 194 | 189 | 1 | 2 | — | — | — | 1 | — | — | 1 |
| | Abortion | 12 | 8 | 2 | 1 | 1 | — | — | — | — | — | — |
| Tissues | Lymphangitis | 15 | 14 | — | — | — | — | — | 1 | — | — | — |
| Totals 452 | | | 431 | 3 | 11 | 1 | 0 | 0 | 3 | 0 | 0 | 3 |

* Reproduced from HARE, R. (1937), *Canad. Pub. Health J.*, 28, 554, 596.

¹ Whilst detailed reference to the literature on mastitis cannot be made, the writer would express his indebtedness to his one-time collaborators, and in particular, Dr. F. C. Minett, his former chief.

(1937), similarly shows the distribution of groups of hæmolytic streptococci found as the cause of disease in man and again it is seen that Group B streptococcal infections play a very minor role.

Table III summarizes the human group B infections which I have been able to cull from the literature of the last five years. It is interesting to note that all the cases

TABLE III.—HUMAN INFECTIONS WITH HÆMOLYTIC STREPTOCOCCI BELONGING TO GROUP B.

| Authors | No. of cases | No. fatal | No. with endocarditis | No. associated with pregnancy |
|---------------------------------|--------------|-----------|-----------------------|-------------------------------|
| Congdon (1935) ... | 1 | 1 | ? | 1 |
| Lancefield and Hare (1935) ... | 7† | 0 | 0 | 7 |
| Colebrook and Purdie (1937) ... | 1 | 1 | 1 | 1 |
| Coffey (1937-8) ... | 3 | 2 | ? | 2 |
| Fry (1938) ... | 3 | 3 | 2 | 3 |
| Hill and Butler (1940) ... | 12* | 4 | 2 | 12 |
| Pomales-Lebrón (1940) ... | 1 | 1 | ? | 1 |
| Rosenthal and Stone (1940) ... | 1 | 1 | 1 | 1 |
| Ramsay and Gillespie (1941) ... | 16† | 2 | 2 | 16 |
| Fry (unpublished) ... | 4 | 2 | 1 | 3 |
| Total | 49 | 17 | 9 | 47 |

* Including some minor infections.

† Genital tract sepsis in 13 cases, none severe.

‡ All minor infections.

§ No data available.

(except one reported by Coffey (1937-8) in which the sex is not mentioned) occurred in women and that 47 out of a total of 49 were associated with pregnancy. Seventeen of the cases proved fatal and at least nine of these had endocarditis. It would be interesting to speculate on the source of these group B infections in women. Taking into consideration the somewhat high incidence of group B streptococci found by Lancefield and Hare (1935) in the vagina of women both ante-partum and post-partum, and the finding of group B streptococci in normal faeces by Smith and Sherman (1938), I suggest that the gut is probably the main source of infection. Lancefield's (1940-41) findings indicate that the same specific types of group B streptococci may be found from both human and bovine sources, although Simmons and Keogh (1940) had concluded that types of group B streptococci indigenous to man and cattle are quite distinct and that human carriers are not, therefore, the source of infection in cattle. Further investigation of this question is necessary. In addition to the infections in Table III, Brown (1939) and Rantz and Keefer (1941) have reported a number of cases of human infections with group B streptococci; these do not lend themselves to inclusion in the table, but the strains were isolated from the blood during life and post mortem, from pelvic abscesses, arthritis, gangrene, sinusitis, &c. More than half the cases in which the group B strains were considered to be of ætiological significance proved fatal. The sex of the cases was not given but probably a few were males; in view of the now almost routine procedure of grouping hæmolytic streptococci from human infections by the Lancefield test or Fuller's modification of it, group B streptococcal infections in the male appear to be uncommon.

At the Emergency Public Health Laboratory in Cardiff, during a period of fourteen months ending on January 31, 1941, a total of 1,283 strains of hæmolytic streptococci isolated from throat swabs of cases of acute upper respiratory tract infection were examined and grouped. Table IV shows that there was only one strain of group B

TABLE IV.—DISTRIBUTION ACCORDING TO GROUPS OF HÆMOLYTIC STREPTOCOCCI ISOLATED FROM THROAT SWABS OF CASES OF ACUTE UPPER RESPIRATORY TRACT INFECTION FROM 1.11.40 TO 31.12.41.

| Streptococcal group | ... | A | B | C | G | Total |
|---------------------|-----|-------|------|------|------|-------|
| No. of strains | ... | 1148 | 1 | 96 | 38 | 1283 |
| Percentage | ... | 89.48 | 0.08 | 7.48 | 2.96 | 100 |

streptococci in the whole series and it was not considered to be of ætiological significance. There were no Group B organisms among strains of hæmolytic streptococci similarly isolated from the nasal passages of 83 subjects.

In milk-borne streptococcal infections in man due to *Str. pyogenes*, the opinion that the udder of cows may become naturally infected with hæmolytic streptococci of human origin was expressed by Savage (1911, 1931, 1937) and supported by Minett (1932, 1937), and Bendixen (1937). Confirmation was adduced by Bendixen and Minett (1938) whilst the demonstration by Pullinger and Kemp (1937) of the general failure of *Str. pyogenes* to multiply in fresh milk under normal conditions of storage gave indirect support to

this view, and there is now a much wider acceptance of the view that milk-borne streptococcal outbreaks in man, which are widespread and persistent, are the result of udder infection. On the other hand the evidence (Allison, 1938; Henningsen and Ernst, 1939) suggests that explosive outbreaks of short duration or sporadic cases do result from direct infection of the milk from a human case or carrier.

The main reservoir of *Str. pyogenes* is the human throat and all udder infections of the cow with this organism may ultimately be traced to this source.

Smith (1939) resolves the control of milk-borne disease in general under three headings: (1) the control of disease in animals, (2) the control of infections and carrier conditions amongst the personnel engaged in the dairying business, and (3) pasteurization. Purely from the public health point of view Bendixen *et al.* (1937) strongly recommend that all liquid milk for human consumption should be pasteurized or boiled, and there seems to be little doubt that universal pasteurization, properly carried out, would lead to the disappearance of milk-borne diseases. Milkers and persons handling milk, suffering from acute infections of the upper respiratory tract, to which one might add septic infections of the skin and those with aural discharges, should be excluded until they are clinically well or found to be free from infection with hæmolytic streptococci and staphylococci. This compares with the current practice in human medicine of excluding midwives and maternity nurses suffering from an acute infection of the upper respiratory tract, sepsis of the skin or infective discharges, from contact with puerperal women, a practice which is now being gradually extended to other special hospitals and to general hospitals.

It is with some trepidation that I mention the use of face masks as an aid to the prevention of droplet infection of milk or the transfer of infection via face and hands to the cow. The wearing of masks in human medicine has spread from the operating theatre to maternity practice, and is now being increasingly employed in surgical and otorhinological wards and in the dressing and treatment of war wounds. The dangers are inefficient masks and the development of a false sense of security, and the main difficulty would be that of prevailing on personnel to wear them, if such a recommendation were thought advisable. There is no doubt that the wearing of efficient masks intelligently used would reduce droplet infection to a minimum, and combined with disinfection of the hands, infection of the udder with group A streptococci could largely be avoided. Whether group A streptococcal mastitis is of sufficiently frequent occurrence to warrant such a step is doubtful.

It is interesting to note that the sulphonamides, even in large doses, appear to be without effect on the course of group B streptococcal infections in man.

Staphylococcal mastitis is of sufficient importance to merit some mention. There is still much to be learnt about the factors involved in the causation and spread of this infection, but human and veterinary medicine meet on common ground in regarding staphylococcal infection as of mutual interest, although from different angles. Staphylococcal food poisoning in man is not infrequently spread by milk, and follows not only the extraneous infection of milk, but also the excretion of enterotoxigenic staphylococci in the milk of cows with staphylococcal mastitis and even in the milk of apparently healthy cows. From the public health point of view pasteurization has been shown greatly to reduce the hazard, as the enterotoxigenic staphylococci are largely destroyed and fortunately the heat-stable enterotoxin is not preformed in the cow's udder. Plans to control mastitis in cows due to *Str. agalactiæ* must inevitably have a favourable effect on the incidence and spread of staphylococcal mastitis.

In conclusion it has been my duty to discuss bovine mastitis from the medical and public health aspect, but in doing so I am not unmindful of the different outlook of the veterinary profession on the subject. Any attempt to solve the problem must take both points of view into consideration and to this end co-operation of the medical and veterinary professions is essential, so that formulation of plans to control bovine mastitis shall not be resolved into control of the spread of group A streptococcal mastitis from cow to man via milk versus control of the spread of group B streptococcal mastitis from cow to cow. Plans to control the one must be complementary to plans to control the other and must be so drawn up as to gain the sympathetic co-operation of the farmer.

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Mr. S. L. Hignett: *The treatment of mastitis especially that type arising from infection with Streptococcus agalactiae (group B streptococcus).*—It is now generally agreed that in most cases the eradication of *Str. agalactiae* mastitis from dairy herds is not a practical proposition. Although some control can be effected by frequent testing of milk samples and arranging for infected cows to be milked last, insufficient is known of the pathogenesis of the disease to permit of eradication as a national plan. Research workers have shown that the previously accepted theory that infection is carried by the milkers' hands does not explain all cases; for heifers which from calfhood have not been in contact with milking cows have been shown to harbour the organism when they first calve down. For this and similar reasons many efforts have been made to supplement segregation by treatment of the affected animals.

In 1923 solutions of acridine dyes were first introduced into the udder via the teat canal. Early workers selected certain dyes, some preparations being found better for latent mastitis, others for clinical cases where the mammary secretion is changed in character. Recently entozon and acriflavine were shown to give 60% and 80% respectively of bacteriological cures in cases of latent mastitis when infused into the udder on two occasions at one week's interval.

Our programme included the introduction of antiseptic solutions (acriflavine, euflavine, dettol and quindoline methochloride (flaviquin)) into the udder via the teat canal, and the administration of sulphonamides by the mouth. Euflavine was made up in 4% lactose solution because it had previously been reported that even less mammary reaction was produced by a neutral acridine dye if used in a solution of lactose. In our experience there was no difference between the reaction obtained with euflavine in lactose solution and acriflavine in water. In all cases where udder infusion was employed the solution was made up in sterile distilled water, wherever possible all four quarters being treated. The udder was well stripped out and washed and 100 to 200 c.c. of antiseptic solution at body temperature were injected through teat syphons into each quarter and immediately stripped out. This was to wash out the galactophorous sinuses. Each quarter was then injected again until the tension was that normally present at milking time; it was then gently massaged. If the cow was in milk the solution was withdrawn after five minutes and the udder was stripped out several times during the remainder of the day. Milk secretion was suppressed for twenty-four hours and except when dettol was used the milk was discoloured for a further twenty-four to forty-eight hours after which it could be used. In the case of dry cows the solution was left in the udder for twenty-four hours and then removed. It was never possible after infusion to recover all the fluid injected. Since our aim was to evolve a practical and economical form of treatment, udders were infused once only. The trial of flaviquin was discontinued because of the severe mammary reaction following its use.

Where sulphanilamide was used it was administered orally, suspended in water, and the best results bacteriologically were obtained by giving an initial dose of 1 g. per 5 lb. body-weight followed by one-sixth of this dose every eight hours for five to seven days. The use of these quantities of sulphanilamide was often accompanied by diarrhoea, dullness, inappetence and reduced milk yield but these symptoms rapidly subsided when dosage ceased. Marked improvement in the nature of mammary secretion in many clinical cases was a feature of this line of treatment. (Such clinical improvement can be obtained with much smaller doses of sulphanilamide).

It was found that the effectiveness of sulphanilamide varied considerably from one herd to another. More recently by the use of the ditch plate technique it has been possible to show that *Str. agalactix* from different herds possess a wide variation in their susceptibility to sulphanilamide.

A large number of cows were treated with *a*-ethyl sulphonamide which is highly soluble in water and diaminodiphenylsulphone which is insoluble. The percentage of bacteriological cures did not exceed those obtained with sulphanilamide but the diaminodiphenylsulphone which was administered in doses of 90 g. did not produce the untoward symptoms which were sometimes associated with sulphanilamide.

In a large number of cows in which the oral administration of sulphanilamide caused no permanent reduction in the streptococcal count, it was observed that there was a very marked decrease in the number of organisms present at about eight hours, after the large initial dose. It was therefore thought advisable to try the effect of such a single large dose followed, eight hours later, at the critical period, by udder infusion. Both acriflavine (1:10,000) and sulphanilamide (0.8%—a concentrated solution) were employed (see table).

| Treated with | Administered | No. of cows | Average No. of quarters infected | Cows cured bacteriologically | Percentage |
|------------------------------------|-----------------------------------|-------------|----------------------------------|------------------------------|------------|
| Acriflavine | ... via teat canal ... | 50 | 1.8 | 20 | 40.0 |
| Eufflavine | ... via teat canal ... | 40 | 1.8 | 18 | 45.0 |
| Detol | ... via teat canal ... | 19 | 2.6 | 5 | 26.3 |
| Sulphanilamide | ... per os ... | 119 | 2.0 | 41 | 34.5 |
| Sulphanilamide | ... per os and via teat canal ... | 42 | 1.5 | 13 | 31.0 |
| Sulphanilamide and acriflavine ... | ... per os and via teat canal | 43 | 1.4 | 22 | 51.2 |
| Controls | | 68 | 2.1 | 4 | 5.9 |

At the present time a controlled experiment is being undertaken to determine the value of vaccines in the prevention and treatment of *Str. agalactix* mastitis.

Attention has also been given to "summer mastitis" (*Corynebacterium pyogenes*), staphylococcal and Group C streptococcal mastitis. The first two have been treated by serum and toxoid respectively. The use of these products appears to save the cow but not the affected quarter. Cases of Group C streptococcal mastitis are rapidly cured both bacteriologically and clinically by the use of sulphanilamide, whereas formerly the affected quarter was always lost. Cases of mastitis due to *Str. dysgalactix* would also seem to benefit by the oral administration of sulphanilamide.

Dr. Robert Cruickshank thought the factors predisposing to bovine mastitis and lactation mastitis in the human might be similar although the predominating causative organisms were different. In the examination of breast-milk from puerperal women he had found that *Staphylococcus aureus* was present in numbers up to 100,000 per c.c. or more in over half the cases without being associated with any clinical infection. Infection when it occurred seemed to be correlated with inadequate emptying of the breast. The cracked nipple, a common precursor of mastitis, was a contributory factor, not because it facilitated passage of the infecting organism, but because it caused pain to the mother during suckling with the result that the corresponding breast was not properly emptied; similarly, engorged breasts were sometimes the precursor of mastitis. The infecting organism in the human was probably often derived from the infant's nose as over 90% of infants were nasal carriers of *Staph. aureus*, so that the organism could be aspirated into the milk-ducts by back-suction. The hands of the milker were the most likely vehicle of the infecting organism in bovine mastitis; if so, prevention of spread should not be impossible.

Mr. H. W. Steele-Bodger said that Dr. Stableforth had not mentioned the condition known as "Black Spot", which was very prevalent. In the past most of these cases had resulted in the loss of a quarter but since Mr. Hignett had informed him that it was frequently due to *Str. agalactix* they had used sulphonamide therapy with great success. Dr. Stableforth had mentioned the value of filling the teats of dry cows with an antiseptic emulsion and he would like to emphasize the success of this preventive measure. He also confirmed Mr. Hignett's evidence concerning the variation in results which were obtained from the use of sulphonamide on different farms.

Dr. H. J. Parish: Mr. Hignett's communication has suggested to me once again the necessity for state aid if the control of diseases of cattle is to be really effective. It is useless to expect the farmer to find the money for expensive prophylaxis and treatment. Staphylococcal mastitis has been influenced to some extent by toxoid, but the results

are not uniformly successful, for the immunity produced is antitoxic and not anti-bacterial. In laboratory animals, injections of staphylococcus toxoid or of antitoxic serum may save the lives of rabbits or mice, but will not entirely prevent abscess formation.

Mr. John Francis: It is difficult to isolate *Str. agalactiæ* from any site in the animal body other than the udder. On the other hand *Str. dysgalactiæ*, *Str. uberis*, group C hæmolytic streptococci, staphylococci and *C. pyogenes* can all be fairly frequently isolated from the nasopharynx and from the vagina. It thus appears that there is some essential difference in host relationship between *Str. agalactiæ* and the other organisms which produce mastitis.

It is now recognized that light infection with *Str. agalactiæ* may be transient and it appears that if sufficiently stringent cultural methods were applied frequently enough that almost every cow would be found to harbour infection at some time or other. For the purposes of control it would therefore seem necessary to define what is to be accepted as an infected cow.

Mr. D. L. Hughes said that the control of streptococcal mastitis based on the old concept, that *Str. agalactiæ* was a strict parasite and the infected udder the only reservoir, would break down.

There were a number of facts which needed emphasizing, including the widespread distribution of the organism, the relative frequency with which it could be recovered from the external surface of the udder of apparently uninfected cows and the ease with which it could be recovered from the hands of milkers between milking. Confusing results had been obtained from regular testing of the milk of cows in herds under good management and the more frequent the tests and the more drastic the technique, the higher the number of cows found to be infected. The irregularity of the excretion of the organisms in the milk in those cows without clinical symptoms was also important.

Was there any justification for elaborate schemes of control and eradication which had so many technical pitfalls?

Mr. Hignett had mentioned the testing of the susceptibility of strains of *Str. agalactiæ* to sulphanilamide by the blood agar ditch technique, in parallel with the clinical treatment of the animal with the same drug. Doubtless many of the disappointing results obtained with this and related compounds could be explained by the resistance of the infecting strain to the action of these drugs and much time, money and labour could be saved by such a preliminary, simple *in vitro* test with the drugs in question, before treatment of the animal was undertaken.

Finally, he would like to see much more extensive research work on treatment with numbers of old and new chemotherapeutic agents. It was likely that with all the drugs we already knew of and many yet to be tested, most cases would yield to one form of treatment or another.

Research on infection in the animal as a calf, although it would be of a protracted and rather expensive nature might yield results of some importance.

Dr. A. D. McEwen said that he and J. D. Paterson had treated twenty to thirty cases of mastitis by the infusion of sulphonamide E.O.S. 2.5 and 5% solutions had been used for cows in milk and 5 and 10% solutions for cows whose udders were dry or in the process of being dried off. The results were encouraging, a marked clinical improvement rapidly being obtained. The treated quarters might or might not be sterilized but if the clinical improvement was maintained, the persistence of a *Str. agalactiæ* infection might be of little significance, as a high percentage of clinically healthy cattle excreted *Str. agalactiæ* in their milk.

He agreed with Mr. Hignett that 4:4'-diaminodiphenylsulphone when given by the mouth was better than sulphanilamide. Therapeutic doses of the former product appeared to be completely non-toxic for cattle. They had treated acute cases of *Str. agalactiæ* mastitis by giving 4:4'-diaminodiphenylsulphone by the mouth for three to four days, and when the acute symptoms had subsided, infusing a solution of sulphonamide E.O.S. into the still clinically infected quarters.

They had tried the effect of infusing 4:4'-diaminodiphenylsulphone into the udders of normal cows but up to the present they had been unable to obtain a suspension or a solution that was sufficiently non-irritant to warrant this method of treatment in cases of mastitis.

Mr. S. J. Edwards said that results achieved in the control of streptococcal mastitis were directly proportional to the efforts made by the farmer and his staff. Where bacteriological testing of milk samples was regularly carried out and proper hygienic precautions adopted the incidence of infection was reduced. In order to achieve this state complete segregation of infected animals, together with the practice of washing the udders of cows with water containing antiseptic and effective sterilization of milkers' hands between milking were necessary.

With regard to the chemotherapy of the disease, recent work carried out by Dubos and by Dubos and Little, on the use of gramicidin appeared to offer a promising field. This substance had been shown to possess highly bactericidal action on streptococci and *in vitro* it could be demonstrated at a dilution 1:1,000,000. The injection of gramicidin-oil suspension in the cow's udder appeared to cause much less irritation than the injection of substances in large amounts of aqueous solution such as had been tried hitherto.

Dr. J. T. Edwards (Pirbright) said he still felt that the ætiology of the common forms of bovine mastitis could not be completely explained in terms of the common type of streptococcus found in the milk drawn from affected udders. Points against its acceptance as the sole cause were: firstly, the difficulty of setting up the disease artificially in a normal udder unless certain drastic steps were taken to introduce the streptococci into the udder cistern; and, secondly, especially when enrichment methods were employed to detect very small numbers of streptococci in the milk, the frequent finding, as had recently been exposed particularly by the results of the Australian workers (Munch-Peterson *et al.*, 1940), of streptococci in the secretions of udders that were not visibly diseased, their transitory appearance in small numbers in the milk of some cows, and their appearance even in the udder secretions of young heifers.

Although difficulty had been experienced in transmitting the disease artificially with cultures of the streptococcus, this did not necessarily exclude its playing the role of the most important pathogen in the series of steps leading to the occurrence of natural bovine mastitis. It was not unlikely that although the common streptococcus of bovine mastitis had a distribution that was almost ubiquitous in some localities, it might be raised in virulence, given certain circumstances which favoured its spread and multiplication, from that of a harmless commensal to that of a true pathogen.

Not all diseases of animals in which streptococci could be cultivated in pure culture from the lesions were now universally accepted as caused primarily by these organisms. Both in strangles and the common equine pneumonia, especially the latter disease, there was some experimental evidence on record, from work done in Germany before the last war, pointing to a virus as the primary causal agent. There was some American work, pointed out by Dr. Stableforth, reporting the presence of a virus in cases of "non-specific mastitis" in cattle (Broadhurst *et al.*, 1939), but the work needs to be confirmed.

Recently, much suggestive information had been forthcoming from studies upon the condition known as "chronic mastitis" in women. In this condition, there was usually no bacterial infection. The subject had been discussed editorially in the *Lancet* (1940 (ii), 423), and he was surprised that day to find Dr. Cruikshank state that in "lactation mastitis" in women, a condition which appeared to follow injuries to the teats and mammary gland, staphylococci were found in the lesions. The "chronic mastitis" on the other hand is a condition which appears to be quite different and commences about seven to ten days before the menstrual flow when the œstrogen content of the blood is at a maximum and there is strong evidence that the condition is due to an excess of œstrogens, and can be alleviated by appropriate hormone therapy. In the modern high-yielding dairy cow it was to be anticipated that disturbances of a similar order were not unlikely to arise, caused by irregularity in the ebb and flow of œstrogenic and mammo-genic hormones during the evolution and involution of the mammary tissues, and subsequently, it could be argued, in localities where the common streptococci of bovine mastitis were ubiquitous the "soil" would be provided for its ready implantation. There was now a good deal of American evidence on record showing that a "non-specific mastitis" does exist in dairy cows, in which no streptococcal or other bacterial infection has gained a foothold, and the hypothesis has already been advanced by Peterson and Hastings (1939) that the common bovine mastitis is the product of two factors, namely, a lowering of resistance due to "non-specific mastitis" followed by streptococcal infection (Francis, 1941).

Some suggestive work had been published a few years ago by German workers (Stang

et al., 1937; Witt, 1936), alleging that the incidence of mastitis ran parallel with the degree of feeding of dairy cattle with imported concentrates such as oil cakes, &c.—so much so that the workers recommended the feeding of cows on pasture and with home-grown produce only, suggesting that the high protein content or other deleterious ingredient of the imported concentrates produces a preliminary irritation of the udder tissue.

The success in specific therapy need not however be adduced as evidence that the streptococci were alone responsible for the disease.

It was not unlikely that in selective breeding—a process which, fundamentally, it now seemed, is one which contrives to modify in a given direction the hormone balances of the body—it might eventually be found more profitable to evolve a type of cow which was perhaps of lower milk-yielding capacity but not so prone to irritative disturbances in the very labile tissue that was that of the mammary gland in the course of its physiological evolution and involution.

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Dr. A. T. R. Mattick (National Institute for Research in Dairying) said that it would be foolish to deny the possibility of a virus being involved in mastitis, but recounted his experience in herds which he believed to be virtually free from mastitis (*Str. agalactiæ*). Cows had been removed from herds if the milk was found to yield, from the quantity examined, even one streptococcus and it was now difficult by a stringent technique to show that infection, however light, existed. The position was that injuries to the udder, although as frequent as formerly, did not now terminate in mastitis. In infected herds it was common knowledge that injuries were as a rule followed by mastitis. It was therefore perhaps a fair inference to say, in respect of contagious streptococcal mastitis, "No *Str. agalactiæ*—no mastitis".

Mr. J. R. Barker said that the discussion had brought to the front the relative position of the parasitism of the invading micro-organisms, the resistance of the dairy cow and certain other unknown factors including the possibility of a virus. Investigations should be made on the bacterial flora of the udders of beef cattle which suckle their own calves.

So far he had not found the *Str. agalactiæ* in samples from this class of animal but *Corynebacterium pyogenes* and a staphylococcus were frequently encountered in the same sample taken from an affected quarter.

Dr. Stableforth (in reply) said that the value of formalin might be assessed by submitting samples for laboratory examination before and after treatment and correlating the results with the clinical findings. They would be pleased to co-operate in any work of this kind. In regard to human infections with group B strains he had made a serological examination of some 18 strains sent him by Dr. R. Hare some years ago and had found that none belonged to any of the serological types commonly found in bovines in Great Britain. *Str. agalactiæ* had not been recovered from the noses of adult bovines and there was no evidence that it occurred in calves, all the evidence still suggested that the main source of infection of the udder was via the teat. The reminder that he had made no mention of so-called "black spot" on the teats was timely; further study of the condition might well be profitable. Whilst *Str. agalactiæ* would live outside the body for a considerable time, there was no evidence that it could multiply there. Various solutions had been tried as vehicles for the bactericidal agent for udder injections and there was some disagreement as to their relative value. They had had better results with distilled water than with vehicles such as lactose solution, which had an osmotic pressure more like that of milk.

Dr. V. D. Allison (in reply) said he had hoped to hear from the veterinary profession some data regarding the incidence and importance of mastitis caused by *Str. pyogenes* (group A), the chief organism which was a cause of bovine mastitis and was also directly inimical to man. From the lack of reference to it in the discussion he gathered that it was rather uncommonly met with and not of importance as a disease-producing agent in cattle.

[April 15, 1942]

Brucellosis and Sterility

Mr. George N. Gould: The control of bovine brucellosis presents many difficulties which are accentuated in war time by the need for the greatest possible production of milk and the restriction of pasturage brought about by the "ploughing up" policy. The ideal solution is agreed by all: the eradication of the disease by early detection of infected animals and the removal—preferably by slaughter—from the herd. As a practical policy in this country I believe it to be impossible of attainment. With this aim in view the United States Bureau of Animal Industry started a scheme for the eradication by agglutination testing of individual herds followed by area testing, and slaughter of reactors—the owners being indemnified—as part of the political policy to reduce the number of cattle in the depressed dairying industry of the U.S.A. in 1934.

Mohler *et al.* (1941) records that in the seventh year of the scheme the cost to America was nearly 7,500,000 dollars and that there were then 61,654 state accredited herds containing 1,114,191 cattle; between 1934 and 1938 1,596,554 cattle were slaughtered or eliminated as reactors. Once eradication had been effected the Americans found, as individual owners and veterinary surgeons had in this country, that the maintenance of the herd free from infection was extremely hazardous, and necessitated the most rigid control of movement, transport and purchase which could not be effected other than by area and actual state control.

In spite of the success claimed the department decided to approve a plan to incorporate the vaccination of calves as an adjunct to eradication. With this end in view large-scale field experiments have been carried out using strain 19, an attenuated vaccine produced by Buck in 1925 which had been found to give a serviceable immunity when injected into cows and heifers but, unlike fully virulent cultures, did not become residual in the udder, did not establish infection in the herd and did not become virulent by passage.

Mohler *et al.* (1941) reports the field trials carried out between 1936-1940 in which 17,000 calves were vaccinated at the age of 5-7 months many of which had subsequently passed up to four pregnancies, 96.2% of parturitions were normal, 3.8% ended in abortion, 82.9% of those which calved normally were negative to a post parturition test, 5.1% positive, and 12% suspicious. Of the 3.8% which aborted 58.7% were negative to post-parturition test, 31.9% positive and 9.3% doubtful. On the basis of the agglutination test only 1.6% of abortions could be attributed to brucellosis. The incidence of natural infection in these herds as revealed by the test at the commencement of the trial was 5,531 (29.2%) and of these reactors 24.1% were still in the herds in July 1940, thus exposing the vaccinated animals to severe natural infection.

In England Dr. McEwen has produced a vaccine Wye No. 2, which, while it gives a highly serviceable immunity, does not produce a reaction to the agglutination test. This vaccine has been utilized under field conditions and in my experience the results are satisfactory. The vaccine is injected into empty cows and heifers and immunity is reinforced annually.

In this country the average incidence of the disease is, without question, much higher than that met in the U.S.A.

In the near future a voluntary scheme is to be introduced, sponsored by the Ministry of Agriculture, in which vaccines of standardized virulence issued by the Ministry will be utilized to effect some control of this disease. The vaccine will be injected into non-pregnant animals of all ages and one type will be suitable for calfhood vaccination.

It is realized that prevention of infection is more important than prevention of abortion, which is only a symptom of the specific endometritis produced by the disease. It is quite clear that vaccines have no curative effect. In early times fully virulent vaccines as recommended by the British Board of Agriculture in 1916 were used, often with a considerable degree of success, to reduce abortion in infected herds. It has been shown that in many instances, such a procedure is followed by localization of the vaccinal organism in the udder and that inoculated animals were thus made chronic carriers of the disease and secreted the organism in the milk. Fully virulent vaccines have, from time to time, being vigorously condemned as being responsible for the introduction, maintenance and spread of the disease, in all countries.

A disquieting feature of official omission in this country is the failure to control veterinary biological products. This applies particularly to vaccines for contagious abortion.

I am convinced that vaccination with an approved, attenuated vaccine of standardized virulence, is the only practical policy that can be applied in this country for the control of this disease, combined with suitable educational propaganda to obtain those methods of improved herd management necessary to minimize the frequency and danger of exposure to infection.

The incidence of brucellosis of man has been stated to be on the increase in recent years and has been estimated at 11 per 1,000,000 per annum. This figure is probably much lower than the true incidence owing to undiagnosed cases and the probable large number of ambulatory or mild cases.

The alleged increase may be accounted for by more frequent diagnosis than in the past but a more difficult point to explain is the low incidence claimed when it must be admitted that *Brucella abortus* is very commonly present in milk.

The importance of *Br. abortus* is not only its possible transmission to man but the tremendous loss of milk caused by bovine infection estimated in the report of the Survey Committee of the N.V.M.A. on Diseases of Farm Livestock (1940) at 40,000,000 gallons per annum. Further factors are the loss of calves and the large incidence of temporary infertility and sterility which is a sequel of infection—whether the animal aborts or not.

The loss of calves is of very great moment for it is essential that our dairy herds be increased to maintain milk production, to maintain the fertility of the soil, to provide adequate replacements for the dairy herds in the future and to be ready for the restocking of the Continent when the war ends. This can best be accomplished by a vigorous policy of disease control with special attention to breeding efficiency and milking capacity.

The loss of milk due to sterility has been estimated at over 100,000,000 gallons per annum.

It is accepted that endometritis of varying degrees is responsible for at least 80% of infertility and sterility in dairy cows, and that about 50% of cases are the sequel of *Brucella* infection. Cervicitis and vaginitis are common complications. The majority of these cases are first degree endometritis and respond readily to a single intra-uterine injection with an appropriate preparation such as aqueous solution of iodine 1:1,000 to 1:500. More serious cases may require repeated treatment, but, except where gross lesions are present as in pyometra or salpingitis, 70 to 80% recoveries can be obtained.

In the last few years there has been a gradual increase in the incidence of *Trichomonas fetus* infection and *Str. pyogenes* infection of the uterus. Both are venereal diseases transmitted by the bull, and are responsible, in individual herds for serious interference with breeding efficiency, heavy loss of milk production and loss of calves. Both conditions respond to efficient treatment by intra-uterine injection and control measures of breeding hygiene.

The treatment of pyometra, both specific and non-specific, has been greatly expedited in those cases where it is not possible to enucleate the corpus luteum, by the use of stilboestrol dipropionate—the synthetic oestrogenic product—following the suggestion of Brownlee and Montgomerie (1941).

Physiological derangement accounts for about 10% of all causes of sterility in average herds; the incidence due to this cause being higher in certain intensively managed farms.

Good herd management and full co-operation of the farmer are important factors in the control of sterility. Records of (1) dates of oestrus; (2) dates of service; (3) dates of parturition; and (4) service records of bulls, are of great assistance in the diagnosis of pregnancy, particularly in the early stages at seven to nine weeks, and in facilitating diagnosis of the cause of infertility where it exists. The service record of the bull is particularly useful in indicating loss of fertility at the earliest possible moment. These records enable cows showing abnormality in the length of the oestral cycle to be withheld from service. Cows showing abnormal vaginal discharge, whether at oestrus or between oestral periods, should be similarly treated. With either of these manifestations it is practically certain that the cow will not conceive, and repeated service often appears to accentuate the abnormality.

Dietetic errors likely to influence sterility can be remedied by the provision of mineral or other supplements. "Silent heat" can usually be overcome by ovarian manipulation. Anterior pituitary hormone preparations and stilboestrol have their respective uses in appropriate cases of physiological sterility.

To control infertility and sterility a system is required to facilitate regular periodic veterinary examination of cows and heifers to permit pregnancy diagnosis and prompt treatment when necessary; the infertile period would thus be made as short as possible, avoiding losses due to long dry periods, maintenance while the animal is unproductive and reduced annual milk yields.

In my experience 80% of affected cows are curable provided early and appropriate treatment is given.

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Dr. A. S. Parkes : *Biological aspects of sterility*.—Fertility in mammals may be taken to mean the capacity to give birth to living young. Conversely, sterility may be taken to indicate inability to do so. Fertility in any vertebrate, and especially in mammals, is the result of a long chain of physiological processes. Breakdown at any link results in failure to produce living young. Three main phases are involved: (a) The production of functional gametes, that is to say ova and spermatozoa; (b) fertilization; and (c) successful gestation of the embryo. Sterility is often classified according to the phase at which failure occurs: Primary sterility—failure to produce functional gametes; secondary sterility—failure to effect fertilization; tertiary sterility—failure to effect gestation.

The failure to produce functional gametes is associated with some abnormality of the testis or ovary, and is usually due to some genetic, anatomical, endocrine or metabolic cause. So far as farm animals are concerned, the condition is more often met with in the female than in the male, especially in those species in which the female has an anæstrous period during which ovarian activity ceases. From our present point of view, the endocrine aspect of gametogenesis is of some interest since it now seems to have been conclusively shown on experimental animals that gonadotrophic activity on the part of the anterior pituitary body is required for normal functioning of the gonad and that the administration of gonadotrophic preparations can produce ovulation or spermatogenesis in a gonad which would otherwise be quiescent.

Failure to effect fertilization when normal ova and spermatozoa are produced is usually to be found in some congenital or acquired abnormality of the accessory reproductive organs, notably of the reproductive tract. In farm animals this condition is more often found in the female than in the male, and may be associated with blockage of the reproductive tract at some point, or with a physiological condition of the mucosa of the tract or of its secretions deleterious to ova or sperm.

Many causes of failure to effect gestation are known. There may be failure to implant the fertilized egg; death of the embryo or foetus, or failure of placental function; at the last moment the birth mechanism may fail. Many of these disturbances are non-endocrine in origin, but I propose to deal only with the endocrine aspects.

The act of ovulation is preceded by the follicular phase of the ovarian cycle, during which a graafian follicle grows to maturity and the reproductive tract is stimulated by oestrogenic hormones produced by the ovary. After ovulation, the corpus luteum grows from the shell of the ruptured follicle and secretes a specific hormone, progesterone, which prepares the uterus for the reception of the fertilized ovum. This preparation always includes the induction of a physiological sensitivity which enables the uterus to produce decidual tissue in response to the presence of the fertilized egg. In many species it is also characterized by extensive morphological proliferation of the endometrium. In every species examined it has been shown that the successful implantation and development of the early embryo is entirely dependent on the presence in the ovary of one or more corpora lutea, and, therefore, failure to develop a functional corpus luteum after ovulation causes sterility. The development of the corpus luteum is almost certainly controlled by the luteinizing hormone of the anterior pituitary body, and deficient activity of the pituitary body thus leads to non-development of the corpus luteum after ovulation. This sequence of events is easy to produce in experimental animals, but otherwise the

pituitary body rarely seems able to initiate the changes leading to ovulation without being able to support the subsequent growth of the corpus luteum.

Although the corpus luteum is necessary for the very early stages of pregnancy the degree to which it is essential during the later stages appears to vary in different species. In mice, rats and rabbits, removal of the ovary, or even of the corpus luteum alone, at any stage of pregnancy, appears to be fatal. In the guinea-pig the corpus luteum can be removed with impunity during the later stages of gestation. In women there are authentic records of pregnancy having been completed after removal of the corpus luteum as early as the end of the second month of pregnancy. In cows most observers are agreed that removal of the corpus luteum during pregnancy results in abortion. Morphological atrophy and therefore, presumably, decline in the physiological function of the corpus luteum at some stage or other of pregnancy, always seems to precede birth of the young.

At this point I must say a further word about the rôle of the pituitary body in pregnancy. Since hypophysectomy leads to atrophy of the ovary, one would expect hypophysectomy to be fatal to pregnancy in much the same degree as ovariectomy. In the early stages this appears to be the case, but in the later stages the situation becomes confused, apparently by the intervention of the placenta, and the pituitary body seems to decrease in importance so far as the maintenance of pregnancy is concerned. Its presence, of course, is essential for the initiation of lactation at birth.

The endocrinology of the placenta is extremely complicated, and the precise rôle of the organ in maintaining the endocrine balance necessary for successful pregnancy is not yet certain. There can, however, be no doubt that endocrine activity by the placenta plays a very important part in the close integration of ovaries, anterior body, and conceptus, and new researches are continually emphasizing this importance. The evidence has been derived from two types of experiment—extraction of active principles from placental tissue, and operative interference during pregnancy.

Several of the sex hormones are found in the placenta of one or other species. Thus oestrogenic substances appear in abundance in the placenta of several species, notably in man and in cattle. The significance of the presence of these substances is obscure, as also is their source of origin. The fact that in several species administration of oestrogenic hormones during pregnancy results in abortion or reabsorption of the embryos puts a teleological difficulty in the way of supposing that oestrogens are elaborated in the placenta. On the other hand, it has been shown that under certain conditions, and in appropriate dosage, oestrogens have a stimulating effect on the corpus luteum, as well as a general growth-promoting effect on the reproductive tract, and may therefore have an essential function during pregnancy. Moreover, oestrogen has been found in the placenta in man after a pregnancy carried through in spite of the removal of the ovaries at an early stage; in this case the placental oestrogen can hardly have been of ovarian origin. Progesterone, which one might expect to be produced in the placenta, especially in species in which the corpus luteum does not seem to be necessary for the whole of gestation, has been found only in very small amounts and only, I think, in the human placenta. Gonadotrophic substances have also been found in the placenta, but only apparently in women and mares.

As regards operative interference, removal of the whole conceptus, foetus and placenta, at any stage in pregnancy, invariably leads to the disappearance of all signs of pregnancy and to the return of the oestrous cycle if the animal is still in the breeding season. Removal of the foetuses alone, however, in experimental animals at any rate, does not necessarily have this effect. Where the placenta are left uninjured gestation proceeds for a certain period with all the usual signs, and the placenta may even be delivered at the normal time. In the mouse, this happens even if the pituitary body is removed with the foetuses. In the cat, ovariectomy causes death of the foetus, but the placenta continue to grow normally for several weeks. In the rat, ovariectomy can be performed without disturbing the pregnancy provided that the foetuses are reduced to one, and all the placenta left in position. This evidence indicates that the maintenance of pregnancy and the initiation of birth are at least partly controlled by placental activity.

It is thus easy to see that damage to the placenta, even if not sufficiently severe to destroy its metabolic functions, might well inhibit its endocrine activity sufficiently to make continued gestation impossible. This contingency must be considered seriously in any discussion of the mechanism whereby infection of the placenta leads to abortion.

Dr. John Hammond: *Sterility in cattle.*—First let us consider sterility in cows from the farmer's point of view in war time. Each year he has to dispose of some cows from his

herd and replace them by others. In war time these replacements are expensive, for the food used to rear them could be much more profitably fed to animals producing milk. Over 20% of the cows leaving herds each year do so because of sterility (Wright, 1933). Much of this sterility is of a temporary nature.

The position of dairy farmers is that while the consumption of milk reaches a peak during the early winter months, it is at this time of year that the production of milk is at its lowest in herds in which an equal number of cows calve each month (Sanders, 1927). In order to have sufficient milk from November to January, therefore, it is necessary to have a high proportion of the cows in the herd calving from September to December. Figures taken from Milk Recording Societies (Hammond, 1927) show that this is far from being the case, especially among the more Northern Counties. Thus in Cumberland only 4.6% of the cows calve in October, whereas 21.2% of the cows calve in March. If the interval between calving and fertile service is calculated the cause for this is seen. In four Milk Recording Societies the average number of days between calving and fertile service was 97 (at 85 days they calve on the same day in the following year). Those cows which calved in September and October, however, went over thirty days beyond the average time before getting in calf again, while at the other end of the scale cows calving in April and May manage to get in calf again about twenty days under the average time. Again, there is evidence that it is more difficult to get autumn calvers in calf again during the winter in the North than in the South; October calvers go forty-eight days over the average time in Cumberland, whereas in Somerset they only go twenty-one days over the average. There is thus, as Roberts (1929) has shown, a tendency for cows to get round to spring calving as they get old; for example, of 1st calvers 17%, of 3rd calvers 35% and of 6th calvers 42% calved in March, April and May. The result of this temporary sterility during the winter months is either that the farmer has to dispose of cows which are not due to calve at the right time of year, and increase the cost of upkeep by replacements, or retain cows in the herd for some time in an unprofitable dry condition. Under present war-time conditions and the high price of down-calving cows in the late autumn the farmer is tending to retain more animals in this unprofitable dry condition during the winter and so reduce the winter milk supply.

To obtain down-calving heifers and cows from August to December it is necessary for the heifers and cows to be got in calf from November to March. It is therefore during these months of the year that the veterinarians should be particularly busy with the early diagnosis of pregnancy and the treatment of those cows which are found not to be pregnant.

Throughout these winter months where, particularly in the northern part of the country, the cows are housed and fed on dry feeds, as distinct from grass or succulents, there is difficulty in getting the cows to breed as compared with the summer months when they are out at grass. In the wild state cattle calve in the spring months, which means service from May to July, and it would appear highly probable with our knowledge of what happens in wild animals with seasonal breeding periods that the fundamental cause of the low breeding activity during the winter months is the relative inactivity of the anterior pituitary gland in producing the follicle-stimulating hormone during this season of the year. This inactivity may express itself in varying degrees in different animals (just as some hens are good winter layers and others not) and so there are various degrees of sterility and modes of expression.

In those animals with the most marked inactivity the ovaries go into anæstrus and no follicles at all are ripened. This condition is particularly liable to occur in young animals, heifers in poor condition or in first calvers which have been milking heavily. It is more frequent too in the North where the cattle are housed than in the South where feeding conditions are better. Bhattacharya, Hammond, Jr., and Day (1941) have shown that injections of 1,500 I.U. of the follicle-stimulating hormone of pregnant mare serum, or of horse pituitary gland, will under these conditions cause follicles to ripen and heat to occur together with conception if served. These injections are the most effective way of dealing with anæstrus cows but I would stress the importance of examining the ovaries to see that the cow really is in an anæstrus condition before the injection is made—for otherwise, if there is a young corpus luteum present in the ovary, there is the danger that cysts may be produced, or if there is a mature corpus luteum in the ovary, that two or more eggs may be shed at the next heat period and so twins, triplets or more produced. In cows treated in this way Hammond, Jr., and Bhattacharya (1942) have obtained up to 30 eggs shed at a time and have produced triplet calves.

In animals with rather more gonadotrophic activity during the winter months there

may be ovulation and the formation of a corpus luteum but no heat, or a very short heat. With heifers therefore it is preferable to run them with the bull rather than to rely on individual matings: if this system is followed it is desirable to make pregnancy diagnosis periodically and remove the heifers known to be pregnant. The animals with "silent heats" (corpus luteum without heat) can be treated by squeezing out the corpus luteum and throwing over the cycle to the follicular phase at shorter intervals than normal. This will usually be sufficient to cause heat. Other animals, especially older cows, appear to ripen their follicles so slowly that large cysts are caused. Day and Hammond, Jr. (1942), have shown that such cysts can be caused experimentally after the ovary has been thrown into the anæstrous condition under the influence of stilbæstrol and before the gonadotrophic hormones have got going again and the normal cycle has been resumed.

In animals with only slightly subnormal gonadotrophic activity during the winter months the normal recurrence of heat and ovulation is not interfered with, but conception is rendered difficult and the animals return frequently to the bull. It would appear probable that in these cases the changes occurring during the cycle are unbalanced in the direction of the luteal phase, for removal of the corpus luteum at an early stage (eighth to twelfth day) of the cycle, which shortens the cycle and throws it over to the follicular phase, is followed by fertility in a high proportion of cases (Hammond, 1939).

The variation in sperm production among bulls probably follows the same course as the variation in fertility in cows (Walton, Edwards and Hammond, 1940). We do not know enough yet about the effects of the hormones on sperm production to be able to treat sterile bulls. The best course we can adopt under the circumstances is to test the semen by means of collection with the artificial vagina (Walton, 1938) and use only those bulls which are fully fertile.

Cases of sterility in cows which occur with much the same incidence at all times of the year I shall not deal with in detail, as these are mainly pathological in character such as pyometra, cervicitis, &c. I would, however, say in conclusion that Day (1942) has found injections of stilbæstrol to be very effective in raising the tone of the uterus and in causing dilatation of the cervix so that discharges are evacuated and the condition clears up.

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Professor G. S. Wilson, referring to the incidence of undulant fever in Great Britain, said that in 1932 he had attempted to estimate the annual number of cases by an indirect method depending on the proportion of sera from patients with pyrexia of unknown origin that contained agglutinins to *Brucella*. Figures collected from eight laboratories in Great Britain and Ireland showed that 3.5% agglutinated *Br. abortus* to a titre of 1:80 or over, and that 21% agglutinated organisms of the typhoid-paratyphoid group to a significant titre. Since there were 2,899 cases of enteric fever reported in England and Wales in 1930, it was concluded that the number of cases of undulant fever in relation to these was in the ratio of 3.5:21, or about 480.

More recently, Dr. Messer of the Northumberland County Laboratory had analysed his figures for the years 1930 to 1941 and had very kindly given the speaker permission to quote them. Of 1,190 sera, 3.4% agglutinated *Br. abortus* to a titre of 1:125 or over and 23.9% agglutinated organisms of the enteric group. These percentages so closely resembled those found in 1932 for the country as a whole that there seemed no reason to alter the previous estimate for the incidence of undulant fever cases in England and Wales, namely about 500 a year.

In collaboration with Dr. Robb-Smith of the Radcliffe Infirmary he had been trying to repeat the findings of certain American authors who had isolated *Brucella* from the lymph nodes of patients suffering from Hodgkin's disease. Material from 15 cases had been examined so far with completely negative results.

In collaboration with Professor Seddon of Oxford he had been examining the frequency of agglutinins to *Brucella abortus* in the sera of patients in orthopaedic hospitals to find

out what proportion of bone and joint lesions were due to infection with this organism. Of 496 sera examined less than 1% contained agglutinins in a titre of 1:80 or higher. Examination of the clinical records was not yet completed, but it seemed justifiable to conclude that *Brucella* infection could not be responsible for more than a very small proportion of bone and joint lesions. Reference was made, however, to a case some years ago of acute osteomyelitis in a child of 2 years of age from whom Dr. Pantou had cultured *Br. abortus*. The child had been given raw Certified milk from a herd in which several cows had recently aborted. *Br. abortus* was isolated from the milk of a number of the cows. The strains all resembled the strain isolated from the bone abscess in the child in growing under ordinary aerobic conditions on primary culture.

Dr. S. J. Folley: I do not find myself in complete agreement with Dr. Hammond's statement that pregnant mares' serum is always the hormone of choice for inducing heat and ovulation in anæstrous cows and heifers. The experiments of Steinach, Stäheli and Grütter showed in 1934 that œstrus and ovulation could be induced in many cases of anæstrus in cows and heifers by an injection of œstradiol monobenzoate. In a limited series of experiments with œstradiol esters we have successfully induced the resumption of ovulatory œstrous cycles in a good proportion of anæstrous bovines. There is no reason why the cheap synthetic œstrogen, diethylstilbœstrol, should not give similar results and, in cases where œstrogen treatment might be expected to cause a resumption of œstrous cycles, the use of synthetic œstrogen would be preferable to pregnant mare serum on the score of low cost.

Contrary to Dr. Hammond's results, in our experiments on the effect of pregnant mare serum on the bovine ovary, we have hitherto found no relation between the phase of the œstrous cycle at which the injection is made and the number of ovulations resulting from the injection.

Lastly, with regard to sterility in bulls, through the kind co-operation of veterinarians, we have had an opportunity of doing experiments on a few bulls giving sperm with low motility (measured by the rate of oxygen consumption) and in three cases found that treatment with chorionic gonadotrophin (pregnancy urine extract) has resulted in an improvement in the motility of the sperm and in the breeding performance of the bull. This would merit further investigation.

Mr. H. W. Steele-Bodger was impressed with the high incidence of *C. pyogenes* infection in certain herds; in these herds there was a tendency to early salpingitis; in one such herd, out of 14 dairy cows examined, 12 were barren, 10 of them had not calved for two years and were nearly dry, and 8 would be permanently sterile. He would like to emphasize the value of stilbœstrol in the treatment of pyometra; this product had also been used with success in the treatment of acute metritis in conjunction with sulphanilamide which was introduced into the uterus mixed with antiseptic soft soap. Stilbœstrol had also been used with success in retention of placenta.

Mr. Hignett had reported that after the removal of the corpus luteum at four months, a cow had carried her calf to term. He understood that stilbœstrol was supposed to have no effect upon the pregnant uterus, but he knew of two instances where an injection of stilbœstrol had been followed by abortion in two or three days.

He had been struck by the number of cows with a record of having calved in September and October, and not having shown signs of œstrus during the whole of the winter; others had calved, had had one œstral period without service and had then gone into a dead anæstrous state; others again had been served, had shown no further signs of visible œstrus and were found to be barren on examination in November, December, January and February. He considered the solution of the problem of overcoming the state of dead anæstrus during the winter months was an urgent one. If 1,500 rat units of pregnant mare serum could be relied upon to have the desired effect then it should be made available for much more general use, and if one could overcome the state of dead anæstrus which one found during the winter months, particularly in heifers, it would materially help in augmenting the milk supply and shorten the periods between calvings in cows.

Mr. N. J. Scorgie said that in regard to the type of infertility in cows and heifers which was manifested by return to œstrus at the normal time despite repeated services, he had tried the method of enucleation of the corpus luteum recommended by Dr. Hammond, but that in his experience this treatment had not been very successful. Enucleation of the corpus luteum at ten to twelve days after œstrus had been practised on a total of 50 cows and heifers; most of the animals had shown œstrus within five days

and were served at this œstrus and at the succeeding one nineteen to twenty-one days later, but only in a small proportion of cases had pregnancy ensued. The animals in question belonged to three different dairy herds, which were tuberculin tested and free from brucellosis. There was no evidence of vaginitis or endometritis in any of the treated animals, and in one of the herds repeated bacteriological examination of swabs taken from the cervix had failed to reveal the presence of any organisms of significant importance.

Referring to sterility and low fertility in bulls, the speaker said that this was of considerable importance at the present time in view of the greatly appreciated value of well-bred dairy bulls. The underlying causes of low fertility in bulls was difficult to determine but American workers had found that under certain conditions cattle do not synthesize all the vitamin C they need for normal functions; one result of this was that the breeding efficiency of bulls is impaired. The workers referred to had reported that the subcutaneous injection of ascorbic acid in appropriate cases resulted in marked improvement in 60 to 75% of the bulls treated. Recently the speaker had had the opportunity of instituting this treatment in a number of selected cases but it was too early yet to assess the results. The type of case being treated was where bulls were failing to get cows in calf and where examination showed the semen to be of poor quality, particularly as judged by viability time of the sperm in storage at 5 to 10° C.: it was obvious that other possible causes of infertility both in the cows and bulls had first to be eliminated before seeking to attribute the infertility to a deficiency of ascorbic acid. For those who were interested, the speaker stated the treatment consists in the bi-weekly subcutaneous injection of 1 to 2 g. of ascorbic acid dissolved in sterile distilled water over a period of five to six weeks.

Mr. L. E. A. Rowson said he had been using stilbœstrol throughout the winter on both anœstrous cows and heifers and the results had been very poor. Most of the animals had shown signs of œstrus with slight swelling of the vulva and discharge, but many had refused to take the bull and had subsided into the anœstrous state again with no heat at the next expected period.

A specific group of heifers had all failed to respond to stilbœstrol and after leaving them a few weeks 1,500 international units of pregnant mare serum had been injected. All but one of the animals had either come into œstrus or ovulated without showing symptoms of it. The one failure was in an animal in extremely poor condition and this may have played a part in preventing œstrus.

Section of Obstetrics and Gynæcology

President—J. M. MUNRO KERR, LL.D., M.D., F.R.C.O.G.

[February 20, 1942]

Observations on Living Eggs of Mammals

By W. J. HAMILTON, D.Sc., M.D., F.R.S.E.

THE methods employed for obtaining living mammalian eggs were discussed and the methods used were described. Some of the photographs were obtained by using a Vickers Projection Microscope which, having the optical system inverted, is particularly suitable. Other photographs were taken with a vertical camera adapted to the microscope. Incidental and transmitted lighting were employed.

Lantern slides were shown of photographs of living unsegmented and segmenting eggs of some ungulates, carnivores and rodents. The vitellus of the egg is surrounded by the zona pellucida which appears to be a homogeneous, transparent capsule (figs. 1, 2 and 4). In the living unfertilized egg the vitellus completely fills the cavity enclosed by the zona pellucida (fig. 1). After fertilization the vitellus undergoes a shrinkage so that a distinct perivitelline space is found (fig. 2). In this space the vitellus and the polar bodies are free to move. In fixed specimens the perivitelline space is obliterated by the contraction of the zona pellucida which becomes opaque (fig. 3).

The appearance of the living vitellus, with special reference to its fatty contents, in the different eggs was described. In the Golden Hamster (a rodent) the vitellus has a yellowish appearance and contains no fatty globules (fig. 2). The vitellus of the ferret (a carnivore) is almost completely filled with fatty globules so that it appears almost opaque. The other eggs shown were graded into intermediate positions between these two extremes. The appearances seen in the living eggs were contrasted with the appearances seen in fixed and stained specimens.

At fertilization the entire sperm, head, middle piece and tail, enters the ovum; the tail, however, is soon no longer visible in the vitellus. On account of the absence of fatty globules in the ovum of the hamster the entire sperm can easily be recognized in the living egg. In other mammalian eggs examined the presence of the head and tail of the sperm can only be recognized after the egg is sectioned and stained.

At the first division two blastomeres are produced which are usually of unequal size. The two blastomeres do not divide synchronously so that a three-cell stage is found; this stage, however, is soon followed by a four-cell stage. The spindles of division at the two-cell stage are arranged at right-angles to each other so that when the division is completed the resulting four cells are arranged in pairs which lie crosswise. The cells of the four-cell stage do not divide at the same time hence stages of 5, 6, 7 and 8 cells are found. At the 8-cell stage there is again asynchronous division until a morula is formed.

In most mammals the egg reaches the uterus at the morula stage and usually on the fourth day after ovulation. Soon after the egg enters the uterus it absorbs fluid and becomes a blastocyst (fig. 5).

A brief description was given of the attachment of the blastocyst of the hamster to the uterine mucosa. This animal is remarkable among eutherian mammals in having a gestation period of sixteen days.

The following cases were discussed:

- (1) Miss Alice Bloomfield, F.R.C.S., reported on a case of primary sarcoma of the ovary.
- (2) Mr. Albert Davis reported three fatal cases of septicæmia due to *Staphylococcus albus* following induction of premature labour with a rubber tube.
- (3) Miss Margaret Basden, F.R.C.S., showed a specimen of a uterus removed on account of a rupture of an old Cæsarean scar.
- (4) Mr. R. Leslie Dodds, F.R.C.S., reported on a case of torsion of a pregnant uterus in which was a fibroid the size of a lemon.

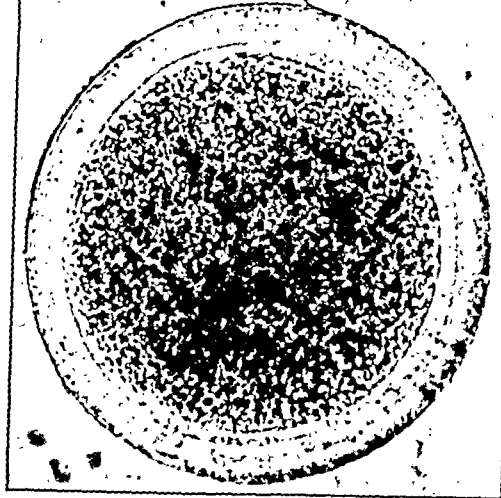


FIG. 1.

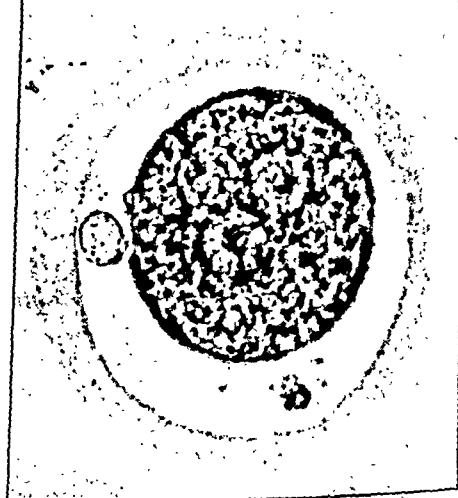


FIG. 2.

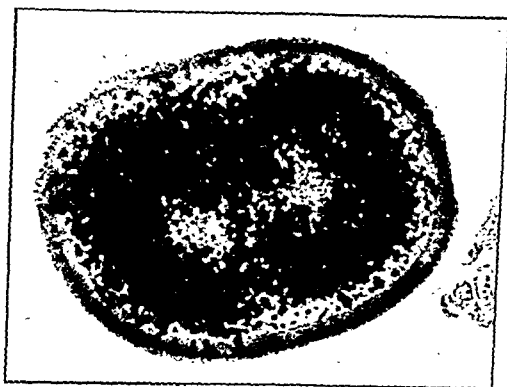


FIG. 3.

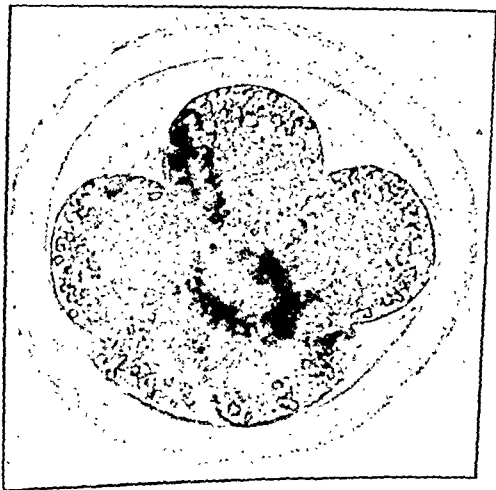


FIG. 4.

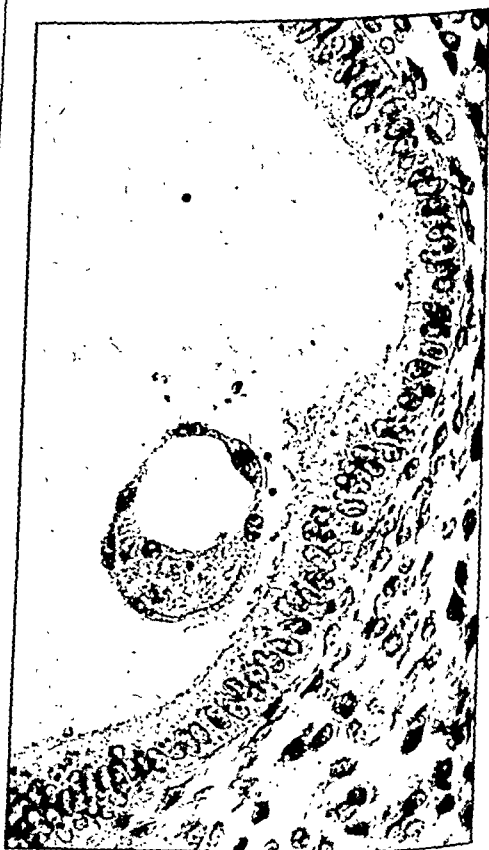


FIG. 5.

FIG. 1.—A photograph of the living unfertilized ovum of the cow. $\times 350$.
 FIG. 2.—A photograph of the living fertilized ovum of the Golden Hamster. A perivitelline space is present and in it the two polar bodies are seen. In the vitellus the pronuclei are just visible. $\times 460$.
 FIG. 3.—A photograph of a 2-cell stage of the ferret stained with osmic acid to show the distribution of the fatty material. $\times 420$.
 FIG. 4.—A photograph of the living ovum of the cow at the 10-cell stage. 6 cells are distinctly visible. The outlines of the others may just be discerned. $\times 350$.
 FIG. 5.—A photograph of a section of the blastocyst of the Golden Hamster lying in a bay of the uterine lumen. $\times 490$.

[May 15, 1942]

DISCUSSION ON MATERNITY SERVICES

Dame Louise McIlroy (*Abstract*): In opening a discussion on maternity services the starting point must be the welfare of the patient and her wishes in the matter. A maternity service scheme must fit in with the general plan of a composite State medical or health service. With the establishment of the Royal College of Obstetricians and Gynaecologists, obstetrics now takes its place on an equal footing with medicine and surgery and is not merely a special branch. It has had close collaboration with the public health services. The introduction of antenatal care has given a much wider scope for the practice of preventive medicine, and teaching has expanded beyond the abnormalities of the bony pelvis and the positions of the fœtus.

The obstetrician now works in collaboration with the orthopædic surgeon, the pædiatrist, the cardiologist and the tuberculosis specialist. The help of the pathologist and the radiologist is frequently required for accurate diagnosis, and much more time is now expended upon the clinical examination of the patient than formerly.

Social services also play a large part in the practice of midwifery. Institutional expenditure in other directions could be reduced if we could reduce or eliminate the blindness due to ophthalmia of the newborn. Mental deficiency due to injuries at birth, could be reduced to some extent and the incidence of tuberculosis could also be lessened, if there were more provision for prolonged residence in sanatoria and facilities for childbirth in these institutions. With more efficient postnatal care the crippling effects of childbearing upon women would also be reduced. Antenatal care would lower the neonatal death-rate due to prematurity, birth injuries and malnutrition.

The economic aspect of maternity practice is of the utmost importance to the State. In my opinion, infant welfare should be under the direction of the pædiatrist after the first month of life. Gynæcology cannot be separated from obstetrics as treatment is required for postnatal complications.

Much of the organization of the maternity services has been haphazard and lacking in co-ordination. This, to some extent, has been inevitable. New ideas and methods had to be tried out by voluntary organizations and proved to be valuable, before public funds could be utilized for anything approaching an experiment.

Legislative reforms, such as the various Midwives' Acts, have brought about great advances in midwifery. Local authorities were given permission to make use of public funds for maternity and child welfare work. It might have been better if, in some cases, these powers had been made compulsory. The Local Government Act of 1929 made a great advance in the establishment of maternity beds in Municipal and County hospitals. The London County Council is an outstanding example of this improvement. It is more satisfactory to have a maternity department in a large general hospital than in isolated units, with the exception of small maternity homes linked up with larger hospitals. In a general hospital auxiliary services are easily obtainable for consultative work, and there is economy of staff and equipment.

In building up a scheme it is well to find out what is essential, what is deficient in existing schemes, and what should be avoided. In some areas under a keen medical officer of health, the arrangements are excellent and the work with the local practitioners is harmonious. In others the standard of efficiency is low. Neonatal mortality on the whole has been very little reduced, and for this the obstetrician must bear the responsibility.

The chief defect in our present maternity services is the limited number of beds in industrial areas. Antenatal and postnatal beds especially are very inadequate. Emergency cases in rural areas may have to be sent long distances because no beds are available and general hospitals may have little provision for maternity cases.

In the future it is probable that domiciliary midwifery will be still further reduced owing to the difficulties of housing and home service. Flats are unsuitable and are noisy, and women should not have their confinements in slum dwellings.

Rest from household cares and good nursing have a marked beneficial effect upon the nerves of women. This is supplied by hospitals and local maternity homes. During the war, however, residents in evacuation areas often are forced to have their confinements in overcrowded homes, as the local maternity homes are booked up for patients coming from cities and towns in more dangerous areas. Patients are sent out of hospital too soon in some cases owing to pressure upon the beds for childbirth. Convalescent homes are also an important part of a scheme for maternity services, for antenatal as well as for postnatal cases.

Antenatal clinics in connexion with maternity hospital staffs are satisfactory, as there

is continuity of supervision throughout pregnancy and childbirth. In country and municipal antenatal clinics, however, the assistant medical officer, whose special qualification is the possession of a Diploma in Public Health, may have had little clinical experience of midwifery. The chances of promotion in such a service, for women especially, are small owing to lack of special training in administrative methods. Local authorities, however, are beginning to recognize this difficulty and to insist upon a Diploma in Midwifery. If complications arise during pregnancy the patient is either sent to hospital or to a local practitioner who may not be very interested in her condition; as she preferred to attend the clinic at the outset of the pregnancy. In cases where a midwife is in attendance at the confinement, a local doctor is called in if any complication arises and, with no personal knowledge of the patient's previous condition, he may criticize the treatment at the clinic. If the patient is seen later at the postnatal clinic, criticism of the methods employed at childbirth may occur. Midwives who do not work with a recognized team of practitioners serving on a midwifery rota find it not always possible to get help in an emergency, and delay may be serious for the patient concerned.

The fault lies in this practice of dual responsibility and absence of continuity of supervision. This may be illustrated by the proverb: "Never swap horses while crossing a stream."

In some areas, especially when at a distance from towns, the local practitioners should be chosen as a team because of their competence to practise midwifery, and consultative clinics and services should be available. Facilities for hospital treatment should be readily obtainable. The patient should not be discharged from the supervision of the doctor and midwife until four weeks after the confinement. This would reduce to some extent the difficulties which are met with in breast feeding.

We owe a great debt of gratitude to voluntary organizations and hospitals in midwifery practice. I am glad that the Minister of Health intends to retain these institutions in a general scheme for medical services. Voluntary contributions will be difficult to obtain after the war, and financial assistance will have to be given by the State if the voluntary hospitals are to retain their individuality. This may mean a loss of administrative control and independence, but the hospitals will gain by being included in the general scheme and given a definite position therein.

This will do away with overlapping and waste of beds in some areas, and will supply accommodation in other areas where hospital resources are inadequate. It would also reduce the long waiting lists for admission and the overcrowded out-patient departments. The voluntary hospitals have done much for teaching by supplying clinical facilities for the medical schools. The healthy spirit of competition does much to make the individual wish to excel. Each special department of the large hospital is sponsored by an honorary head or "chief" who feels himself responsible for its efficiency, and by having a say in the appointment and control of his staff, expects whole-hearted co-operation and loyalty.

Consultants are now paid in hospitals such as those of the London County Council, some being whole- and others part-time. The question is, will the future generation of consultants be willing or able to serve as honorary members of the voluntary hospital staff? The answer is not far to seek. The traditions of the voluntary hospitals must not be lost as they are a bulwark against a completely socialistic scheme of State service. Co-ordination of the hospitals is being achieved to some extent by the establishment of the Emergency Medical Service hospitals and by the Nuffield Provincial Hospitals' Trust. These are on the lines of central councils and hospitals with regionalization of areas. The Council of the King Edward's Hospital Fund for London has done much to improve the buildings and equipment of the voluntary hospitals. Would a whole-time State Medical Service give the same incentive to original research as under the voluntary system? Would promotion depend upon clinical or administrative ability?

In general practice midwifery has declined to a great extent, owing to the efficiency of the midwives' services, the establishment of clinics and hospital beds, and also because of the unsuitability of patients' homes. The decline of the birth-rate is also a factor.

The National Health Insurance does not directly include the treatment of midwifery patients, nor does it give special consultant services and hospital accommodation. The panel doctor is too busy to take up midwifery practice with its inadequate remuneration, and he objects to the risk of criticism from local health authorities if cases should go wrong. Cottage hospitals have a limited number of local practitioners in attendance, and others may be excluded from the treatment of their patients when admitted. Small maternity homes should be established where the family doctor can admit and be responsible for the treatment of his own patients. Consultants and auxiliary services should be available from a larger supervising hospital centre.

In rural areas the family doctor would act in a part-time capacity as he would have

to undertake other branches of medical practice. Women prefer local hospitals where they can be kept in touch with their families and relatives. The family doctor has been the trusted adviser and confidant of his patients as is the priest and lawyer. He is chosen because of the personal element. He knows the home conditions and circumstances as no whole-time practitioner or consultant can know them. In official centres case records are open to the inspection of a staff who are under no Hippocratic oath. Consultation clinics do much to relieve the anxieties of the general practitioner and discussions can take place as to diagnosis and treatment. The family doctor is, moreover, the first line of defence in the case of disease. But the doctor must have adequate training and must keep up to date by regular refresher courses in larger centres.

A team of doctors and midwives working in collaboration with a keen medical officer of health would ensure good results in these areas. Local clinics or centres could be established for interviewing and treating patients, but such centres would have to be instituted by the local health authority, as otherwise there would be difficulties of administration. Payment for work done would be given by the health authority and collected when possible from those patients who could afford to pay or who are specially insured. If any criticism arises it should be considered first by the committee of local practitioners and consultants before being judged by a lay board of management.

In Scotland domiciliary midwifery is more in the hands of the general practitioners than it is in England and Wales, and the midwives work directly under the doctor. The Scottish Board of Health Act (1937) was a scheme for this co-ordination of practitioners, midwives and consultants, and it has been established in a large number of areas. Lanarkshire, with its central maternity hospital at Bellshill, is a notable example of its efficiency. The patient has a choice of doctor from the local team of practitioners. The consultants on the staff of Bellshill hospital must hold the membership or fellowship of the Royal College of Obstetricians and Gynaecologists.

The Russian public health services seem to serve as a basis for many discussions on proposals for a State medical service in this country.

In Russia all medical education is free and all doctors are whole-time practitioners under the U.S.S.R. The whole aim of the service is the prevention of disease. Every citizen has a right to free medical treatment. Some of the expense is recovered by contributory schemes of health insurance. Women in employment get full pay for the eight weeks before and the eight weeks after confinement when off work.

The medical practitioners get study leave and retire on pensions. In the local areas are health centres or polyclinics and these are linked up with larger centres and hospitals. The teaching schools and research departments form the centre of the scheme. Local and central committees or soviets control administration.

A maternity service should give the patient a choice of doctor at the periphery and consulting and specialist services should work up towards the centre. There should be a central advisory committee composed of representatives from the universities, the Royal College of Obstetricians, the British Medical Association, Central Midwives' Board, College of Midwives, hospitals, and other organizations interested in the practice of midwifery. The administration of such a maternity service should be in the hands of those who have a considerable knowledge of the practical side of obstetrics as well as of public health.

Mr. Eardley Holland: The present state of maternity services may be fairly judged by considering the trend of maternal mortality. It is logical to take the maternal mortality rate as a yardstick by which to measure the quality of the service, and it may be assumed that the rate bears a fairly constant ratio to the amount of maternal injury and ill-health.

During the last five years the rate has shown a definite fall, both for the deaths from puerperal sepsis and for those from other causes. The deaths from sepsis have fallen about 60%, and the latter about 40%. Had this gratifying fall not occurred it would have been a grievous disappointment in view of the efforts that all concerned have made during the last twenty years. But the maternal mortality rate could be made to fall considerably more, and could probably be brought down to about half of what it is at present.

I think we are all agreed that obstetrics is far better taught, both to medical students and pupil-midwives, in large maternity hospitals or maternity units of general hospitals of university standard. This is not only because of the largeness and variety of the material but also because those institutions facilitate the training of young specialists. Even now there are not many teaching hospitals in London large enough to train young obstetric specialists.

The list of the respective number of maternity beds at hospitals in association with undergraduate medical schools shows Glasgow at the top, then come Edinburgh, Leeds, Liverpool, Manchester, Belfast, Newcastle and Sheffield; and then, in order of diminishing numbers, come the London medical school hospitals headed by University College. This position must be altered. Young specialists must be trained in large obstetric units, such

as exist at Glasgow and Edinburgh and at some of the English provincial university centres.

What I have said with regard to medical students applies also to the teaching of pupil-midwives. This teaching suffers a great deal from the fact that much of it takes place, alike for Part I and Part II, in petty maternity schools.

Since September 1939 I have been at work in a certain county as obstetric consultant and one of my duties is to investigate every maternal death. This county has a very low maternal death-rate—in 1939 only 1.8 per 1,000. One of the generalizations that can be made from a study of the 60 consecutive deaths which I have investigated is that 40% of them occurred from shock and collapse.

From this it follows that institutional midwifery, so long as the institution is in the first class, is safer than domiciliary, for the simple reason that facilities for blood and plasma transfusion are always at hand. Another point about maternal mortality is that many of the deaths are wrongly certified. Another is that the confidential reports on maternal deaths that are made to the Ministry of Health are extremely valuable and a mine of information, provided they are closely scrutinized and supplemented by personal interviews and by examination of records by an expert obstetrician. Another generalization is that far more deaths occur from lack of judgment or skill during labour than from imperfect antenatal care.

In the emergency maternity service of this county (run on behalf of the Ministry of Health for evacuee expectant mothers) there are a number of maternity homes and two maternity hospitals (converted mansions). All have a staff of first-class midwives, and each home has a general practitioner (selected as carefully as possible) to take medical aid calls. The maternity hospitals, each with R.M.O., and at one of which I myself live, support the whole system; abnormal cases are taken into them, and emergencies come in by ambulances.

The results have been successful. In the two years 1940-41 there were 5,500 births in these homes and hospitals, and six mothers died, either in the homes and hospitals or in other institutions to which they had been transferred. After deducting one "associated death" (a case of severe puerperal insanity, with death from septic parotitis and myocardial failure, in a mental hospital) five direct puerperal deaths remain, giving a mortality rate of 0.9 per 1,000. Among the 5,500 births there were 390 cases of hypertensive toxæmia of pregnancy (including 8 eclampsias), 60 Cæsarean sections, 206 postpartum hæmorrhages, 27 placenta prævias, 367 forceps deliveries, 50 manual removals of placenta and so on—a good mixture of abnormal cases. A mortality of 0.9 per 1,000 with clinical material like that in improvised institutions is surely a demonstration of the safety of institutional midwifery. The credit goes primarily to the midwives, and then to the general practitioners who responded to the medical aid calls, and third, perhaps, come the specialized services and leadership at the central hospital.

I have some figures for the London County Council, kindly sent to me by Dr. Allen Daley, which are equally remarkable. The total number of confinements in 1938 in London was 66,000, of which 45,000 were institutional, and no fewer than 21,000 of these were in L.C.C. hospitals. The mortality for the booked cases was only 0.9 per 1,000.

In planning for the future, it may be said that there is already in existence a national maternity service, namely, that administered by local authorities. But admittedly it has inherent defects. It is in the hands of 62 county councils, 83 borough councils, and 283 district councils, all of them separate and independent maternity and child welfare authorities, each carrying its own maternity hospitals and homes, its own antenatal clinics, and its own salaried services of midwives. Whitehall has to deal with all these independent obstetric units. On the whole this form of service has worked well. But the system lacks form and unity. The administrative units are far too numerous, and, with few exceptions, far too small. There is no co-operation, so far as I know, between the independent units; and even within the individual units there is usually imperfect co-operation between the various elements—e.g. the voluntary hospitals, the local authority hospitals, the medical officers of the local authority, the general practitioners (this criticism does not apply equally in all units)—and the standard of the work done in some of the small maternity institutions is low.

It is difficult to see how there can be any leadership or inspiration in the present maternity service; there is no provision for such, either at the centre or the periphery. The standards of work attained by the different local authorities naturally show great differences. The most lamentable thing that has happened since the last war has been the separation that has come about between the Officials in Whitehall and the practising Obstetricians and teachers.

I am in favour of a National Maternity Board or Council responsible to the Minister, with the local authorities as its agents (just as the local authorities are now the agents of

the Ministry for the Emergency Medical Services). Upon this Board all interests would be represented. Nearly all are agreed on the advantages of regionalization, with units of sufficient size to allow the full development of obstetric services. The central component would be the regional obstetric centre, with the central hospital. Central hospitals would be of university standard, with, at the lowest, 100 obstetric beds and a suitable number of gynaecological beds. If they could be associated with, though architecturally separate from, a general hospital, so much the better. At the head of each there should be an Obstetric specialist of high standing, with his assistants. Some would be teaching schools for specialists, practitioners, students or midwives.

Such an obstetric standard would provide leadership and inspiration, by example and precept, to the peripheral components, by which I mean the general practitioners, the midwives, and the smaller hospitals and maternity homes. Over these the central hospital chief would exercise a general benevolent clinical supervision, both as regards institutional and domiciliary work. The central hospital would be a great inspiration to the obstetrically-minded general practitioner. The creation of an Obstetric Centre such as that in each region would be, I think, the most important factor in the whole of the future obstetric service of the country.

Finally, I think that midwives should be given more responsibility and better status. The service could not possibly be run without the obstetrically trained and interested general practitioners. Some practitioners are becoming more and more interested in obstetrics, and others less and less. That is a movement in the right direction; for it will lead to a process of selection amongst them as regards midwifery practice.

Sir Alexander MacGregor (Medical Officer of Health, Glasgow) agreed with Mr. Eardley Holland that the rules of the Central Midwives' Board might be relaxed so as to give the trained midwife more latitude in her duties. In Scotland the law had gone a good deal further than in England, and had set up a domiciliary medical service which was now operated by a large number of local authorities. The Glasgow scheme had not yet come into being owing to the war, but it was being clearly shown that the tendency was all towards greatly increased accommodation for lying-in patients in institutions. This movement, which had been growing for many years, had been much accelerated since the war began; it was one which women themselves desired, and which was eminently advantageous both on public health and social grounds. For these reasons, therefore, he thought that a maternity service should include all the facilities available to women and not lay the emphasis on the purely domestic side of midwifery, as the Maternity Services Act had done. Provision of additional indoor accommodation, both for antenatal treatment and for lying-in, should be one of the first aims of post-war hospital policy.

The proportion of confinements which took place in institutions in Glasgow was 45%—much lower than the 60% figure for London. Another feature was the popularity of the antenatal centre, to which more and more women were coming each year. His view was that the maternity hospital (preferably the maternity unit of the general hospital) with its antenatal centre, along with its associated antenatal and postnatal clinics in the area of the city round about it, should be the pivot of a maternity scheme, and that the antenatal clinics and the work in the hospital should be carried out by a group of trained obstetricians, who should be available on call to the midwife of the district. It was intended to base the Glasgow scheme on this principle. Only 47 out of over 600 medical practitioners notified over 20 births per annum in 1938 as having occurred in their private practice.

If there was to be any future legislation in the sense of introducing a complete, well-knit, and harmonized maternity service, great latitude should be allowed to different areas. What Mr. Eardley Holland had pictured as an ideal scheme for a county area—which he did not oppose for a moment—might not serve for large industrial cities. Of course, this proposal for a whole-time service had given rise to a great deal of powerful opposition, and had not yet been formally approved. But he did not wish to introduce a service which would perpetuate confinements in small houses, the service should be based to an increasing degree on the maternity hospital and the antenatal clinic functioning together.

Mr. Arnold Walker said that there was one fundamental difference between a maternity hospital and other hospitals; in general, the maternity institution cared for the healthy while other hospitals were concerned only with the treatment and care of the sick. The maternity institution, of course, dealt with many cases that were pathological, and its chief value to the community rested in its ability to treat the pathological efficiently and expeditiously, but that did not affect the argument that a maternity institution had as its primary object the safe conduct of a mother and her baby through a natural but hazardous period of their lives.

This point of view was tending to be lost, chiefly because of the great increase in the maternity departments of general hospitals. Unless the authorities regarded the maternity department as something different from the rest of the hospital, and made it as independent as circumstances allowed, childbirth tended to become just another disease. This unfortunate attitude had a very real effect upon the pupil-midwives who were trained in these maternity departments, and, from such information as was at present available, it appeared that a much lower percentage of pupil-midwives trained in schools attached to general hospitals eventually practised midwifery than was the case with pupils trained in special maternity hospitals.

The principle of the maternity department attached to the big hospital was likely to be extended at the expense of the special maternity hospital. The idea appealed to the tidy mind of the administrator, and a great deal too much stress was laid on the value of the ancillary services which were available.

Turning to the question of the medical staffs of maternity institutions, in general, voluntary institutions were controlled by a number of obstetricians who exercised their control as a committee, while municipal institutions were controlled by one individual. The qualifications of this individual were varied. He might be the medical superintendent of a large general hospital, who might or might not be interested in midwifery; he might be the medical officer of health, who probably knew nothing about practical midwifery; he might be a whole-time assistant medical officer with some experience of midwifery, and, lastly, he might be a practising obstetrician.

Almost all municipal institutions now made use of the best obstetric skill available. Some, like Middlesex County Council, employed whole-time obstetricians, and others employed obstetricians as consultants who were called in when the resident medical officer considered it necessary. In these cases the consultant was concerned only with the particular problem put before him. When the medical officer had had a reasonable amount of experience this system worked fairly well, but it was more than likely that the consultant would be interested only in the particular problem with which he had to cope rather than with the efficient running of the department as a whole.

Control by the obstetrician seemed to be the ideal method, provided conditions were such that he was able to keep in close touch with all that was going on. The link between preventive and clinical medicine was perhaps closer in midwifery than in any other branch, and the role of the obstetrician should primarily be to prevent complications, and secondarily to treat such as he was unable to prevent. These principles had been in operation at the Willesden Maternity Hospital during the past eleven years during which the speaker had been consulting obstetrician. While all administrative matters were dealt with by the M.O.H., complete clinical control was left to the obstetrician. In 9,000 cases, the booked mortality rate was under 1:1,000.

In planning the maternity institutions of the future, the most important single factor was unification of control. There was nothing new in this idea, and it had for long been the practice in Dublin.

There was one point he wished to mention concerning the position of midwives in maternity institutions. Obstetricians were, he thought, in general agreement that the foundation upon which the maternity service now and in the future must be built was a well-trained body of midwives supported by an adequate but limited number of equally well-trained medical practitioners. In domiciliary practice no one questioned the fitness of the midwife to care for the normal case, and everyone rightly trusted her to call in medical aid when an abnormality arose. In the great majority of maternity institutions this principle was accepted, but certain incidents had cropped up recently which showed that in some places experienced midwives were treated as nothing but maternity nurses. The effect of this mental attitude on the part of the medical officers had been disastrous to the morale of the department. Good midwives would not put up with it, and pupils left the school with a false idea of the place of the midwife in the maternity service. For over eleven years he had considered the midwives to be in charge of all normal cases at Willesden and had made them responsible for calling his house surgeon or himself.

Dr. W. Allen Daley (Medical Officer of Health, London County Council) said there were five elements to be considered. The first was the patient. In this democratic country the patient would have the last word, and it was finally the patient, expressing her opinion through the local authorities, who would say whether or not this service was to be primarily a domiciliary or a hospital service. So far as L.C.C. hospitals were concerned the expression of the views of the patient was significant: in 1930 there were 10,000 confinements in L.C.C. hospitals, and in 1938, 21,000. There would probably, however, always be some demand for domiciliary confinements.

The second element was the midwife. She must have adequate training and refresher courses.

The third element was the doctor, who must also have adequate training. It was now much better than it had been, but owing to shortage of beds in the London teaching hospitals the resources of the L.C.C. maternity units were used largely.

The view has been taken that the qualification to be demanded of general practitioners called in by midwives under the rules of the Board should not be simply willingness to attend but that this must be linked with some evidence of competence.

The fourth element was the system itself. The service should function as a whole. The domiciliary service should be linked more closely with the hospital service, and the antenatal service should be a constituent of both. There should be maternity departments in general hospitals rather than *ad hoc* maternity hospitals. Mention had been made of the relative values of part-time and whole-time service in institutional midwifery. The L.C.C. in its maternity units had adopted the system which had just been outlined by Mr. Walker. Experienced part-time obstetricians were in charge, and this had been of great value to the service, but circumstances differed and in some areas a whole-time officer might be preferable. He agreed that the obstetrician should live in or near his hospital.

Reference had been made to one-man administration as against administration by committees. In the L.C.C. service very great benefit had been derived from conferences with all their obstetrical experts to consider each maternal death and questions of general principle.

The last element was administration. He thought this should be in the hands of the major local authorities, as they were the hospital authorities. Schemes were afoot for cementing the partnership between municipal and efficient voluntary agencies, and that was the line on which development would most usefully take place.

Cold water had been thrown upon political control. Having been employed by politically elected municipalities for over thirty years he felt that democratic control in this particular service was a great driving force. He had never had the slightest difficulty in getting any advances in maternity work approved by his committees and he knew that in some areas it was the politically elected councils which had themselves pressed for developments. In this service there was no difficulty in obtaining authority to expend public money.

Dr. Dick Read said that those who worked in the homes had a different point of view from the teachers in hospitals. Obstetrics was the greatest and most far-reaching branch of our science. It had never been below the level of medicine and surgery; production plants were usually more important than repair shops.

Dr. Walker's vision of the individuality of maternity services apart from general hospitals was undoubtedly correct. Mr. Holland had remarked that "the habitual vision of greatness was very unlikely to be seen in obstetrics". Academic obstetrics, perhaps, but the habitual vision of greatness of all healthy-minded women was childbirth. The birth-rate was falling; our profession was not blameless; the public had not been taught; the trend of culture and civilization had been allowed to inhibit the laws of nature. It was largely our business to rectify this absurdity. The reconstruction of communal life demanded a new philosophy. It would not be found in maintenance or repair; it would be found in those spiritual and physical forces which prompt reproduction of the species.

The birth-rate was a national problem of fundamental importance, and a special Ministry of Reproduction, including experienced obstetricians of administrative ability, should be demanded of the Government.

There was good reason to believe that politicians would welcome and assist such a design. The greatest opportunity that any group of scientists in the world had ever been given for the introduction of new standards, economic, domestic and philosophical, was in the hands of the obstetricians of this country to-day if they had the foresight, the unselfishness and the courage to emerge from the carapace of conservative principles.

Dr. Winn Everett said that she wanted to emphasize the necessity of retaining the general practitioner in any scheme for improving the maternity services of the country.

This necessity was apparent for two reasons. The first was the personal relationship between doctor and patient. The second was that the number of doctors required to staff an efficient maternity service would render the exclusion of the general practitioner a practical impossibility.

In the scheme for an improved maternity service, which she wished to outline, a list would be drawn up in each area of doctors wishing to practise obstetrics and willing to attend post-graduate courses in the subject.

There would be central consultants—obstetrical specialists—wholly responsible for the organization of the service in the area under their control. Under these central consultants, there should be local consultants. They should have had at least ten years'

post-graduate experience of obstetrical practice, with some experience of general practice and they should hold a higher degree in obstetrics.

As regards antenatal care and hospitals—maternity cases fell into two groups: (1) private cases, whose own doctor would be responsible for antenatal care; and (2) midwives' cases, who should attend antenatal clinics which should be run by the local consultants. All abnormal cases should be seen by their own doctor in consultation with the local consultant.

Adequate local hospital accommodation should be provided, the adequacy obtaining alike in regard to the number of beds, including private wards, and the equipment. The central consultant should be sufficiently mobile to work at these local hospitals.

The President, in summing up, said that what had transpired chiefly from this discussion was that institutional midwifery was increasing. In the L.C.C. area, before the war, over 60% of cases were attended in institutions. These people went voluntarily, and the institutions, when well run, showed extremely good results, as Mr. Eardley Holland had stated. The maternal mortality rate in some institutions was approaching the *irreducible minimum*. That meant that domiciliary midwifery must become less and less until, in industrial centres, it would entirely disappear. It would never entirely disappear in sparsely populated rural areas.

Here he would stress a most important point—this development might affect the medical curriculum. It might well be asked: Why give *extensive training* to a large body of undergraduates who were not going to practise obstetrics? A similar contention might be raised in respect to surgery.

As regards midwives, if there were to be a very limited domiciliary service in the industrial areas there would be very few midwives practising in those areas—they would be transferred to maternity hospitals. Here he wished to support Dr. Walker in his confidence in allowing the trained midwife to supervise normal cases in institutions. It was most important that such responsibility should be delegated to her.

Further, there should be one head—not several heads—in a maternity hospital. That had been the principle in the Rotunda Hospital, Dublin, for wellnigh two hundred years. Until "Masterships" were established in maternity hospitals the ideal staffing of these hospitals would not be reached and, lastly, it was most important that the "Master" should reside in or close by the hospital.

He wished to remind Dame Louise Mellroy that schemes practically identical with the one she had outlined were drawn up more than ten years ago—one by the late Professor Blair Bell and the other by himself. The outline of both schemes would be found in the *Lancet* for 1931 (ii), 367. In his own view the central directing body must be an *ad hoc* body, which he had termed the "Central Obstetric Board"—advisory committees were of no use, for the advice was generally put into a pigeon-hole and forgotten. He thought a sympathetic consideration would be given by the Ministry in Whitehall and at the Department of Health for Scotland if a sound scheme were brought forward for a "Central Obstetric Board" for each country. If such a body were established peripheral differences would be more easily settled—the Regions would have representatives of all agencies concerned in maternity and infant welfare. The service must be established at the centre, and from the centre the organization must spread outwards.

[March 20, 1942]

CLINICAL MEETING AT THE BRITISH POST-GRADUATE MEDICAL SCHOOL

Cases were shown by Dr. Meave Kenny, Dr. K. M. Harding and Professor James Young. Mr. Green-Armytage showed slides and photographs indicating the pitfalls in salpingography.

Pathological specimens were shown by Dr. A. I. Ross, and Dr. D. Daley and Professor James Young. Dr. Meave Kenny gave a demonstration of the precision stereoscope.

Section of Physical Medicine

President—MAJOR G. D. KERSLEY, R.A.M.C.

[May 16, 1942]

JOINT MEETING WITH THE MEDICAL SOCIETY FOR THE STUDY OF
VENEREAL DISEASES AT THE ROYAL VICTORIA HOSPITAL

DISCUSSION ON GONOCOCCAL ARTHRITIS AND "RHEUMATISM"

Major G. D. Kersley: Rheumatism associated with gonococcal infection has three striking characteristics, the variety of the manifestations, the difference in duration between time of infection and onset of symptoms and its reaction to hyperpyrexia.

What are the criteria for considering a case of rheumatism as gonococcal? Is the man who has rheumatic attacks for years and who gets an acute flare up ten days after a G.C. urethritis to be classed as G.C. "rheumatism"? Similarly what of the man who has had a past gonococcal infection with or without arthritis who gets an acute flare up of rheumatism coincident with return of a urethral discharge which does not appear to be specific in origin? What of the spondylitic who has, years ago, had gonorrhœa (or may have had) and who has only a few pus cells on prostatic massage with no evidence of gonococci?

In a consecutive series of 50 cases treated as gonorrhœal rheumatism four fall into the group which had rheumatic symptoms previous to any known infection but were definitely flared up by a G.C. infection. It seemed likely from the similarity of their symptoms to those they had previously experienced that the infection had reacted in the same way as any other non-specific infection, causing a flare up in a sensitive individual.

In this series there were also four cases where the onset of rheumatism coincided with a recurrence of discharge, in which gonococci were not found, at times ranging from two months to twenty-one years after gonorrhœa.

One of these cases had gonorrhœa in 1924 but no rheumatic symptoms. The man remained fit until 1930 when, coincident with the recurrence of discharge, he developed a poly-articular synovitis which cleared up in four or five months. In 1941 the same sequence of events occurred. No gonococci were found in the discharge which only lasted a few days. The sedimentation rate was raised to 95 mm. with a white cell count of 11,000. The rheumatic symptoms cleared up in about two months. Was this due to a non-specific infection in the prostate, which was laid open to such an infection by the original damage by the gonococcus, or were gonococci still lurking in his prostate?

Excluding any cases of doubtful aetiology for the moment, the clinical syndrome, if one can call it such, is still very varied. The brunt of the attack commonly falls on the fascia, tendon sheaths and ligaments, the plantar fascia and sheath of the tendo achillis being particularly frequently involved and particularly difficult to treat. Synovitis sometimes progressing to arthritis, especially of the larger joints such as one or both knees, is common. The amount of effusion may be great, wasting is marked but the intensity of the pain varies from slight discomfort on movement to intense pain allowing no rest. The reason for this is not clear. It does not vary with the acuteness of onset of the swelling. The painful type is at times liable to be confused with acute gout.

Monarticular and polyarticular forms occur according to Hensch in the ratio of 2:3. From review of the literature he was of the opinion that it was between 1% and 3% of cases of gonorrhœa that develop arthritis. In Lieut-Col. King's series of 2,689 cases of urethritis it occurred in 2.4%. Weissenbach considered that of 1,445 cases of chronic arthritis 8.8% had a gonococcal infection as their basis.

Certain other conditions occur in association with G.C. rheumatism so commonly as to be suggestive in diagnosis. Freyberg found that conjunctivitis and iritis headed the list with 20%, that keratoderma blennorrhagica occurred in 3% and that calcaneal periostitis was also common. In our series there were three cases of keratoderma blennorrhagica, i.e. 6%, and two of marked calcaneal periostitis.

The sedimentation rate is said to be raised, as is the white count. The results of the complement-fixation test, which should be so useful, must be critically examined. As carried out at many centres it is valueless, and even when a satisfactory antigen is available

it is not a 100% diagnostic answer. Hench in a review found it positive in 80% of proven cases and 60% of probables. It has been found to be positive twenty years after an infection and is not a criterion of activity. A positive complement-fixation test may often be obtained from the synovial fluid, but it is certainly a no more delicate test in the synovial fluid than in the blood; moreover there may be a negative reaction even when gonococci can be isolated.

In our series of cases the average age was 25 but this was obviously influenced by the fact that the group under examination were on military service.

In considering the length of time between the first evidence of infection and first rheumatic symptoms, out of a group of 37 cases, in 3 the symptoms were simultaneous. In 27 the average lapse of time was a little under two weeks while in the remaining 10, the average time was four years. In this latter group were included 3 cases with lapse of three months between the arthritis and the "rheumatism".

The frequency with which various parts of the body were affected was in the following proportions: Knees in 30 cases; ankles 14; fascia (mainly plantar or in the region of the tendo achillis) 10; wrists 8; back 6; feet 6; hands 4; elbows 4; acromio-clavicular, shoulder and neck joints in 2 each; temporo-mandibular joint once.

The sedimentation rate was below 10 mm. in 5 cases, between 10-25 mm. in 4, 25-50 mm. in 10 and above 50 mm. in 6. The white cell count varied from 7,000-17,000, average 10,000.

In 37 cases the average hospitalization was 3.8 months and of these cases 27 were discharged to duty cured and 10 although improved had to be invalided out of the Army.

Treatment.—Apart from local treatment by heat, counter-irritants, massage, rest, &c. the therapeutic field may be subdivided into chemotherapy and hyperpyrexia.

Various preparations of the sulphanilamide group have been used by various techniques with some success. Hench estimates the cures at 60% and the treatment though unpleasant is easy and safe. There has been one death recorded in a series of 5,000 cases. The aim is usually to produce a concentration of sulphanilamide in the blood of 6-10% for ten to fourteen days and a similar concentration also occurs in the synovial fluid. Certain cases cannot take the drug or are sulphanilamide fast and some have actually flared up during its administration. Our treatment of rheumatic cases by this means has been somewhat disappointing and does not agree with the brilliant figures of Bauer and Short.

Fever therapy has been used by injecting T.A.B. (10 or more million) intravenously or by the more difficult but far more satisfactory method of hyperthermy. Here the thermolabile characteristic of the gonococcus is utilized, the body being raised to a temperature of 106-107° F. and maintained at that temperature for six to ten hours and two to four sessions are usually required. By this means Hench estimated that 90% of acute cases and 60-80% of chronic cases of G.C. arthritis are cured. The treatment necessitates, however, expensive apparatus and extreme care and skill if calamities are not to ensue. With care and experience, however, the dangers are not unduly great and are well justified by the results, there being only one death at the Mayo Clinic in giving 2,600 treatments to 620 cases.

The combined treatment of sulphanilamide and hyperthermy is still under trial but the results are very promising.

The assessment of the value of various treatments by Culp gave pride of place to sulphanilamide and intravenous mercurochrome rather than to hyperthermy. Trautman found either sulphanilamide or hyperthermy gave results markedly superior to that obtained by local treatment alone. Hench records 10 cases yielding to combined sulphanilamide and hyperthermy treatment but resistant to both when applied separately. With regard to the benefit from other treatment, the table below shows the number of cases treated by various kinds of physiotherapy. Where possible not more than one type of treatment was started at a time and sufficient time was given to assess its apparent benefit to the patient before anything else was tried. Only where improvement was striking after a particular treatment was a note made to this effect.

| Treatment | Number treated | Number markedly improved by treatment | % improved |
|-------------------|----------------|---------------------------------------|------------|
| Pelvic short wave | 12 | 9 | 75 |
| Local short wave | 13 | 3 | 23 |
| Ionization ... | 15 | 7 | 46 |
| Heat and massage | 11 | — | — |
| Prostatic massage | 3 | — | — |
| Faradism ... | 8 | — | — |

Pelvic short-wave was carried out by means of antero-posterior application of glass electrodes.

Ionization was applied using saline. Where the joint was very painful and swollen the anode was used as the active electrode in order to obtain its analgesic effect on nerve endings and to employ any osmotic dispersal effect on the effusion. Where the condition was more chronic and there was periarticular thickening, the cathode was used as a counter-irritant.

Involvement of the plantar fascia was the lesion by far the most resistant to treatment and seemed to benefit little from any form of physiotherapy.

So far 17 G.C. rheumatic cases have been observed under treatment by hyperthermy, the average number of treatments being between three and four per individual. All have received relief from their pain at least for some days after even their first treatment, spasm and swelling have usually decreased or disappeared and a number have volunteered that they felt better, their sedimentation rates have dropped and they have started to put on weight.

Only the plantar fasciitis cases have proved entirely resistant. It is too early to estimate exactly how many men this treatment will save from discharge from the Army. So far only the worst cases have undergone hyperthermy treatment yet only two have proved resistant, 12% instead of our previous figure of 27%. There has, however, been one death.

Summary.—After analysis of the case histories of 50 cases of gonorrhœal "rheumatism" there appear to be two groups, those complaining of rheumatic symptoms shortly after development of a specific urethritis, usually within two weeks and at all events within three months (81%), and those where a flare up of a long-standing infection originally gonococcal seemed to be responsible.

2-4% of cases of urethritis developed arthritis.

Certain clinical features have been analysed; the knees and ankles were chiefly affected and the fascial structures were next in frequency, the latter being especially resistant to treatment.

The length of hospitalization required, 3.8 months, and the large percentage of resistant cases, 27%, is stressed.

Treatment is discussed and where hyperthermy is not available pelvic short wave coupled with local galvanism appears to give the best results. The importance of hyperthermy is borne out by our figures.

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Lieut.-Col. Ambrose King: In dealing with the metastatic lesions of gonorrhœa the venereologist is primarily concerned with the investigation and elimination of causative foci of infection. For this purpose no distinction need be made between joint and connective tissue infections since they are due to similar causes and run a similar course. The number of cases under consideration in this series is 61 and includes all such cases treated during a period in which the total admissions of patients for the treatment of urethritis were 2,719, made up of 1,784 in whom the gonococcus was found, and 935 in whom the diagnosis was non-specific urethritis. The incidence of metastatic lesions in this group was therefore just over 2%. These cases may be divided into three main groups according to the time relationship between causative genital infection and metastatic lesions.

The groups are as follows:

- (a) 34 patients developed metastatic lesions in the course of the primary urethral infection either before treatment was instituted or during treatment.
- (b) 4 patients who had had a previous attack of arthritis developed a fresh attack or recrudescence of urethritis associated with a recurrence of arthritis.
- (c) 22 patients had metastatic lesions of a subacute or chronic type associated with chronic prostatitis and occurring without obvious determining cause months or years after the original attack of urethritis.

DIAGNOSIS

The association between metastatic lesions and genital infection may be obvious or may require careful search and the application of specialized pathological tests. The principal diagnostic criteria are:

- (1) *Urethral discharge.*—This is the immediate and obvious diagnostic sign which

it is not a 100% diagnostic answer. Hench in a review found it positive in 80% of proven cases and 60% of probables. It has been found to be positive twenty years after an infection and is not a criterion of activity. A positive complement-fixation test may often be obtained from the synovial fluid, but it is certainly a no more delicate test in the synovial fluid than in the blood; moreover there may be a negative reaction even when gonococci can be isolated.

In our series of cases the average age was 25 but this was obviously influenced by the fact that the group under examination were on military service.

In considering the length of time between the first evidence of infection and first rheumatic symptoms, out of a group of 37 cases, in 3 the symptoms were simultaneous. In 27 the average lapse of time was a little under two weeks while in the remaining 10, the average time was four years. In this latter group were included 3 cases with lapse of three months between the arthritis and the "rheumatism".

The frequency with which various parts of the body were affected was in the following proportions: Knees in 30 cases; ankles 14; fascia (mainly plantar or in the region of the tendo achillis) 10; wrists 8; back 6; feet 6; hands 4; elbows 4; acromio-clavicular, shoulder and neck joints in 2 each; temporo-mandibular joint once.

The sedimentation rate was below 10 mm. in 5 cases, between 10-25 mm. in 4, 25-50 mm. in 10 and above 50 mm. in 6. The white cell count varied from 7,000-17,000, average 10,000.

In 37 cases the average hospitalization was 3.8 months and of these cases 27 were discharged to duty cured and 10 although improved had to be invalided out of the Army.

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Pelvic short-wave was carried out by means of antero-posterior application of glass electrodes.

man brand of peptone which before the war was superior to other brands on the market. The finished medium should have a pH of 7.3 to 7.5 and should be warmed to 37° C. before inoculation. All precautions must be taken to exclude contamination and the medium should be exposed to air for the minimum time. A specimen of prostatic vesicular fluid, the largest possible quantity, should be expressed on to the surface of medium contained in a Petri dish and incubated for three to five days. One negative result is by no means conclusive and the test should be repeated on several occasions if necessary. A positive result is obtained when colonies of organisms give a positive oxidase reaction and on smear preparation show an abundance of typical gram-negative diplococci. Unfortunately, it is seldom possible to obtain these organisms in pure culture, and the sugar fermentation reactions cannot be applied. Taken in conjunction with the history and clinical signs, and the general morphology of the organisms there can be little doubt in such cases that they are gonococci.

The oxidase reaction is obtained by the application of one of two dyes to the surface of the medium:

1% dimethyl-phenylene-diamine hydrochloride, or its equivalent tetramethyl solution. Price always uses the dimethyl preparation for it affects fewer groups of organisms than does the tetramethyl dye. Colonies of gonococci and certain others are stained purple black by the reagent when properly prepared and kept.

A practical point about the use of these reagents is that they should be freshly made up in distilled water before use and that the powder should be kept in brown-coloured phials or bottles, since oxidation readily occurs on exposure to light and air. Even when partially oxidized the reagents fail to act satisfactorily and the gonococcal colonies may be missed owing to pseudo-reactions giving an extensive brownish-purple or black coloration.

The test is entirely dependent upon careful and accurate technique, and any relaxation of the standard results in failure to grow gonococci when they are present. Cultures made in this way frequently clinch the diagnosis of gonorrhoea when all other tests have failed to show the presence of the causative organism.

(6) *The complement-fixation test for gonorrhoea.*—This is a valuable though much maligned test; few in this country have mastered the technique. The difficulty appears to be in the preparation of a reliable antigen. The test should be positive in about 90% of cases of acute gonococcal arthritis, but the percentage is much smaller in more chronic cases and in connective tissue infections. A positive test obtained with a reliable technique is strong evidence of gonococcal infection. The test as used in this group of cases did not give helpful results.

TREATMENT

The following methods of treatment have been used to eliminate causative foci of infection:

Urethral irrigations.—Apart from routine general measures this is the oldest form of treatment. Unquestionably it remains a useful treatment and posterior urethral irrigations by means of weak, warm (105° F.) potassium permanganate solution may give prompt success in cases of metastatic gonorrhoea which have failed to react to other methods. A skilful careful technique is essential for the success of this treatment. Errors in technique are likely to lead to trauma of the posterior urethra and extension of the metastatic infection.

Prostatic massage.—This procedure is commonly practised in the treatment of subacute and chronic metastatic infections. In such cases there is almost always evidence of chronic infection of the prostate and the treatment is applied to promote drainage of the infected prostatic secretion. It has seemed to me to be of value in the occasional case in which there is gross infection of the prostate with pocketing of pus as shown by attacks of fever combined with symptoms such as aching pain in the perineum or groins. In such cases the massage should be carried out gently not more than once a week. I have never been convinced of the value of routine prostatic massage given once or twice a week, or more often, in the treatment of chronic symptomless prostatitis whether associated with metastatic infection or not.

Sulphonamides.—All but six of the patients in the present series received one or more courses of sulphapyridine, the amount given varying from 18 g. to 49 g. and the period of administration varying from three to fourteen days, the largest amount given in any one full day of treatment was 10 g. and the smallest 3 g. Eight patients received a course of sulphanilamide in addition. A small group, which was treated with mechanically induced high fever received sulphonamide in the eighteen hours preceding fever—either 10 g. or sulphanilamide, 6 g. of sulphapyridine or 7 g. of sulphathiazole. Sulphapyridine was used in preference to sulphanilamide as being the more effective drug. Extensive use of sulphathiazole has been prevented by the fact that this drug is in short supply. The

suggests the relationship. It may present itself in three ways: (a) There may be a purulent discharge in which the gonococcus is found. This was so in 21 cases in this series.

(b) There may be a purulent discharge in which careful search fails to show the gonococcus. This by no means rules out the diagnosis of gonorrhœa and may on the other hand be evidence of poor tissue reaction against the gonococcus. 22 cases in this series were of this type. In such cases repeated smears and cultures may finally show the gonococcus if delay in treatment is considered justifiable. Even though repeated tests may be negative, they do not rule out the diagnosis of gonorrhœa. The gonococcus sometimes appears suddenly in the secretions later, even while the patient is under treatment, or may be found in the genital tract of an infected partner. The organism may also be found at a later date in cultures from the prostatic secretion.

It will be noted that the proportion of metastatic lesions occurring in patients with so-called non-gonococcal discharges (22 in 915, 2.4%) was twice as great as those occurring in those patients in whom the gonococcus was found (21 in 1,783, 1.2%).

(c) In other cases, 18 in this series, there is no detectable discharge or other manifest sign of urethral infection on examination during the day. It is essential in all such cases that the patient should be examined and a smear taken in the early morning before the urine is passed, and that this first morning specimen of urine should be inspected. Latent undischarged infection may occur and the symptoms may be so slight as to escape the patient's notice. A further possibility is that the patient may have taken drugs of the sulphonamide group without disclosing the fact.

I suggest that conditions such as arthritis and fibrositis, which occur in association with genital infections without isolation of the gonococcus but which are clinically indistinguishable from similar conditions occurring in the course of gonorrhœa, are, in fact, truly gonococcal in origin, but that the available pathological methods fail to demonstrate the organism.

(2) *Urine*.—The urine commonly shows naked-eye changes which are indicative of infection. In acute and subacute infections the urine is turbid or hazy or contains heavy pus threads. On microscopy after centrifuge the smears show masses of leucocytes. In certain chronic cases, and others in which sulphonamides have been administered, the urine may show no evidence of infection. In such cases it is essential to examine the first morning specimen of urine which usually shows some sign if the urethra or its communicating glands are infected. The all-night urine test is of particular importance in the type of case with latent infection in which there is no detectable urethral discharge.

(3) *Rectal examination*.—This may or may not give evidence of recent or old-standing prostatic or vesicular infection such as localized or generalized changes in the consistency of the prostatic tissue or detectable swelling tenderness or thickening of the walls of one or both seminal vesicles. To find such changes is the exception rather than the rule. Occasionally Cowper's glands are a source of infection, and if they are palpable in these cases they may be presumed to be infected.

(4) *Prostatic smears*.—The presence of more than an occasional leucocyte in the prostatic secretion must be considered abnormal and indicates chronic prostatitis. I believe that such an excess once present is likely to persist throughout life, whatever treatment is applied. In such cases the prostate usually feels quite normal to the examining finger. The association of chronic prostatitis and metastatic lesions, with or without the history of past urethral infection, is a very common one. The early morning urine usually gives some additional evidence of genital infection.

(5) *Prostatic cultures*.—The successful cultivation of the gonococcus from the prostatic secretion is vital to the proper study and accurate diagnosis of metastatic gonorrhœa. The two necessities for this test are a suitably reinforced medium and a differential stain for the detection of the colonies of gonococci. Unfortunately the technique is difficult and highly specialized. Under present conditions this test has not been available to us. Dr. Orpwood Price, with whom I first had the opportunity of using this test, was kind enough to give me a note of his present views on this technique, and because I believe the matter to be of paramount importance I have summarized these views which closely agree with my own on the clinical points involved, and are as follows:

The difficulty lies in the preparation of a suitable medium which should consist of an agar broth base enriched by the addition of a natural albuminous fluid containing a high percentage of proteoses—such as whole blood, hydrocele fluid or egg white. War-time difficulties emphasize the importance of attention to detail. The lean meat from which the broth is made is often not fresh but has been frozen and perhaps re-frozen many times. In consequence there is a likelihood of the formation of amino-acids which do not favour growth of the gonococcus. Comparative failure over a period of time was traced to impure agar powder, and difficulty was also experienced in replacing the Ger-

Section of Urology

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Hæmospermia

By GEOFFREY PARKER, F.R.C.S.

THE earliest reference which I can trace to the condition known as hæmospermia is made by Galen who observes that the over-use of any organ in the body makes it necessary to call upon the reserves of "humours" of the other organs. Ambroise Paré observed the condition but his comments are of no scientific value. Coming to more recent times Dalandeterie in 1813 reported two cases of hæmospermia in youths. Demarquay wrote a paper on hæmospermia (1863) and reported a case in a man of 26 with a hydrocele and epididymitis who was a masturbator. He collected four cases from the literature and attempted a classification, the value of which is negated by later observations; he does, however, mention the interesting fact that hæmospermia may occur following orchidectomy, presumably from injury and bleeding into the stump of the vas on the operated side.

Hugues (1894) wrote an excellent paper on hæmospermia and included a large number of references. This is the first important scientific contribution to the subject that I could find. Hugues reports three cases, the first of which was associated with an urethro-prostatitis and fistula in ano, and the second with a stone impacted in the prostatic urethra. Both these cases had painful hæmospermia, and the third case was one of excessive masturbation. This author also gives a classification to which I have little to add. Hugues puts genital tuberculosis as the commonest cause, and at the end mentions that hæmospermia may also occur following the sudden arrest of rectal bleeding from piles. The venous connexion between the prostatic plexus and the hæmorrhoidal veins being an intimate one, if the factor, such as cirrhosis of the liver which caused the rectal bleeding, be still operating, then vesiculo-prostatic bleeding with hæmospermia may be expected. This seems to me a sound enough suggestion, but nobody since then has observed it.

In 1894 the first American publication that I can find on this subject appeared. Lydston reported five cases and considers that the commonest causes of hæmospermia are congestion behind a stricture, seminal vesiculitis and what he calls "unbridled license, short of physical indulgence".

In 1899 Jonathan Hutchinson reported the case of a man of 40 who had a four years' history of hæmospermia. The man had, and there was also a family history of, periodic purpuric rashes. There was no report of a urethroscopy having been done, and Hutchinson suggested a diagnosis of a small angioma in a seminal vesicle.

Chute in 1903 cites a case reported by Guelliot of a man who had eleven emissions in one afternoon and seven more the following day. Many of these latter being blood-stained, and Kroner records the symptom in a unique and fatal case, as the first manifestation of scurvy.

Nelken reported two cases in 1910 and for the first time puts forward the theory of "hæmorrhage *ex vacuo*" which is quoted and supported by many later authors.

This theory postulates that when an orgasm occurs after a period of prolonged continence, the over-distended vesicle is suddenly decompressed and a hæmorrhage occurs into it in the same way that it may occur if a distended urinary bladder or hydrocele have been too rapidly emptied surgically.

With respect to the many supporters of this theory I do not myself feel that there is much in it, because, while a man may have been continent for many years, it is common knowledge that the normal requirements of sexual life are expressed by involuntary nocturnal emissions which may after a time assume a periodicity in their frequency not unlike the menstrual cycle. There seems to me therefore little reason why prolonged abstinence should produce a pathological distension of the vesicle.

Shropshire, writing from St. Louis in 1912, draws a distinction between hæmospermia due to bleeding from the vesicles and the prostate. He says that when the blood comes

results obtained by the administration of sulphonamides in these metastatic cases have been unimpressive. Certainly none has shown striking improvement. When improvement has occurred the extent to which the sulphonamides have contributed to this has been difficult to assess. In some cases the signs have indicated diminution of the severity of the urethral infection, but there has been no concomitant improvement of affected joints or fasciæ. The causative focus of infection in such cases is likely to be in the prostate or seminal vesicles and experience has shown that severe infection of these organs is often very resistant to sulphonamide therapy. Other workers have claimed much more satisfactory results from use of the sulphonamides.

In view of our failure to obtain good results we consider that the combination of high fever with sulphonamides is a more promising field for investigation, but again the relative values of two treatments applied simultaneously are very difficult to assess.

Protein shock.—40 of these patients were treated with fever induced by the intravenous injection of stock T.A.B. vaccine and the results were assessed in a recent paper by Nicol (1942). He found that the best results were obtained in those patients, 30 in number, who developed metastatic lesions during or soon after the administration of sulphonamide preparations. 25 of these patients were cured and 5 were improved as a result of this treatment. Those patients who developed metastatic lesions before treatment was begun made a less satisfactory response. The pain was relieved in most cases but usually the improvement was only temporary—of the 10 patients in this group 3 showed considerable improvement, but the other 7, after temporary improvement, failed to respond. The probable explanation is that in the last group the spread of the infection was of longer standing. There is no doubt that the sooner pyrexial treatment is applied to these cases the better the chance of obtaining good results. Massive doses of T.A.B., such as 700 million organisms in a single dose were used at first. Most patients were given much smaller doses administered by the "Divided Dose" method. By this technique half the dose was given at first and the remainder after four hours, when the temperature had begun to rise from the first injection. The initial total dose was 50 million organisms. This modification gave more satisfactory results and the toxic effects were less severe. It was seldom possible to produce a rise of oral temperature to more than 104° F. by either of these methods.

Hyperthermy.—Mechanically produced high, prolonged fever has been claimed as a specific for the treatment of gonococcal arthritis and almost all the early reports indicate that 90% or more of patients were cured or markedly improved. Trautman (1940) stated that in a series of 129 patients suffering from gonococcal arthritis 117 or 87% were cured or improved as the result of pyrexial treatment, and his figures in a more recent series (1942) are almost identical. Our earlier experience at the London Hospital in the treatment of recent cases of acute gonococcal arthritis with fever sessions of five to eight hours at 106° to 107° F. using the Kettering hypertherm were remarkably satisfactory and there were no failures in a small series. The number of patients treated in the present series is 16, a group which is small and very variable as to type and duration of infection, and from which no conclusions can be drawn. With one exception, a case of severe plantar fasciitis, all have been improved. Eight patients were suffering from chronic conditions of long standing in which complete cure was not to be expected. Two were cured and five remain under treatment having shown considerable improvement as a result of the fever sessions. Temperatures of 106° to 107° F. have been maintained for eight hours in each session and treatments have been repeated at intervals of five to seven days if required. The largest number of sessions given in any one case is six. Most of these patients were treated with fever alone, but in a few the fever sessions were preceded by the administration of sulphonamides. No assessment of the comparative value of fever alone and fever plus sulphonamide can be attempted. The treatment has its dangers and requires a high standard of nursing care for these dangers to be avoided. The more skilful and experienced the nursing the less likelihood of complications. The total number of patients who have received this treatment at this hospital is 110 and there has been one death from heat stroke. About half this number were previously treated at the London Hospital without a fatality.

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There was slight hæmaturia on the night of operation, and since then, i.e. during the last eleven months, no more blood has been seen, in either the semen or the urine. One would have expected this man to have had hæmaturia as well as hæmospermia, but apparently prostatic engorgement was an essential feature of the event.

I propose to cysto-urethroscop him at regular intervals just as we do for vesical papillomata.

CASE III.—A married man of 60 had noticed blood in the sperm for the preceding four or five weeks. There was also nocturnal frequency up to four or five times, and hourly by day. No blood had been seen in the urine, and the immediate specimen was quite clear. There was a history of many attacks of gonorrhœa. On examination there was a tense cystic swelling in the upper pole of the right epididymis and one right inguinal gland was enlarged. *Per rectum* there was some simple lateral lobe enlargement of the prostate.

On cysto-copy there was no intravescicle projection and no apparent source of bleeding.

On the verumontanum there was a large distended venule with a fresh blood-clot above it in relation to the utriculus masculinus.

It was not obvious that bleeding was coming from the dilated venule and so I left it alone. I then tapped the spermatocele and found only the usual opalescent fluid. Cytological examination showed no red cells.

An emission a fortnight after tapping contained some stale blood and there was no further trouble until the spermatocele reformed, when the hæmospermia again occurred.

With the fear always in my mind that I might be dealing with a very early carcinoma of the epididymis, in addition to the spermatocele, though admittedly this is a very rare condition, I advised operation and duly removed the spermatocele. The testicle itself appeared to be quite normal. I kept trace of this man for two years and there had been no return of the trouble.

I do not understand the mechanism of this case; I think it may well have been a simple congestive phenomenon and the cure produced by the removal of the spermatocele simply a coincidence.

CASE IV.—This seems to be the oddest of the four cases. It is that of a Russian, aged 42, who came to me complaining of difficulty and frequency of micturition and a poor stream, combined with hæmaturia, hæmospermia and decreasing sexual power.

He also gave a history of frequent, spontaneous and very painful erections. There was some pain in the left testicle and inguinal canal. There was no history of gonorrhœa and the Wassermann reaction was negative. The external genitalia appeared normal, as did also the prostate and vesicles, palpated *per rectum*.

On cystoscopy the bladder was normal, but in the prostatic urethra to the left of and encroaching on the verumontanum there was seen a cyst (fig. 2), which was probably about the size of a pea.



FIG. 2.—Case IV.

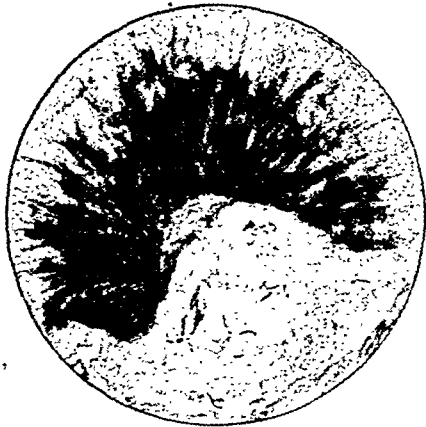


FIG. 3.—Case V.

It was covered by smooth shiny epithelium, over which small blood vessels were coursing. It looked very like a simple ovarian cyst in miniature.

Through an operating urethroscope I pushed a sharp diathermy needle into the cyst and turned on the current. The cyst appeared almost to explode, and at once the instrument was blocked by a thick glutinous fluid not unlike normal semen.

After washing out the urethra I could see no trace whatever of the cyst or its contents, nor could any other cyst be seen under the mucous membrane of the rest of the prostatic urethra. Since operation there have been no further spontaneous painful erections and no further hæmaturia or hæmospermia. That was in June 1932. Six months after operation he wrote saying that he had no further genito-urinary trouble of any kind, but that normal sexual desire was still diminishing. Coitus occurred about once a month and hæmospermia had not occurred again.

I do not know the origin of this cyst but I imagine that this was mainly a mechanical problem as far as symptoms were concerned. The cyst no doubt blocked the ejaculatory ducts and produced congestion and back pressure in the vesicles and pain down the vas to the testicle. The hæmorrhage occurred probably into the vesicle and involuntary and unconscious contractions in the internal vesical sphincter, associated with the lesion itself as a sort of trigger mechanism, may well have been the exciting cause of the spontaneous erections.

CASE V.—This case was referred to me only five days ago. It is that of an unmarried man of 23, with a long history of active bilateral pulmonary tuberculosis and many hæmoptyses. One week before I saw him he had had a nocturnal emission which was pink. There was no pain, but he awakened immediately afterwards, and found the pink seminal staining.

from the vesicles the semen will be uniformly stained pink or red, while if the bleeding is prostatic the semen will be streaked with blood and small clots. This is, I think, what one would expect and is in accordance with my personal observations.

Kenneth Walker (1923) considers that acute seminal vesiculitis is the commonest cause and genital tuberculosis the next commonest. He also mentions sexual excess and systemic blood diseases.

Marion (1935) has never himself seen a case of hæmospermia which was found to have a serious underlying disease as the cause, and has noted on the contrary that the condition tends to clear up spontaneously. He does, however, make a classification based on the literature.

Ashkar and Issa in 1935 reported seven cases of hæmospermia due to bilharziosis, the infection being of the hæmatobium type, with terminal spikes. In all cases the seminal vesicles only were affected and not the prostate; they also note that the vesicles are frequently found infected without hæmospermia ever occurring.

Dr. Parkes Weber had a case of a man who had hæmospermia for thirty years and the blood-stained ejaculations only finished when old age naturally terminated testicular activity.

In the correspondence on the subject in the *Brit. M. J.* of November 9 (1940), a number of theories were put forward. These ranged from constipation to enlargement of the prostate and the male menopause, as possible causes of hæmospermia, but very little evidence was supplied in support of them.

Here is an account of five personal cases:

CASE I.—This is a simple case of trauma. The man was a homosexual of 66, who received a sharp squeeze in the right testicle.

Pain was immediate and very intense. He was sent to me by his doctor twenty-four hours later, still in very great pain, and in addition stated that he had had an emission during the night following the accident, presumably due to local stimulation by the injury. The semen had been dark red, as well as streaked with bright blood. On examination, the right testicle was exquisitely sensitive and could only be palpated with the utmost gentleness after giving $\frac{1}{2}$ gr. of morphia intravenously. There was no discoloration of the scrotum and the testicle, still smooth and globular, and separable from the epidermis, was only a little larger than the left. There was a slight hydrocele. Rectal examination was negative.

The condition presumably was a hæmorrhage within the tunica albuginea and the great pain due to the rise in tension within that inelastic structure.

With rest in bed, cold local applications and morphia for the first two days the condition settled down, and a further emission ten days later was only slightly brown stained.

Subsequent emissions were normal but there was still some testicular neuralgia six months later, presumably due to fibrosis round the nerve endings.

CASE II.—A young Spanish waiter gave a history of a sudden onset of hæmospermia not associated with pain or other symptoms. Every subsequent ejaculation over a period of ten days was blood-stained, and no emission had been free from bright blood, in varying quantities.

There was no history of gonorrhœa, the external genitalia were quite normal, as was also the prostate, and the vesicles were not palpable or tender. In view of what was subsequently found it is curious to note that the urine was clear.

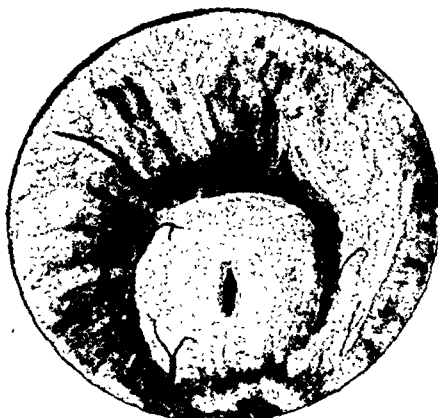


FIG. 1.—Case II.

Cysto-urethroscopy showed a normal bladder and bilateral clear effluxes. On the left side of the verumontanum near its base there was seen a simple papilloma differing in no respect from those commonly seen in the bladder. As the patient was a man with whom I could scarcely communicate by the spoken word, the examination was done under a general anæsthetic.

There was, therefore, no difficulty in this case in doing immediate fulguration through an operating urethroscope. No other papillomata were seen.

Section of Odontology

President—HAROLD ROUND, M.D.S.Birm., L.D.S.Eng.

[April 27, 1942]

Fractures of the Jaws: Should Teeth and Comminuted Bone be Removed?

By D. GREER WALKER, M.Dent.Sc., M.B.

A STUDY of the opinions held on this question shows a curious inconsistency: while the majority of dental surgeons seem to be in favour of removing teeth in the line of fracture, most of them think that badly comminuted bone should be conserved because its removal often leads to the necessity for bone grafting. Is the assumption that there is such a wide difference between teeth and comminuted bone justified and, when bone dies in the mandible, are the results so much less detrimental than those of dead teeth? I have included both subjects in this article because, while no one would assert that dead bone and teeth are identical in their behaviour, they have a good deal in common in certain cases.

There are some important factors which, indirectly, influence treatment: (a) The age and sex of the patient; there is a stronger case for preserving the front teeth in a young girl than in an elderly patient. Reluctance to extract teeth in children and young adults is accentuated by the fact that their mouths are freer from infection and healing is more rapid. (b) The personal wishes of the patient cannot be entirely disregarded: some people are unwilling to part with their teeth, particularly incisors, and while treatment should not be prejudiced on this account, an attempt should be made to save them if there is a reasonable chance of a good result. (c) When other injuries occur in conjunction with jaw fractures, surgical dental treatment may have to be postponed or curtailed: the most serious injury must naturally be treated first, but this is no reason for complete neglect of the fractured mandible, and one of the simpler forms of immobilization can generally be carried out. The circumstances in which preliminary treatment can be given vary considerably: at advanced dressing stations, for instance, it is impossible to give more than first aid, and a suitable form of treatment can only be planned and carried out when the patient reaches the base hospital. While extractions that appear inevitable should be done as soon as possible, no tooth, unless it is so loose that it can very easily be removed, should be extracted unless some stable form of fixation can be applied. Great harm may result, particularly if the extraction is difficult and portions of roots have been left. Such preliminary treatment turns what might have been a simple issue into a complicated one. The factors which have a more direct influence on treatment are: I.—The exact relation of the teeth to the fracture. II.—Condition of the teeth. III.—Condition of the bone. IV.—Value and interpretation of radiograms. V.—The age of the fracture when treatment is begun and the influence of this on the treatment.

I.—THE EXACT RELATION OF THE TEETH TO THE FRACTURE (*Illustrations on p. 23*)

Carious and infected teeth that are not directly involved in the line of fracture should be retained as they may be an invaluable aid in splinting the jaw. When a fracture occurs in the body of the mandible it may not directly communicate with the tooth complex but there are many cases in which the socket of the tooth is actually involved. In Case 1 the fracture is between the 45 but does not communicate with either: in Case 2 the radiogram and drawing show that the socket of the 7 is definitely fractured. This does not refute the argument that the tooth complex is stronger than the bone, but rather shows that in certain cases the force causing the fracture is so applied that its maximum effect falls upon the tooth socket. It is obvious that the premolars should be retained in Case 1 while in Case 2 the 4 should be extracted because of the direct involvement of the socket. Case 3 is interesting because bone can be seen on the anterior surface of the 7, the tooth complex remaining intact while the apex of the 5 is lying denuded of its socket in the anterior fracture line. The 7 was retained in this case because it did not communicate with the fracture, but the 5 was extracted. These three

Since the bleeding he had noticed a heavy feeling in both testicles and groins and down the inner side of the legs, which he described as like rheumatism.

He stated that he rarely masturbated and there was no history of urinary disturbance or frequency of micturition.

Per rectum, the left vesicle was enlarged, hard, nodular and tender; the right vesicle was not palpable; the prostate was normal in size and shape and was not tender. The testes, epididymides, and vasa were normal.

On cystoscopy I found the bladder normal, with clear effluxes from both kidneys.

The upper half of the prostatic urethra was normal, but the distal three-quarters of the verumontanum was covered by pale granulation tissue so that its shape could scarcely be determined, and on the distal slope of the verumontanum there was a shallow ulcer thinly covered with a greyish green slough, and the movements of the urethroscope in this area were painful even after instillation of 4 per cent. novocain. (See fig. 3, page 25.)

I propose to do a double vasectomy with injection of 10% carbolic into both vesicles via the cut vasa, as this seems to me a unique opportunity to save this man at least some of the inevitable miseries of genito-urinary tuberculosis.

I think it might be worth while to do a diathermy cauterization of the granulation tissue on and around the verumontanum. It seems to me likely that this is a very early case of hæmic spread from the lung lesion to the left vesicle with secondary involvement of the prostatic urethral mucosa.

Classification of the aetiological factors in disease is very important. But there is a tendency in recent years to give percentages on too small a number of cases.

Here the majority of references to cases date back many decades, when the exact diagnosis was often unknown and mere surmise. Therefore it is not possible to-day to give more than an impression of what should be done and what is most likely to be found in cases of hæmospermia.

Scheme for case investigation: (1) History taken to elicit story of: (a) Masturbation; (b) sexual excess; (c) gonorrhœa; (d) associated local pain. (2) Examination of urine. (3) Rectal examination. (4) X-ray examination. (5) Cysto-urethroscopy.

The condition most likely to be found in order of frequency would appear to be: (1) Gross masturbation or sexual excess (2) Tuberculous vesiculitis. (3) Acute vesiculitis, irrespective of causal organism. (4) Mechanical urethral obstruction, e.g. urethral stricture, calculi, prostatic cysts, and neoplasms, simple and malignant. (5) Chronic congestive vesiculitis of gonococcal origin (6) Prostatic congestion due to (a) Simple adenoma; (b) long-postponed orgasm. (7) Trauma, surgical or otherwise. (8) Blood diseases, e.g. leukemia, purpura, and scurvy. (9) Rare possibilities, local and systemic: (a) Primary disease of the testicle and epididymis; (b) carcinoma of a seminal vesicle; (c) cirrhosis of the liver, with leak of portal system through prostatic plexus; (d) bilharzial vesiculitis.

I am much indebted to my house surgeon at the French Hospital, Dr. Galewski, for helpful suggestions and assistance with the German translations.

In the subsequent discussion, Mr. Clifford Morson and other speakers mentioned a number of cases which they had seen, where the hæmospermia appeared to be due to hypertension without any local disease, and relief of the hypertension cured the condition.

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In similar fractures small apices might be left with safety, and this would certainly appear advisable if the difficulty of extraction is likely to outweigh the reasons for it. It is obvious that extraction of teeth in a badly comminuted fracture is much easier than in one of the subperiosteal variety.

Infection.—The degree of infection in the teeth is closely bound up with similar changes in adjacent bone, so we must consider also, at this stage, all infection specially connected with the teeth that is actually in the bone. Case 10 A shows a subperiosteal fracture extending obliquely backwards from the 8 to the angle of the jaw, and there appeared to be connexion with the apical infection of the last molar. The second molar was also badly infected but this was not involved in the fracture. The relation of the infected zones is clearly shown in the drawing. Because the fracture was subperiosteal, it passed through the bony wall of the abscess cavity and did not in fact communicate with the chronic inflammatory tissues: if it had been displaced, these tissues would have been disrupted and the infection directly connected with it. When there is no displacement I think the teeth may safely be retained in such a case until the fracture is "sealed off", and removed after immobilization has been in progress for at least three weeks. In Case 10 extraction was postponed so as to find out whether the presence of the teeth would retard recalcification of the fracture, but it did not appear to have done so: they were removed after three months, and radiogram 10 B, taken just before this was done, shows a good result. Case 11 was admitted to hospital with swelling of the jaw which had appeared a few days before admission: the jaw had been fractured six weeks previously but had had no treatment. A fracture line apparently involving the third molar is seen in 11 A and lack of immobilization had led to slightly increased decalcification. The 8 was partially erupted, the anterior cusps just showing, and no movement of the fragments could be detected. The history showed that the original traumatic swelling had disappeared one week after the injury, but a certain degree of trismus had remained. This was a predisposing cause of the flare of infection underneath an already infected gingival flap which caused the later swelling evident on admission. As in Case 10, the fracture skirted the zone of infection and, as the injury was already of six weeks' duration and the fracture partially united, all that was required was treatment for the infected wisdom tooth, which was extracted with care: radiogram 11 B. Case 12 was an unusual fracture of the symphysis which had been weakened by a large cystic cavity in that region. Radiograms A and B show the cyst and considerable apical infection of the adjacent teeth: neither this nor the cyst is in communication with the fracture. The cystic cavity was not opened, the teeth were retained, and the mandible was immobilized for five weeks: a good result is seen in radiograms 12 C and D, taken two months later. This case might have been complicated if the teeth had been removed and the cyst cavity opened.

Conclusions.—It is of paramount importance to decide whether infections such as those evident in the last three cases are connected with the line of fracture or separated from it by a thick layer of chronic inflammatory tissue through which the enclosed infection cannot pass. It would, of course, be a mistake to leave teeth if the infection connected with them has direct access to the fracture but in my experience this occurs only when the fragments are displaced, thus causing rupture of the inflammatory tissues. In subperiosteal fractures a better result is likely to be obtained by postponing extraction until the fracture is "sealed off".

Stage of development and eruption of the teeth.—These present two different problems: first we have the various stages of development in children's teeth and secondly, in older patients, partially or completely unerupted teeth which, although fully formed, have been prevented for some reason from coming into alignment with the others.

Fractures of the mandible are rare in children and their mouths are comparatively free from infection, which no doubt partly accounts for the characteristic rapidity in bony repair. Case 13 was a child of 9 who had, among other injuries, a bilateral fracture in the 7,3 regions: there was a little displacement on the right side but none on the left. Radiograms A and B show the fractures and in particular the unerupted teeth concerned. It was thought inadvisable to remove these, and immobilization produced a very satisfactory result as can be seen in 13 C and D, taken a couple of months later. Early treatment undoubtedly contributed largely to the success in this case but the effect of delay is obvious in Case 14. This child, aged 9, was admitted to hospital six weeks after her jaw had been fractured. The infection then evident was due to the developing 17 which had been displaced so that it was lying between the lingual periosteum and the bone: this tooth had not been removed nor had adequate immobilization been applied. In this case removal of the displaced tooth and fixation of the fracture would probably have been the best immediate treatment and delay caused considerable infection

cases illustrate the need of finding out whether there is any connexion between the teeth and the fracture, but this is sometimes extremely difficult and radiograms may be entirely deceptive, which may account for the dictum that the teeth on both sides of the fracture line should be removed. In some cases this may be indicated but I do not think it should necessarily be the routine treatment. In Case 4 there is an obvious fracture in the 14 region: this tooth was removed and, after some decalcification had occurred, a radiogram showed that the 13 was also involved. This fracture might be described as subperiosteal: one tooth in the line was retained and the other was extracted and healing was uneventful. Until some decalcification has occurred the exact extent of some of these fractures may not be apparent, and a routine "follow up" of radiograms should be carried out in all cases. Case 5 shows a fracture between the 871 teeth, the third molar being involved while the second was not: the theory that the third molar should be kept for a short time to assist in control of the posterior fragment was followed with disastrous results. Control of the posterior fragment should not be allowed to influence the decision in favour of retaining this tooth if extraction is the better course on principle. It may seem bold to take this view since the introduction of the "pin" method of immobilization but I think the tooth should only be kept if the other reasons for doing so are decisive.

Conclusions.—It should first be ascertained whether the teeth are definitely involved in the fracture and if they are, the relative merits of retaining or extracting them should be carefully weighed. If there is a tooth in the posterior fragment, its utility for the purpose of control should not be allowed to override all considerations in favour of extraction.

II.—CONDITION OF THE TEETH (*Illustrations on pp. 23-26*)

There are four aspects of considerable importance in treatment: Extent of the trauma, degree of infection, stage of development and eruption.

Trauma.—Teeth subjected to injury may have either their roots or their crowns fractured. Case 6 demonstrates a fracture in the third molar region, the anterior root of this tooth being broken, and there was definite communication between the root and the fracture. Both tooth and root were carefully removed with any loose pieces of alveolar bone likely to sequestrate and radiogram 6 B, taken five weeks later, shows a satisfactory result. The risk entailed in keeping such a tooth far outweighs its possible use in preliminary control of the posterior fragment. Fracture of the crown of the tooth is a more obvious injury, and when this occurs the pulp cavity is open to infection. In Case 7 the crowns of the 654 were knocked off by a piece of shell which also caused a slight subperiosteal fracture, and, assuming that there was connexion between the teeth and the fracture, the question arose as to whether these pulp cavities, when they became infected, would prejudice the treatment. I think a fracture of this type should heal quickly enough to become "sealed off" before any infection can reach it from the pulp canal and, when the canals are left open, they are less likely to give rise to acute infection. The roots, if they are left, can be removed after about three weeks when the fracture is partially united and unlikely to take any harm in the process. The trauma caused by removing them immediately would be more detrimental than the danger of keeping them for three weeks: actually in this case it was impossible to contemplate extraction at an earlier stage because of the patient's other injuries. Radiogram 7 B, taken five months later, shows that union was complete. Case 8 A shows a large decalcified zone with the root of the 51 on the anterior margin. The delayed union seen in this radiogram, which was taken on admission to hospital six months after the injury, should not be ascribed to delay in removing the root so much as to lack of adequate immobilization: cases like this demand a long period of fixation which ought not to be curtailed or interfered with, and although extraction of the root and treatment for infection was undoubtedly the right course, fixation was even more important. Recalcification is seen in 8 B taken after the jaw had been immobilized for two months: the laying down of bone is obvious in the "lipping" of the lower border. In Case 9 extraction of the third molar had been attempted under an anæsthetic, but the anterior root was left behind and no fixation was provided. The patient was admitted to hospital a week later with much swelling of the jaw: the root was then removed and the jaw was fixed to the upper teeth for two and a half months. Radiogram 9 B was taken four weeks after this. Such a long period of immobilization was unnecessary from the point of view of union, but it was prolonged for the purpose of hastening and observing the recalcification, which is shown in 9 C, taken three months after the injury, to be almost complete.

Conclusions.—These four cases demonstrate fractures with broken teeth in which the treatment was successful, but we cannot assume that removal is always the better course.

result was clinically satisfactory. Posterior alveolar fractures occurring in the maxilla have the added complication of close proximity to the antrum, and in removing the teeth related to them there is a danger of establishing connexion between the antrum and the mouth. Actually the line of fracture does not involve the apices of these teeth, the outer and palatal walls of the antrum being broken instead. Case 20 shows a fracture in the 87654 region caused by a blow: the teeth in this broken fragment were not removed and the jaw was splinted for seven weeks, at the end of which time it seemed unnecessary to extract them. Cases 18, 19 and 20 were all young people with healthy teeth and conservative treatment appeared to be justified; but in Case 21, a patient aged 40 with a fracture of the 123456 region, there was considerable gingival and apical infection. Here, apart from the injury, removal of the teeth was long overdue and those involved in the fracture were extracted with the fractured portion of the alveolus. Radiogram 21 B, taken a couple of years later, shows that a considerable loss of bone was involved in this operation. While it was obviously necessary to remove these teeth, I think it would have been wiser to try to preserve so large a fragment and this could probably have been achieved by extracting the infected roots immediately but postponing removal of the other teeth until there was some union in the fracture. In Case 21 infection was present before the injury, but Case 22 demonstrates the marked degree that may develop afterwards. In this both teeth and alveolus were fractured in the 321123 region (22 B) and there was also a fracture in the 32 region of the mandible (22 A and C). When I first saw this case after six weeks, all these teeth were loose and there was a good deal of infection in the bone: the extent of the injury made retention of the teeth a very doubtful proposition and, when infection was added to this, it became imperative that the teeth, portions of roots and sequestered bone should all be removed as soon as possible. Following this operation progress was good but the inevitable delay in union caused by infection is seen in radiogram 22 D, taken after six months.

Conclusions.—In extensive alveolar fractures there are usually strong indications for extraction, particularly when they are associated with infection; but the teeth may safely be retained in cases that are not of a serious nature. If large portions of the alveolus are fractured, as few teeth as possible should be removed until some union has taken place.

Complete fractures.—It is sometimes difficult in the less severe cases to determine whether fractures of the mandible are simple or compound, but it is generally assumed that the majority are compound. I think that in certain fissure or stellate fractures there is no rupture of the soft tissues and no communication with the mouth; and these might therefore be more correctly classed as simple or subperiosteal fractures. Case 23 was struck on the side of the jaw by a landing aircraft: on admission there was pain and tenderness in the region of the blow but all other clinical signs of a fracture were absent. Radiogram 23, taken six weeks after the accident, shows a good example of the stellate type: in all probability it had no communication with the oral cavity and there was no need to extract the teeth. This kind of fracture is rare: the common type met with in civil life is the uncomminuted fracture of the mandible with a vertical or oblique fracture line, the displacement depending upon the site. The problem in both is primarily that of the relation of the teeth to the fracture and it is unnecessary to add to what has already been said on the matter. In comminuted fractures and those with loss of bony tissue the extent of damage to the bone is of an extremely varied character and the second problem under discussion, "should comminuted bone be removed", begins to arise. I must therefore give a more detailed account of the following cases. We have already discussed the comminuted alveolar fracture and its treatment: there are two other types, the one in which the main damage is to the lower border of the mandible and the other with fragmentation distributed more or less evenly throughout the body (Case 24). This man was struck on the chin by a piece of shell and when he was admitted to hospital five days later the lower anterior teeth were painful and there was a large hæmatoma under the tongue, but no tear in the buccal mucosa. Apart from a four-tailed bandage no form of immobilization had been applied. Occlusion of the teeth had not been disturbed. The metal had not entered the mandible: the wound had been allowed to close and drainage had not been maintained. Radiogram 24 shows extensive comminution of the anterior part of the mandible, the damage being more severe in the lower part of the symphysis. It is commonly supposed that union in alveolar bone is rare and that since this part of the mandible is transient, it is unnecessary to attach much importance to preserving it, but we must apply this theory with some reservation. In Case 24 I would confine extractions to a minimum as the alveolar bone may be the only existing link in continuity if the smaller fragments concerned in the fracture should sequestrate. Apart from this reason, if all the teeth near the fracture were removed it would entail loss of the 5432112345 which would open up a very large bony surface in a fracture that

in the bone, so that when the case was treated eventually, it appeared necessary to remove not only the displaced tooth but the first molar also. Sequestration followed (14 B) and the result, shown in 14 C, was non-union. Whatever treatment had been applied after six weeks it is doubtful whether union would have taken place; but I think that while it was right to extract the first molar, it would have been better to wait until the infection was under control. Permanent teeth differ from deciduous teeth in that they occupy a relatively larger area of the mandible. This is especially true of the first molar, whose removal is likely to cause a good deal of trauma and open up a large surface of bone to infection if the fracture has already become infected. Case 15, a boy of 14, fractured his jaw through the 8|3 regions and the anterior fragment was displaced slightly downwards. The fracture was reduced and the jaw fixed for four weeks by wiring the teeth together but the teeth were not removed. The unerupted 8| had no adverse effect on union, which took place uneventfully. If this case had not had immediate treatment and infection had developed, the result might have been similar to that in Case 14. Cases 16 and 17 show fractures through the third molar region in adults, this tooth being unerupted in both. It is clear that the trauma involved in removing these fully formed and deeply embedded teeth would constitute a graver danger than that of retaining them, which is only a potential one. Unerupted teeth in adults should generally be retained in the simpler fractures, at all events for a time; but in the more severe types it is often found that the reasons in favour of extracting these teeth are so strong that they outweigh consideration of the harm caused by additional trauma.

Conclusions.—Broadly speaking, a conservative line of treatment is indicated in fractures involving developing and unerupted teeth. The former may be regarded as free from infection and unlikely to cause trouble: the latter may be retained at some risk but this is more than counterbalanced by the trauma that would result in removing them.

III.—CONDITION OF THE BONE (Illustrations on pp. 27, 28)

Fractures of the jaw have been described as partial and complete and under this classification I shall briefly discuss five cases of partial or alveolar fractures before proceeding to the more important type.

Partial fractures.—These in everyday life are almost invariably alveolar; but in war time we meet with cases in which part of the lower border of the mandible is shot away although there is still continuity in the upper part of the bone. The latter type has little bearing on the subject under discussion as repair is generally uneventful. It is only in war injuries that cases of alveolar fractures are seen with much comminution and loss of bone and teeth. With extensive damage of this kind a careful débridement should be carried out and all pieces of tooth, damaged teeth and loose pieces of alveolar bone removed. When the case is seen immediately after the injury this operation may be accompanied by suturing the gum: in some cases seen at a later date when infection has developed, it is wiser to control the infection before intervening surgically. There are, however, many cases in which damage is not so extensive as to call for removal of teeth. Alveolar fractures are more common in the maxilla than the mandible where the incisor region is the only part likely to be fractured. In those of the maxilla there is a more favourable prognosis with regard to the teeth as can be seen in Case 18. This patient, aged 22, was kicked in the mouth by a horse, causing a depressed fracture of the alveolus in the 432| region. The teeth were displaced *en bloc* inside the bite and the crown of the 1| was broken, but the apices of all remained intact (18 A). The fracture was reduced the following day and a splint was cemented to the upper teeth (18 B) and kept in situ for three months. The pulp canals were not filled until some months later so as to find out if this delay would have any adverse effect on the healing of the fracture: satisfactory union is seen in 18 C, taken shortly after the canals were filled. In such cases a more correct procedure, and one minimizing infection, would be to devitalize these teeth at the time of reduction, filling the pulp canals but postponing apicoectomy for consideration later. Case 19 shows an uncommon fracture of the mandible: this patient, aged 20, was kicked in the front of the mouth while playing football and sustained a transverse fracture of the alveolus across the symphysis. The four incisors which were lingual to the canines were displaced labially *en bloc*. A splint was cemented in position the following morning, but the impacted displacement was not corrected as it produced no abnormal occlusion apart from an edge to edge bite, and added trauma might well have led to the loss of the teeth. The splint remained in position for two and a half months, a period that might have been shortened, but it was thought best to err on the safe side. Radiograms 19 B and C, taken six months and a year later, show no evidence of infection and the

Conclusions.—The general tendency towards conservatism in removal of comminuted bone is sound provided that the comminution is not too extensive and there is no gross loss of bony tissue. It is important that a decisive line of treatment should be followed and each case should be treated without prejudice. The simple ones respond to conservative methods while the period of treatment in those associated with loss of bone may be considerably shortened by radical débridement.

IV.—VALUE AND INTERPRETATION OF RADIOGRAMS (*Illustrations on p. 29*)

The help of radiograms in establishing the exact injury and extent of damage to the bone is easily perceived, but a point to which we perhaps pay rather less attention is the degree of decalcification that can be seen in connexion with some fractures, and its significance. This decalcification may be caused by lack of adequate immobilization and resulting traumatic hyperæmia, but in other cases infection undoubtedly plays an important rôle and accounts for the infective type. In the mandible we generally find that prolonged trauma in the fracture, caused by lack of suitable immobilization, leads to an early onset of infection. The following three cases are good examples of extensive decalcification. Case 28: this man fell from a window seven feet high and was admitted to hospital with concussion, but a jaw fracture seems to have been undetected as no treatment was given. After returning home he developed a swelling and when I saw him six weeks after the injury, this was of considerable size. Occlusion was undisturbed but abnormal movement in the $\overline{3}$ region suggested a fracture: radiogram 28 A confirmed this and showed an excessive degree of decalcification. The patient was admitted and after a splint had been cemented to the lower teeth the abscess was opened externally: eight days later, when infection was under control, the $\overline{3}$ was extracted and drainage was established by connecting the original abscess incision with the buccal sulcus adjacent to the socket. In this type of case with acute infection the sequence of the treatment is very important: the fracture should first be immobilized and secondly the infection controlled by adequate drainage, and only after this should any teeth that may be detrimental to the result be removed. The case is different if infection is not acute, as in chronic sinuses: here there is little danger in removing the teeth and establishing drainage at the same time, once the jaw has been immobilized. In Case 28 it might be assumed that the premolar should be extracted as well as the $\overline{3}$. If the decalcification were chiefly infective this would probably be advisable, but when trauma has played so large a part for as long as six weeks it is found that recalcification will probably take place around this tooth, as shown in radiograms 28 B and C, taken ten weeks and twelve months after immobilization was begun. In this instance the jaw was splinted for three months: delay in union was caused primarily by increased and prolonged trauma and secondarily by infection. The prognosis in such cases is good if adequate immobilization and elimination of infection are rigidly carried out. Case 29 was an epileptic who fell and struck his chin on the ground. He attended hospital next day with some swelling of the jaw, but no obvious movement of the fragments. Radiogram 29 A showed a fracture involving the $\overline{5}$ region, this tooth being unerupted and lying vertical in the body of the mandible. In view of the nature of this fracture it was thought inadvisable to extract the tooth but construction of a splint was begun: the patient, however, did not return to hospital to have it cemented in position until three weeks later when the swelling had increased. After the jaw had been immobilized the abscess was opened externally but the tooth was not extracted until some time after the infection had been controlled and the splint had to be removed and another one made to replace it. The first mistake was lack of care in planning the treatment and it taught me to refrain from including in the splint any doubtful teeth or from covering over any that are unerupted, for these cannot be removed with it in situ. The second mistake was in leaving the tooth so long. The problem here was twofold: in the early stages of the fracture the trauma caused by removing the tooth would have been too extensive to justify this procedure. The development of severe infection on the other hand indicated early extraction; but a better balance might have been struck between the two courses and the tooth should have been removed at the earliest possible moment after infection was under control. Mismanagement of immobilization and delay in extraction retarded bony union and radiogram 29 B shows a large zone of decalcification four months later which might be mistaken for non-union, the edges of the fragments tending to become rounded off. Fixation had to be prolonged to nine months and 29 C shows the extent of recalcification sixteen months after the injury: the almost eburnated ends of the fragments can still be seen surrounding the new bone and it is interesting to see recalcification in the alveolar bone also. Case 30 fractured his jaw in the $\overline{3}$ region by a fall and had attended a hospital where a splint

had already become infected. In a case of this kind it is better to immobilize the fracture and treat the infection before extracting the teeth, and drainage should be established as a routine procedure for sequestration is certain to occur. This policy was carried out in Case 24. When it was thought opportune, a month later, the four incisors were extracted and subsequently the canine also. The result was bony union. Treatment in Case 25 was a difficult problem as the possibility of union was doubtful, and I think it is worth while to discuss the alternatives that presented themselves. This case was a young girl of 20 who received a penetrating wound of the right cheek, the bomb fragment lodging in the mandible. This and several pieces of bone were removed shortly after the injury and the wound was sutured: the teeth were then wired together with eyelet wiring. Six days later the wound in the cheek seemed to be healing normally and there was little clinical evidence of active infection in the mandible, but in spite of this apparently favourable clinical picture, radiogram 25 showed loss of part of the lower border and severe damage in the tooth-bearing area of the 7543|, the 7| being dislocated out of its socket. The first thing to be decided in this case was whether there was any ultimate possibility of bony union. If this question could be answered in the affirmative the obvious treatment would be extraction of the 754| with any small pieces of alveolar bone, and immobilization of the jaw. But the fact that the 8| was unerupted made control of the posterior fragment impossible and this was a definite handicap in the treatment. It might be argued that the 7| could be kept for a time for this purpose, but the value of keeping it would probably be outweighed by its potential danger; and for how long in any case could it be retained? This question can be answered by consideration of the fact that the fracture would obviously need a long period of immobilization and the 7| could only be kept for a fraction of this time. If this conservative line were followed and sequestration allowed to take its course, the result might be union or it might not, and in these circumstances would not radical treatment be the surer method? Personally I think that the loss of the lower border of the mandible coupled with fracture of the alveolus and the problem of controlling the posterior fragment turned the scale in favour of performing a radical débridement as soon as possible, as this would at least secure healthy bone ends for an early graft. Case 26 emphasizes the importance of decisive treatment. This man was admitted to hospital with a fractured mandible caused by a bullet which entered the cheek as seen in radiogram 26 A. There was no apparent laceration of the mucous membrane and occlusion was undisturbed. First the bullet was removed, dependent drainage was established and the 2|, of which the crown had been badly broken, was extracted: secondly, seven days later, it was thought necessary to remove the 543| also, with the alveolar bone islet; and lastly, four weeks later, some sequestra were removed. One might justly ask whether all these operations were essential within five weeks or if it would not have been better to include them all in one at the outset, and immobilize the jaw without further interference. While one school of opinion might advocate a minimum of surgical intervention in the early stages of this case, another would prefer to remove the 5432| immediately and possibly the alveolar bone also. In a case of this kind it is essential that the treatment should be carefully planned.

In comminuted fractures the decision as to which teeth should be extracted, and how much bone removed, is by no means easy. It rests primarily upon whether a graft is likely to be necessary or not. The maxim that no bone should be removed is firmly rooted because the possibility of union, however remote, is generally present. Treatment on this assumption is justified in many cases, but there are others in which it is obvious from the very beginning that bone grafts are inevitable, and sequestration simply delays and complicates the issue. However strongly we may hold this radical point of view it would be rash to dogmatize, as there are borderline cases in which the pros and cons are too evenly weighted for a fair decision: in these I think the policy of temporization is justified until the stage of sequestration is reached, when the condition should be carefully considered and, if sequestration is unduly severe and prolonged, the radical method of treatment should be adopted. Case 27 is an example of what might be termed the borderline type. This patient had multiple wounds: the foreign body seen in the radiogram was in the tongue, and the entry wound in the jaw was kept open and "through and through" drainage established. No teeth were involved in the fracture and at first there seemed to be a reasonable chance of union, but radiograms 27 B and C, taken at intervals of two and seven weeks respectively, showed that the condition was becoming progressively worse. It was then decided that a bone graft could not be avoided and the sequestra between the fractured ends of the bone were removed as seen in 27 D; this procedure undoubtedly limited the period of infection. In looking back, I think this treatment might with advantage have been applied some weeks earlier. The wait and see policy can be carried too far.

and the external one. After this operation progress was uneventful and 32 B, taken two months later, showed good recalcification. The splint was now removed and bony union was clinically confirmed. Little can be said in favour of retaining teeth in such a case, but it is important when removing them to leave behind no portions of the alveolus that are likely to sequestrate and delay union: further, when a cavity of this kind is kept open with packing for a short time, the possibility of a blood-clot breaking down and becoming a source of infection likely to interfere with healing is remote.

When there are more severe injuries than that of the jaw the patient may not be transferred until a week or ten days after the accident; and when they endanger life to a greater degree, this interval is necessarily much longer. Success in these cases largely depends upon the treatment given to the jaw fracture in the meantime, and this is well illustrated by Cases 33 and 34. Case 33 had an aeroplane crash which caused a fracture of the symphysis and a deep wound of the lower lip communicating with it: the $\overline{21}$ was knocked out of its socket. A couple of days after the accident a splint was cemented to the lower teeth and the patient was transferred eight days later, when all that could be seen was a slight discharge from the wound below the chin. Radiogram 33 A showed that there was an unerupted tooth involved in the fracture. Owing to the treatment so far carried out it was possible to operate two days after admission and the unerupted tooth and remaining incisors were extracted: it had been thought beforehand that two of the incisors might be saved but at the time it was found necessary to remove them all. They had been included in the splint, but this was achieved by cutting through the necks with a fissure bur and elevating the roots out of their sockets. The wound in the mouth was packed and the one under the chin enlarged for better drainage. Radiogram 33 B, taken two and a half months later, showed an advanced stage of union and the splint was removed. There appears to be no advantage in connecting the submandibular drainage with the mouth unless infection is likely to persist for some time. Case 34 was a woman aged 35 who received multiple injuries during an air raid. After her head and chest injuries had been treated for five weeks she was transferred to the Jaw Centre with a fractured mandible in the right premolar region. This had not been satisfactorily immobilized with the result that there was a considerable amount of discharge from a sinus below the fracture, and radiogram 34 A showed decalcification and sequestration. A splint was cemented to the teeth and an operation was performed in which the external sinus was explored and many small fragments of bone were removed. The gum was then reflected over the fracture in the premolar region and the sequestered alveolar fragments were removed. The wound was irrigated with saline and a piece of corrugated tubing was passed through into the mouth: after this had been left for a week it was reduced in length and frequent irrigations were given. Radiogram 34 B, taken three weeks later, showed that there were no loose fragments of bone remaining. I think it would have been incorrect to extract any teeth at the original operation as it would undoubtedly have increased sequestration: if an infection of this kind is controlled first, fewer complications are likely to arise. It should be emphasized that in this case there was no curetting and very great care was taken in the removal of the sequestra. Three weeks later the adjacent teeth were extracted and bony union occurred in due course.

The reason for transferring a case to a Maxillo-Facial Centre after an interval of some months from the time of injury is generally delayed union which is caused either by inadequate immobilization or some persistent form of infection. Case 35 was sent to the Centre four months after he had been wounded by a bullet. The radiograms showed a fracture in the 456 region with evidence of delayed union and there was a cavity of residual infection at the alveolar end of the fracture. This type of case usually responds within a reasonable time to treatment consisting of immobilization followed by extraction of adjacent teeth and opening of the infective cavity into the mouth. As the infection was chronic in Case 35, all this was done in one operation. In old-standing injuries of this kind the period of fixation must be prolonged according to the degree of delay in union, and any associated residual infection must be eradicated.

Conclusions.—In treating a case immediately after the injury the problem might be described as purely traumatic. After a short interval infection begins to play a major part and due respect must be accorded to it.

Before concluding this paper I must make some reference to cases in which the results are atypical and also to the danger of basing assumptions as to the right line of treatment upon issues which, on a wider view, prove to be exceptional rather than general. A brief analysis of the two following cases may be of value as they are somewhat unusual. The first was treated on the maxim of retaining the teeth and the second on exactly the reverse. Case 36, a man of 46, had a fracture running obliquely through the right body of the mandible (radiogram 36 A) as well as bilateral fracture dislocations. The

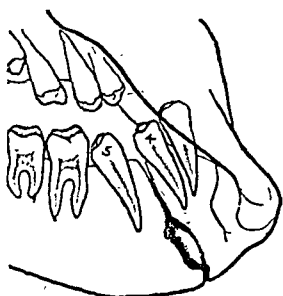
had been cemented to the lower teeth: he had returned ten days later with a swelling in the region of the fracture and one week later the $\overline{4}$ had been extracted and the abscess opened externally. When I first saw him six weeks after the accident it appeared from the history that at some stage in the treatment the original splint had been replaced by an arch wire method of fixation which was still in situ. Acute infection was still present and the original abscess incision was still discharging. Radiogram 30 A showed changes indicating an acute infective condition of the bone and there was a chronic apical abscess at the root of the $\overline{5}$. A week later the infection appeared clinically to have subsided and it was thought safe to extract the $\overline{35}$, but this was followed by a very acute condition characteristic of osteomyelitis, involving both sides of the mandible: 30 B, three weeks later, showed the extent of the infection on the same side as the fracture. In 30 C, taken after an interval of six months, recalcification was evident but it was incomplete in the lower border and a zone of residual infection appeared in the form of a cavity around the $\overline{8}$. The lesson to be learned from this case is that extreme caution should be observed in the removal of teeth when decalcification is the result of an acute infective process rather than of traumatic origin.

Conclusions.—The value of radiograms in the treatment of fractures is not limited to showing the extent of the trauma: they are essential also on account of the subsequent changes they may indicate, and of these the most important is that of infection. Due attention should be paid to abnormal decalcification even in the absence of corroborative clinical findings.

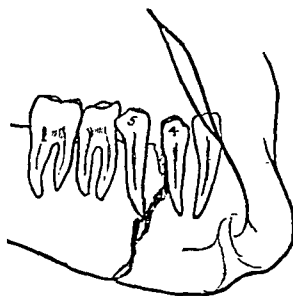
V.—THE AGE OF THE FRACTURE WHEN TREATMENT IS BEGUN AND THE INFLUENCE OF THIS ON THE TREATMENT (*Illustrations on pp. 30-32*)

A rough analysis of jaw fractures at the present time shows that while many cases are admitted to a maxillo-facial centre within twenty-four hours of the accident there are others in which various reasons make it impossible to transfer them to one of these hospitals until some time has elapsed. Variations in the time between injury and admission must necessarily influence the treatment in each case.

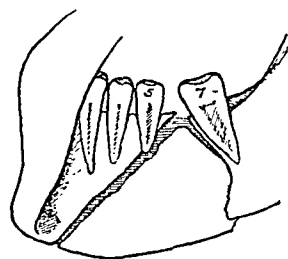
Case 31, an air-raid casualty, was treated within a few hours of the injury by a mobile maxillo-facial team at a hospital some distance from the Centre. It is unfortunate that more cases cannot be seen as early as this, for immobilizing a fracture at this stage and working in a field where infection has not had time to develop opens up great possibilities in the treatment. In this case a missile of unknown type had caused extensive damage and loss of soft tissue and bone in the right cheek and underlying portion of the mandible which made radical débridement necessary. All fragments of teeth and bone were immediately removed except one large piece which was well attached to the soft tissues. Radiogram 31 A shows the extent of comminution before this operation and in B, taken three months later, the rounded ends of the two fragments can be seen with the retained portion of the lower border united to the anterior one. At this early stage it was a comparatively easy matter to perform the débridement through the cheek wound, but if the case had been admitted to the Centre some days after a primary suturing, it would have been impossible to do more than maintain or establish drainage and leave sequestration to take its course, thus delaying the final issue. Case 31 is exceptional: there is usually an interval of twenty-four hours or more before the patient is transferred which often means that he has already been anesthetized for excision and suture of the wounds and the jaw may have been immobilized either then or later, when the patient has recovered from the anæsthetic. It would be unwise to repeat the anæsthetic for some days unless it is absolutely necessary, and this means delay in operative procedure. If immobilization is inadequate a more stable form can be applied in the interval, and drainage can be established by removing sutures in the most dependent end of any wounds related to the jaw. This, combined with chemotherapy, application of heat and irrigations to the mouth, should check developing infection. Case 32, a man of 36, was also an air-raid casualty. A laceration of the left cheek had been excised and sutured and the fracture in the left canine region supported by a suitable bandage. Radiogram 32 A showed a comminuted fracture of the $\overline{34}$ region with an islet of bone containing these two teeth. By the time this case reached the Centre twenty-four hours after the injury, a large hæmatoma had developed in the cheek wound: this could be only partially evacuated and drainage was established in the lower part of the wound, which was below the fracture. A cap splint was cemented to the teeth, heat was applied to the jaw and the mouth was irrigated. After four days all swelling had subsided and the wound was healthy, so the $\overline{34}$ were removed along with the alveolar fragment. The wound in the mouth was packed open and there seemed to be no reason for establishing connexion between this



CASE 1



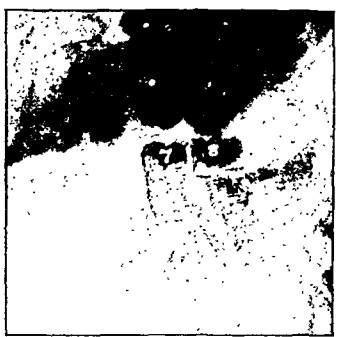
CASE 2



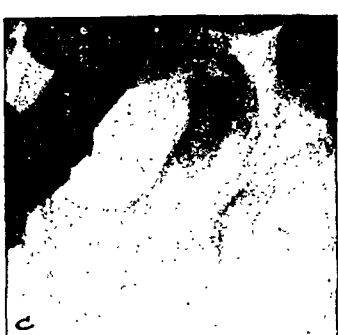
CASE 3



CASE 4



CASE 5



CASE 9

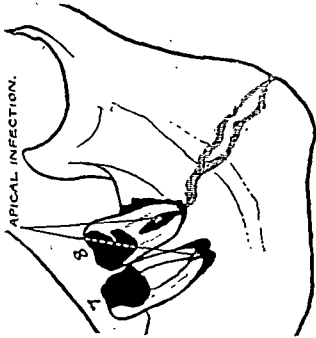
fracture was badly compounded into the mouth but none of the teeth were loose. Only a few teeth were present in the maxilla and a splint was cemented to these, the lower teeth being wired to it for five weeks. Three weeks later the 85 $\frac{1}{2}$ were removed as it was thought they might be a potential danger, but union was unaffected by the presence of the others involved in the fracture (radiogram 36 B). At first sight this case seems to bear out the advisability of retaining teeth, but I think the successful result should partly be attributed to removal of the 85 $\frac{1}{2}$ and that the time of extraction, as in the next case, was a very important factor. The major problem in treating fractured jaws is not the method of immobilization so much as prevention of infection or how to deal with it when it has developed. Considering the type of fracture in both these cases, it is surprising that there was but little infection and union took place within a reasonable time. Case 37, a woman of 53, was an air-raid casualty with a comminuted fracture of the right mandible (radiogram 37 A) and lacerations of the face: when she was transferred to the Centre a week after the injury the wounds had been excised and sutured, the one relating to the fracture drained, and the jaw immobilized by eyelet wiring. Five days after admission there was little evidence of infection but the 8543 $\frac{1}{2}$ were extracted on principle, in case they should give rise to trouble, and radiograms 37 B and C, taken three weeks and four months later, showed a good result. This case might be cited in favour of extracting teeth in like circumstances but it should be noted that this was not done until twelve days after the injury and that the time factor referred to in connexion with Case 36 was partly responsible for the good result.

On reviewing over 300 cases for this paper, I have tried to show that it is unwise to dogmatize on the two subjects under discussion, and to plead for an open mind. In picking out cases to illustrate the need for a less rigid viewpoint in treatment I have made no attempt to cover the subject but simply to stress some of the more important considerations. The two main dangers likely to result from extraction of teeth are trauma and infection, but with bone, infection is our chief concern. In removing either it is most important to choose exactly the right time. With bone it has always been a fixed principle that this should be done either immediately after the injury or not until it has separated. The problem is less easy to solve in the case of teeth: in the early stages of a fracture the trauma caused by extraction is often a grave danger and later the best time depends upon the degree of infection present. I think it can be said of teeth in the line of fracture and comminuted bone in fractured jaws, that while we may hold a general bias in favour of one course or the other, we cannot be oblivious to the value of each in appropriate cases.

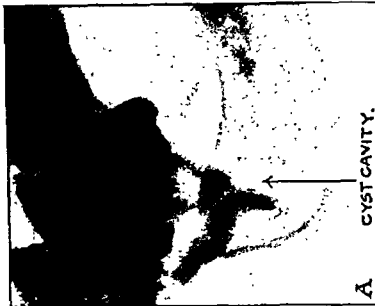
Some of these cases have been treated at the Maxillo-Facial Hospital at East Grinstead and for collaboration in the treatment I am indebted to my surgical and dental colleagues. I acknowledge permission from the Dental Surgeons at the Middlesex Hospital to include some of their cases, and to Mr. S. A. Riddett for one of his. My thanks are due to Professor Russ and the Medical Research Council for the invaluable help of Miss Clephan in reviewing the series of cases and the preparation and illustration of this paper.

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CASE 10 (Mr. A. L. Packham)

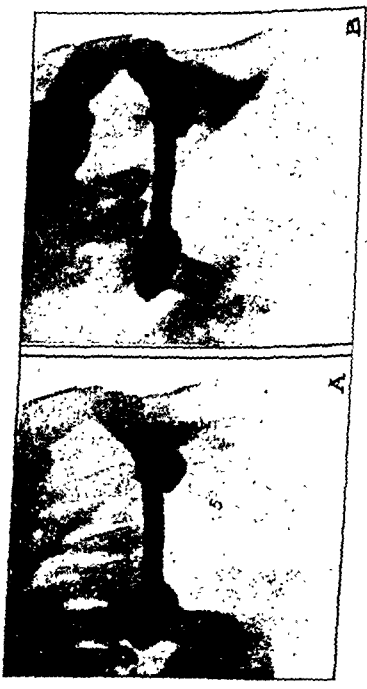




CASE 6



CASE 7



CASE 8



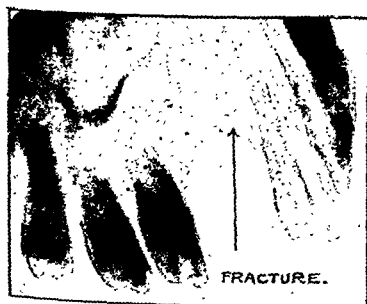
CASE 11 (Mr. H. W. Brown)



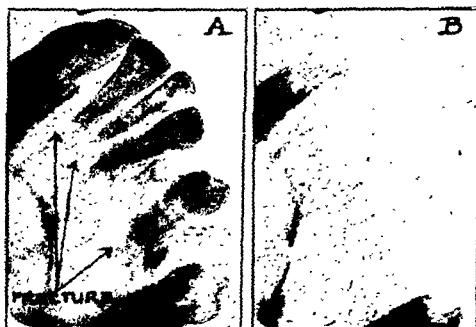
CASE 18



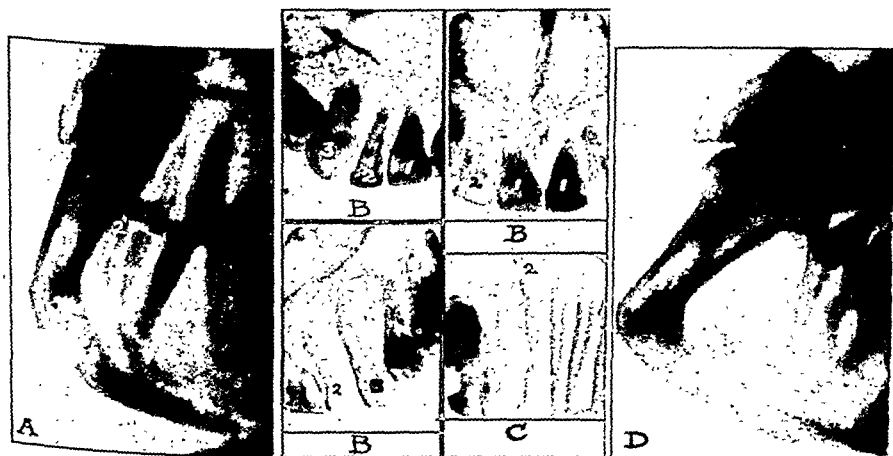
CASE 19



CASE 20



CASE 21



CASE 22



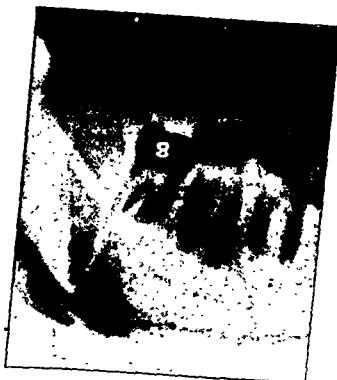
CASE 13



CASE 14



CASE 15 (Mr. A. T. Pitts)



CASE 16



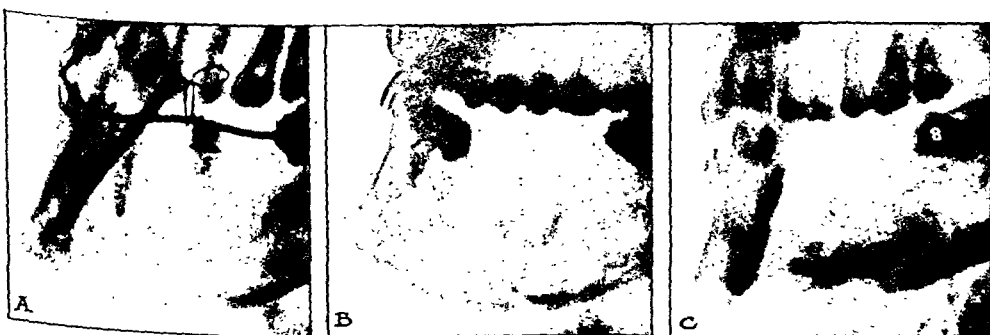
CASE 17 (Mr. Warwick James)



CASE 23



CASE 29



CASE 30



CASE 23



CASE 24



CASE 25



A



B



C

CASE 26



A



B



C



D

CASE 27



CASE 35

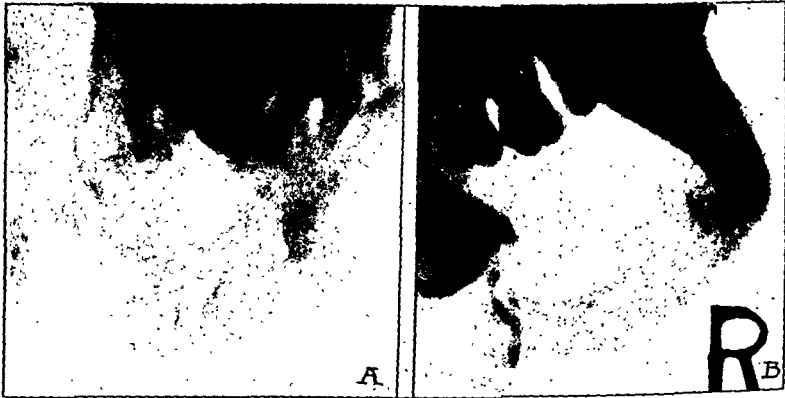


CASE 34



CASE 37

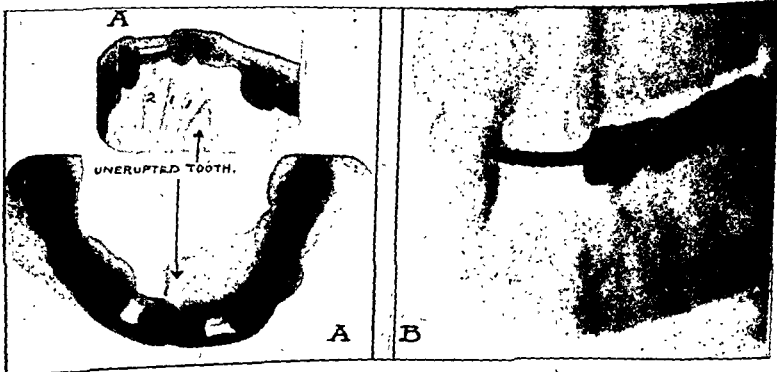




CASE 31



CASE 32



CASE 33

Section for the Study of Disease in Children

President—A. G. MAITLAND-JONES, O.B.E., M.D.

[March 28, 1942]

MEETING AT CELL BARNES HOSPITAL (ST. BARTHOLOMEW'S), ST. ALBANS

The Place of Child Guidance in a Pædiatric Unit. [Summary]

By J. LOVEL BARNES, M.R.C.S.

At Cell Barnes Hospital the Child Guidance Clinic is run in close co-operation with the Children's Department. The results have been very successful. The procedure has been for the psychiatrist to see cases that have already had a full physical investigation. The majority of children who have been investigated from this point of view have been suffering from asthma, enuresis, doubtful chorea, nervous tics and behaviour disturbances. The result of treatment in these cases has been very successful, particularly in those children suffering from asthma, in which a definite emotional factor was established.

An attempt has been made to assess the extent of the emotional factors, at the same time bearing in mind that in a certain percentage of them the physical factor may also be present. Where emotional factors have been detected, the children have either been treated in the out-patients' department at St. Bartholomew's Hospital, if they live in the London area, or in the Hertfordshire Child Guidance Clinic at Hill End Hospital.

A simplification of the methods of diagnosis and treatment has been attempted. Actually no very elaborate play material has been used and I have, in the case of the children treated at St. Bartholomew's Hospital, been working entirely on my own without even the assistance of a social worker. I stress this point in order that others who might be tempted to follow this experiment should not be dissuaded from so doing by the thought that a team of workers and a great deal of elaborate material must necessarily be forthcoming.

One of the simplest and most effective ways of getting a child to express ideas about which it is inarticulate, is to get that child to draw and fortunately this is a method which involves a minimum of material. Children's drawings were shown, illustrating this point and also progress in treatment.

The Surgical Treatment of Congenital Defects. [Summary]

By RAINSFORD MOWLEM, F.R.C.S.

The plastic surgeon's chief contact with pædiatrics is in the surgery of congenital defects. Not all require operations during early infancy but, as those who do are frequently in poor general condition, two main provisions are essential: the assistance of a skilled pædiatrician both before and after operation, and a surgical team accustomed to meet and deal with the special surgical and anæsthetic conditions of early infancy.

The optimum time for operation.—Most of the rarer congenital defects can safely be left to the later years. *Hypospadias* and *epispadias* do not usually call for intervention until about the age of 6 years. In the former the reason for delay is the necessity of constructing the urethra to ensure both rectal and sexual potency. Usually only minor degrees of *epispadias* lend themselves to plastic repair, the alternative being ureteric transplantation. *Absence or atresia of the vagina* may also be detected in childhood but is not operated upon until puberty. *Syndactyly* and *polydactyly* are usually operated on between the second and fourth year, unless further delay is necessary for identification of the primary centres of ossification.

Hæmangioma.—The small raised cavernous type with capillary elements on the surface, which appear after birth, are liable to spontaneous cure. The deep-seated truly cavernous type and the well-defined surface capillary type do not show such constant behaviour. The latter may improve, the former probably will not. The optimum treatment is either radium or X-ray, but the initial radio-sensitivity of these growths decreases fairly rapidly after the first year. It may not therefore be justifiable to await spontaneous cure when the treatment is so simple.

Cleft lips and palates.—In the repair of these conditions the primary necessity is the



A



A



B



B



Section for the Study of Disease in Children

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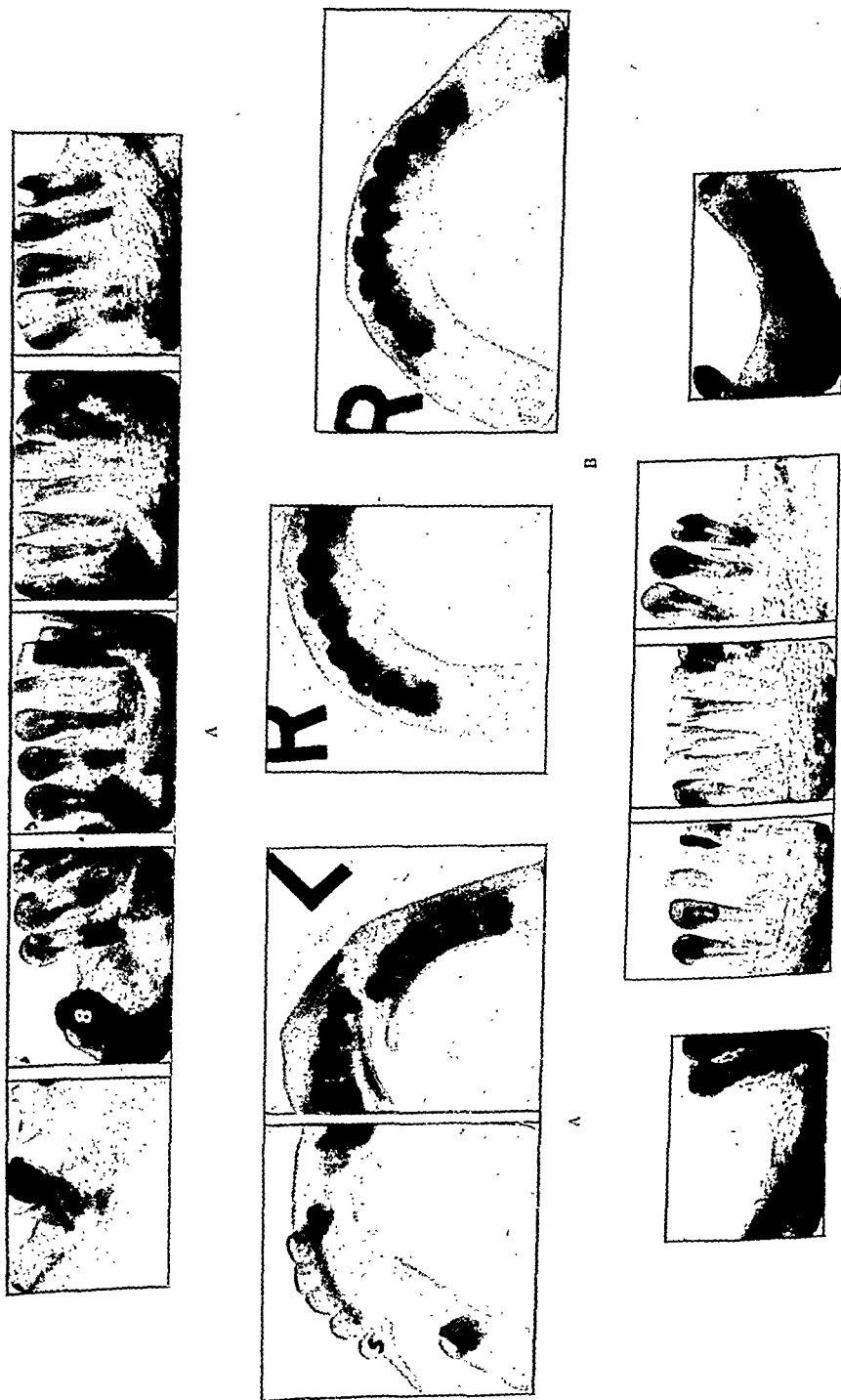
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Hæmangioma.—The small raised cavernous type with capillary elements on the surface, which appear after birth, are liable to spontaneous cure. The deep-seated truly cavernous type and the well-defined surface capillary type do not show such constant behaviour. The latter may improve, the former probably will not. The optimum treatment is either radium or X-ray, but the initial radio-sensitivity of these growths decreases fairly rapidly after the first year. It may not therefore be justifiable to await spontaneous cure when the treatment is so simple.

Cleft lips and palates.—In the repair of these conditions the primary necessity is the



CASE 36 (Mr. S. A. Riddell)

Section for the Study of Disease in Children

President—A. G. MAITLAND-JONES, O.B.E., M.D.

[March 28, 1942]

MEETING AT CELL BARNES HOSPITAL (ST. BARTHOLOMEW'S), ST. ALBANS

The Place of Child Guidance in a Pædiatric Unit. [Summary]

By J. LOVEL BARNES, M.R.C.S.

At Cell Barnes Hospital the Child Guidance Clinic is run in close co-operation with the Children's Department. The results have been very successful. The procedure has been for the psychiatrist to see cases that have already had a full physical investigation. The majority of children who have been investigated from this point of view have been suffering from asthma, enuresis, doubtful chorea, nervous tics and behaviour disturbances. The result of treatment in these cases has been very successful, particularly in those children suffering from asthma, in which a definite emotional factor was established.

An attempt has been made to assess the extent of the emotional factors, at the same time bearing in mind that in a certain percentage of them the physical factor may also be present. Where emotional factors have been detected, the children have either been treated in the out-patients' department at St. Bartholomew's Hospital, if they live in the London area, or in the Hertfordshire Child Guidance Clinic at Hill End Hospital.

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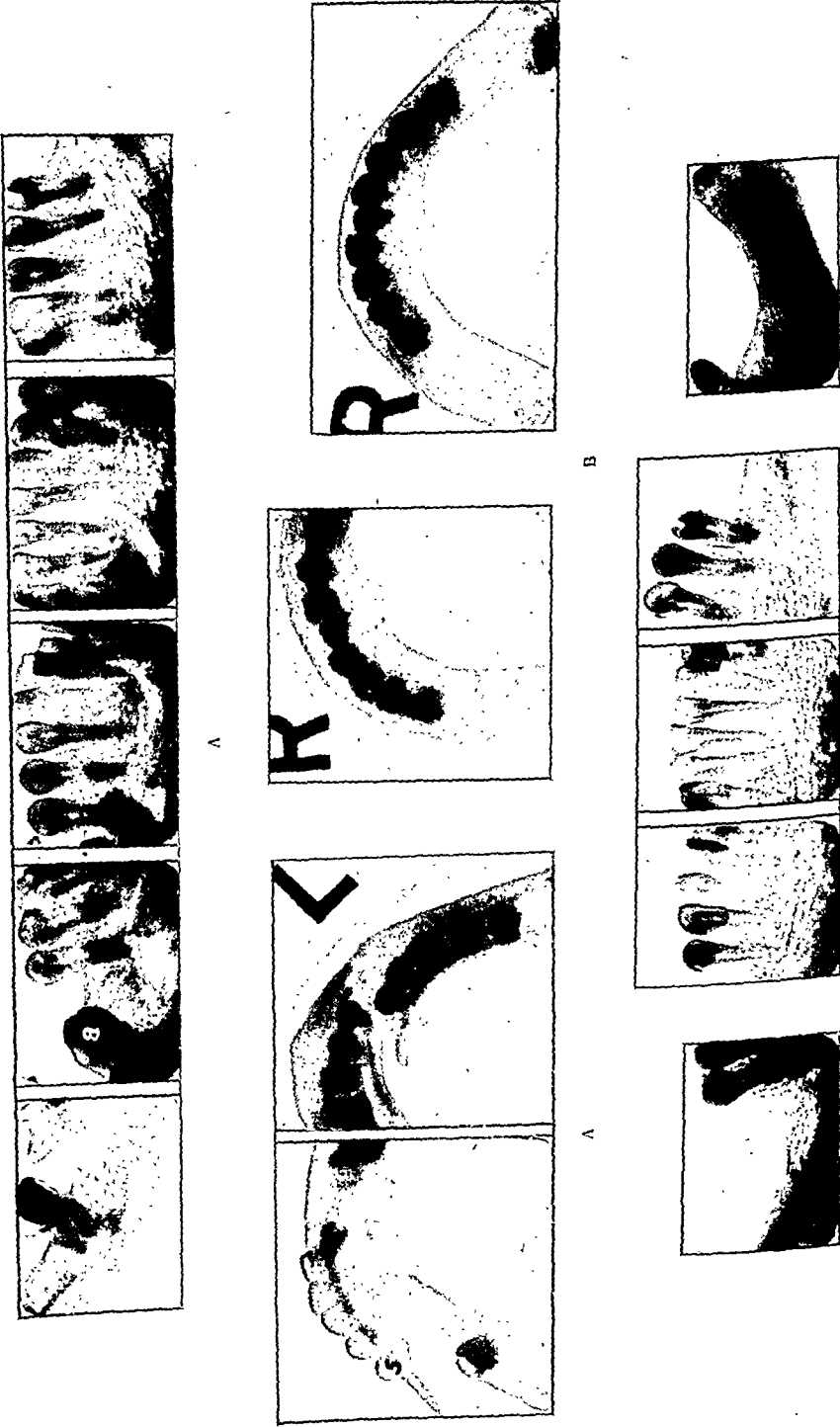
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provision of a normal speech mechanism. Repair after "palatal speech" has been established is not sufficient to restore normal enunciation and a long period of speech training will be required. In my opinion this disadvantage can be eliminated by operating sufficiently early to enable the child to develop spontaneously good speech and sufficiently late to be safe. My own practice is to operate at the earliest possible time. Although the first forty-eight hours is said to be the best, this is rarely possible. If this opportunity is missed it is unwise to give an anæsthetic until nutritional advancement is well established. My own rule is to wait until the child weighs at least 10 lb. The repair of a complete cleft involving both lip and palate falls into two stages with an interval of three months between them, so that if it is hoped to complete surgical procedures before the end of the first year an early start is essential. With cleft of the lip only there is less urgency as there is no associated functional disability.

The disadvantages of such early intervention are chiefly increased technical difficulties. These can be overcome by the team which is accustomed to work on such small children but the team must be constant. In my own experience the mortality rate for a large series of cases is considerably less than 1%. In operations on isolated children in other parts of the country without the use of the usual team the mortality rate rose on one occasion to 60%.

The complications of early operation are chiefly respiratory. The great majority of small children show a reactionary temperature of as much as 100° F. on the day following operation. A few develop bronchitis, apparently due to nasal obstruction temporarily imposed by an adequate operation. Pneumonia is very rare. Both these complications can be reduced by confining the operations to the spring and summer months, necessitating postponement in some cases to a later period than would otherwise be justifiable. The risk of post-operative nasal infection which jeopardizes repair has been decreased by sulphanilamide which may be insufflated into the nose. Lastly a rare complication, not often stressed in this country, is hyperthermia pallida. I have met with six instances, the first fatal. The child's temperature rises extremely high in the first few hours after operation. It is pale, has a respiration rate between 50 and 90 per minute, an uncountable pulse, and if untreated soon dies from exhaustion. In the fatal case no significant post-mortem findings were discovered. Though atropine is assumed to have a bearing on this condition, as much as $\frac{1}{80}$ of a grain has been given to one case without aggravating it. That it is due to so overloading the child in the theatre that its heat exchange mechanism is completely disorganized, is borne out by the lessened incidence since mackintoshes, pneumonia jackets and such impedimenta have been discarded. The treatment is to lower the temperature to about 97° F. by rectal wash-outs of ice water and to maintain it at that level for at least one hour. The heat-controlling centre apparently recovers and though there is usually a small spike of temperature on the next two or three days, the child's general condition remains good.

The difficulties encountered in operating upon small children are so great that success depends on close co-operation by the paediatrician, in preparing the child for operation and advising treatment during convalescence, and the surgical team equipped to deal with structures which are minute and an organism which is intolerant of prolonged insult. Such a combination achieves good results and reduces the mortality rate to the minimum.

Threadworms in Children in England. [Summary]

By MAY R. YOUNG, B.Sc.

THREADWORM, oxyuris or *Enterobius vermicularis* has man as its only natural host and is found in persons of both low and high social status.

Fifty (42%) of 119 children in Cell Barnes Hospital (St. Bartholomew's) have proved positive for threadworms by the examination of three cellophane anal swabs per child, whilst two swabs per child revealed infestation in 22 (55%) of 40 resident London nursery school children.

Necropsy material shows adult threadworms to be twice as frequent in the large as in the small intestine (Jones, 1941). The mature female passes out of the anus during relaxation and sleep to lay between 5,000 and 15,000 ova. By causing pruritus the eggs get on to clothing and fingers and if swallowed bring about reinfestation. Each ovum contains an infective larva which hatches out in the duodenum. After two months it reaches maturity, mates and passes down the intestine where it becomes attached to the mucosa by three labia at the oral end. Some may enter the appendix. Gordon (1933) on examination of over 26,000 appendices concluded that the threadworm is a negligible cause of appendicitis; whereas Wax and Cooper (1941) found *Enterobius vermicularis* in 8 out of 1,016 operative cases of appendicitis, 6 of these showing inflammatory reaction.

Diagnosis.—Threadworms may be seen either migrating or in the stool or microscopic ova found in the stool or on cellophane anal swabs. Considering the high incidence in children the finding of worms is rare; whilst 5% of the 119 children showed ova in the stools (three per child having been examined by zinc sulphate concentration flotation method) 42% of the 119 were positive by the examination of three cellophane anal swabs per child. This swab, designed by Hall in 1937, consists of a square inch of cellophane folded over the rounded end of a solid glass rod and held in place with a rubber band. Of the 119 children in St. Bartholomew's Hospital one swab per child revealed 26 positives, two swabs 40 and three swabs 50. American workers have shown that seven swabs per individual taken on alternate days reveal 99% of the total number of persons having threadworm infestation.

To compare the incidences in the two groups of children the examination of two swabs per child show 33.6% of the hospital children and 55% of the resident nursery school children to be positive. American workers have shown that treatment must accompany strict hygienic measures to eliminate threadworm infestation.

My acknowledgment and thanks are due to Professor R. T. Leiper of the London School of Hygiene and Tropical Medicine and to the Medical Research Council for whom the investigation into intestinal parasites was commenced in 1939.

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Congenital Dermal Sinus.—J. E. A. O'CONNELL, F.R.C.S.

Female, 4 years old. She was shown after excision of a congenital dermal sinus in the lumbar region associated with recurrent attacks of meningitis.

History.—When 8 months old the child was admitted to hospital with meningitis which responded to sulphanilamide. At the age of 18 months signs of meningitis recurred. Bilateral myringotomy produced no improvement and following admission to St. Bartholomew's Hospital a port-wine stain was found in the lumbosacral region with a small central pit from which sebaceous material could be expressed (fig.). The meningeal



Port-wine stain over congenital dermal sinus.

signs continued and the left leg was paralysed. Attempts at lumbar puncture produced a purulent fluid containing epithelial squames, polymorphonuclear leucocytes and coliform organisms. Cisternal puncture showed a turbid fluid containing the same organisms.

At operation the dermal sinus was traced through a defect in the neural arch of the fourth lumbar vertebra communicating with a large abscess within the dura and lying among the roots of the cauda equina. The track and part of the abscess wall were excised and drainage instituted. The wound healed after several months and now two and a half years later the child is asymptomatic.

Sulphæmoglobin(cyth)æmia and Anæmia Neonatorum.—A. WHITE FRANKLIN, M.B.

J. T., born 20.2.42, male.

First child. Forceps delivery at term resulted in a left facial palsy. Birth-weight 7 lb. 2 oz. Breathing was bubbly for two days but, on breast and bottle feeding, progress was normal until 27.2.42 when he vomited bright green curds during the night. Next morning skin was green and lips blue. Bowels were open normally on 25.2.42 but not again until on admission to hospital (28.2.42). At this time he looked ill, skin was of green hue, sclerotics not being discoloured, and lips and finger nails were a dark blue. Liver and spleen were not enlarged. No drugs had been given to mother or baby. Eight dark green stools were passed during the night and vomiting continued sporadically, sometimes bright green, but pigment not examined. Blood (1.3.42) appeared dark brown and on 2.3.42 sulphæmoglobin was found in a concentration of about 8%. It was intracorpuseular and Schumm's test on the plasma was negative. On 6.3.42 the skin was less green and the cyanosis was less, but mucous membranes were pale and by 10.3.42 he appeared markedly anæmic, the skin being tinged faintly green.

Urine contained much urobilin only. Stool cultures grew no abnormal bacteria.

Serum bilirubin was increased on 6.3.42. Citrated blood transfusions (*) were given 10.3.42: 70 c.c.; 11.3.42: 40 c.c. [5.4.42: 50 c.c.; 19.4.42: 50 c.c.]

| | | | | | | | | | | | | |
|---------------------------|------|-----|------|---|---------|------|------|------|----|--------|-----------|------|
| 1942, March | 1 | 2 | 3 | 6 | 9 | 10* | 11* | 11 | 17 | 20 | 23 Apr 5* | 13 |
| Hb. % | 112 | 100 | 80 | — | 40 | 44 | 64 | 94 | 92 | — | 82 | 80 |
| R.B.C. millions per c.mm. | 4.34 | — | 3.79 | — | 1.31 | 2.10 | 3.73 | 4.10 | — | — | 4.18 | 4.28 |
| Blood sulphæmoglobin | ? | 8% | — | ? | 8% less | — | — | — | — | absent | — | — |

Differential films were normal throughout and at no time were nucleated red cells seen. W.R. (23.3.42) negative.

In this case intracorpuseular sulphæmoglobin apparently developed spontaneously on the seventh day of life and was followed by an acute anæmia which responded to intravenous citrated blood. At no time was there clinical jaundice or enlargement of spleen or liver, nor was there ever erythroblastosis.

Postscript.—The baby failed to thrive, though neither anæmia nor sulphæmoglobinæmia recurred. Breast milk, high carbohydrate and high protein feeding were unsuccessful, maximum weight (12.3.42) being 6 lb. 5 oz. and the last weight (5.4.42) 5 lb. 10½ oz. Stools were frequent (seven to eight a day) but not watery and there was no dehydration. Death occurred at 7 weeks (23.4.42). Autopsy (Dr. Joan Ross) showed an emaciated infant with bronchopneumonia and multiple lung abscesses, and suppuration at sites of transfusions.

Bone-marrow showed normal erythro- and myelo-poiesis with a high proportion of megalokaryocytes. Liver and spleen contained masses of iron-containing pigment. Suprarenal and other endocrine glands were normal (pancreas not examined).

[April 25, 1942]

MEETING AT WINDSOR EMERGENCY HOSPITAL

Actinomycosis.—CHARLES PINCKNEY, F.R.C.P.

J. P., male, aged 12.

In September 1941 while evacuated to Wales noticed lump on left side of neck.

December 1, 1941: Came under observation. A cold abscess was observed over the middle third of the left sternomastoid.

December 5: Abscess incised. The pus smear showed streptothrix of the actinomycosis group with mycelia.

Given a course of M & B 693. Total dose 16 grm.

December 28: Potassium iodide commenced: dose gradually increased till a daily dose of 90 gr. was taken.

December 30: Further abscess over the lower third of the left sternomastoid muscle incised.

January 9, 1942: Another small abscess below previous one incised.

April 7, 1942: Discharged. Scars of three abscesses firmly healed, no underlying induration. Still continuing with potassium iodide 90 gr. daily.

Three Consecutive Cases of Pink Disease Cured with Parenteral Liver Therapy.—CHARLES PINCKNEY, F.R.C.P.

(1) C. D., female, aged 10 months.

Came under observation December 12, 1941. Birth-weight 7 lb. 4½ oz. Present weight 17 lb. 4 oz. Breast fed till 8 months, mixed diet since. When aged 8 months became very miserable; loss of appetite, hands and feet red and peeling.

On examination.—Very miserable child with marked photophobia. Hands and feet "pink" and desquamating. Sweating over trunk with rash present. Tachycardia. Musculature flabby.

Given hepatex 1 c.c. twice weekly for 8 doses.

Discharged January 16. Weight 18 lb. 3 oz. Cheerful, no photophobia, no tachycardia, hands and feet normal colour.

(2) S. B., female, aged 9 months.

Came under observation December 18, 1941. Present weight 17 lb. 9 oz. Birth-weight 5 lb. 9 oz. Breast fed till December 4, mixed feeding since. When aged 8 months became miserable; lost appetite, hands and feet red.

On examination.—Very miserable child, photophobia, hands and feet "pink" and desquamating. Sweating over trunk and rash present. Tachycardia. Musculature flabby.

Treated at first with vitamin B by mouth with no improvement.

January 15, 1942: Given hepatex—1 c.c. twice weekly for 8 doses as an out-patient.

April 16, 1942: Weight 18 lb. 12 oz. Cheerful, no photophobia, skin dry and smooth, hands and feet normal colour.

(3) J. F., female, aged 12 months.

Came under observation April 1, 1942. Present weight 17 lb. 3 oz. Birth-weight 7 lb. 14 oz. Still being breast fed, but mixed feeding started as well, at 6 months.

When aged 11 months became miserable; lost appetite, goes to sleep head buried in pillow, hands and feet red.

On examination.—Miserable child, photophobia present, hands and feet "pink" No rash. Tachycardia. Musculature flabby.

Given hepatex 1 c.c. twice weekly for 8 doses.

Discharged June 1, 1942, weight 19 lb. 1 oz., cheerful, no photophobia, no tachycardia, hands and feet normal colour.

Diabetes Insipidus Treated by Slowly Acting Pituitary Preparations.—DONALD COURT, M.B., M.R.C.P.

This boy was first presented to this Section in April 1939 (Court and Taylor). He remains a case of idiopathic diabetes insipidus.

Now aged 7 years. Weight 45 lb. General health good. During a recent period in which all treatment was stopped, the daily fluid intake quickly reached 200 fluid ounces. For the past three years he has been treated with pituitary emulsion and, more recently, with pitressin tannate in oil. The former gave excellent control with $\frac{1}{2}$ c.c. dose, twice weekly. It was abandoned owing to the delayed formation of paraffinomata at the injection sites. This unfortunate sequel is mainly due to the beeswax present in the emulsion. Also, through "Evacuation", only intermittent supervision was possible and it was subsequently found that the injections had been given subcutaneously instead of intramuscularly as instructed.

For the past six months pitressin tannate in oil (Parke Davis & Co.), 1 c.c. twice weekly, has been used with very satisfactory results (Greene and January, 1940). This appears to produce no local or general side effects. The control achieved with these preparations is shown in the following table.

| Extract. Pit. Lig. (B.P.) | No. of days | No. of injections | Hours per injection | Av. daily intake Fl. oz. | Av. daily Output Fl. oz. |
|----------------------------|-------------|-------------------|---------------------|-----------------------------|-----------------------------|
| 0.25 c.c. and 0.5 c.c. ... | 14 | 28 | 12 | 64 | 65 |
| Pituitary emulsion ... | 24 | 8 | 72 | 61.5 | 49 |
| Pitressin tannate ... | 44 | 21 | 50 | 82.5 | 70.5 |

It is hoped to publish a more detailed account of slowly acting pituitary preparations in the near future.

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Hirschsprung's Disease. Treated by Spinal Anæsthesia.—DONALD COURT, M.B., M.R.C.P., and J. K. HASLER, M.B., D.A.

Male, aged 8. Healthy parents. First child. First seen 29.7.41. Constipated since birth. In the first two years would go four to five weeks without defæcation. Intermittent diarrhoea also occurred during the first three years, but not since. Liquid paraffin regularly and intermittent enemata had been the mode of treatment prior to admission. This had produced fairly frequent small motions but had had little effect on the abdominal enlargement. He had become rather shy and anxious owing to comments about the size of his abdomen from his schoolfellows.

On examination.—Weight 48 lb. Sallow complexion. Distended, tympanitic abdomen with visible coils of large gut. Circumference at the umbilicus, about 30 in. Diagnosis

confirmed by straight radiograms and barium enema. Cystography also revealed a large bladder. Response to enemata unsatisfactory.

30.8.41 and 9.9.41: Spinal anaesthesia.

11.9.41: Normal bowel action began and continued until his discharge ten days later.

20.4.42: Now having one or two motions daily, much larger than ever before. Circumference of abdomen $23\frac{1}{2}$ in. A further barium enema suggests a partial decrease in the size of the bowel. The enema was spontaneously and fully expelled.

Details of treatment.—In accordance with accepted views on colon enervation, it was decided to give sufficient spinal anaesthetic to paralyse the anterior nerve roots, up to and including the 6th dorsal segment. As the patient was considerably younger than those to whom spinal anaesthetics are usually given, no guide to exact dosage was available. Premedication, nembutal gr. iii, was given. The patient was placed in the left lateral position and 1 c.c. of heavy percaïne solution was injected after being mixed with an equal quantity of the patient's C.S.F. He was then turned on to his back and the head of the table was tilted slightly downwards. The patient was under continuous observation for an hour or more but very little visible peristalsis was seen and only a small amount of flatus was passed through a rectal tube. Priapism occurred and remained for several hours. The patient remained asleep throughout the proceedings but was "light" enough to react to a pin-prick above the level of anaesthesia. On examination he was found to be anaesthetic up to the intercostal space below the nipples. The posterior roots had therefore been paralysed up to the 5th dorsal segment. Under spinal anaesthesia the posterior roots are usually paralysed to about two segments higher than the anterior; we may therefore assume that in this case the anterior roots and their accompanying sympathetics had been paralysed up to the 7th segment.

Ten days later the procedure was repeated. This time 1.1 c.c. of heavy percaïne was used with a slightly greater downward tilt of the table. Priapism again occurred and vigorous peristalsis was seen all over the abdomen. This continued for more than an hour but without defecation. Anaesthesia was tested and the upper level was found at the intercostal space above the nipple, i.e. at the 3rd dorsal segment, therefore the sympathetics were affected to the level of the 5th dorsal segment.

Comment.—The beneficial effects of spinal anaesthesia in cases of true Hirschsprung's disease, were first described by Stabins, Morton and Scott (1935). This has subsequently been confirmed by Telford and Simmons (1939) and others. The improvement in general health, bowel function and radiographic changes in the calibre of the bowel in this boy, are in keeping with previous results and very gratifying. Effective premedication meant that he was unaware of the procedures involved.

Two further points require emphasis—the level to which anaesthesia was necessary and the delayed but satisfactory restoration of adequate bowel function in the absence of spontaneous defecation during the anaesthetic.

We wish merely to record our findings, leaving the explanation of this remarkable effect until more facts are available.

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Four Cases of Pulmonary Tuberculosis in Association with Meningitis.—NORAH SCHUSTER, M.B.

Four cases of tuberculous meningitis were all associated with pulmonary primary lesions and none of them showed any sign of ever having had an alimentary infection. The source of infection was clear in one instance only; in the others no tuberculous contact could be traced.

The primary focus was in all cases in or near the apex of the lungs and not in the more characteristic situation near the interlobar fissures.

Meningitis seemed, in 2 cases, to be a terminal event in hæmatogenous dissemination and in 2 cases to be due to breakdown of a pre-existing focus in the brain as described by Rich and others.

CASE I was a girl of 7 years who had always been delicate and had measles a year before. The primary complex was indicated by a small caseous focus in the left upper lobe and a large caseous bronchopulmonary gland. The most striking feature was extensive infection of the pulmonary vessels, many of which, even of large size, were filled with caseous material teeming with tubercle bacilli. There was intense military dissemination and ample infection of the blood from the pulmonary vessels to cause diffuse meningitis without postulating antecedent foci in the central nervous system.

CASE II was a girl of 5 years with symmetrical healing foci in both apices, indicated by encapsulation and calcification. Near the left one there was a small crop of recent tubercles

which had probably followed measles and whooping-cough three months before. There was no miliary spread and the only other tuberculous lesion was in the central nervous system. A tiny tubercle was demonstrated on the surface of the cerebral cortex and there was diffuse meningitis with caseation and partial organization. It is suggested that the latter was the result of rupture of a cerebral focus formed about the time of the primary complex, or at the time of exacerbation three months ago.

CASE III was a boy of 7 months with a history of bronchial catarrh. The main feature was a fairly large cavity in the right apex with tuberculous bronchopneumonia surrounding it. There was hæmatogenous infection of the lungs, spleen and central nervous system. The presence of tiny recent tubercles in the meningeal arterioles, the large quantity and lack of organization in the exudate makes it probable that the meningitis was the final culmination of blood infection.

CASE IV. A boy of 11, had small partially healed foci in both apices, numerous fibrohyaline tubercles on the pleura and enlarged caseous bronchopulmonary glands. The symptoms had been suggestive of cerebral tumour (3rd nerve palsy, &c.) and meningitis did not appear till the last days. It is highly probable that pre-existing foci were present in the brain though they were not seen at the post-mortem.

There had been no opportunity of determining whether any of these children had shown signs of antecedent cerebral lesions to account for the meningitis, and it is suggested that it might be instructive to make special inquiries into the history of similar patients. It might also be interesting, in view of MacGregor and Green's observations, to examine the cerebrospinal fluid for tubercle bacilli of all children with tuberculosis before meningitis sets in, and of all children with any form of cerebral irritation.

Reference.—MACGREGOR, A. R., and GREEN, C. A. (1937), *J. Path. and Bact.*, 45, 613.

[May 22, 1942]

JOINT MEETING WITH THE MATERNITY AND CHILD WELFARE GROUP OF THE SOCIETY OF MEDICAL OFFICERS OF HEALTH

DISCUSSION ON THE PREVENTION OF CHRONIC LUNG DISEASES IN CHILDHOOD

Dr. Alan Moncrieff proposed that by definition tuberculosis and asthma (primary allergic) be excluded from the discussion which should be limited to chronic and recurrent bronchitis, and the group called variously chronic pneumonia, pulmonary fibrosis, unresolved pneumonia, damaged lung—all tending to end in bronchiectasis. He first suggested certain general considerations concerned with infection and resistance. Under infection had to be considered that which came from outside (exogenous) and that which was present in the child's own respiratory tract. Environmental (exogenous) infection was essentially a public health problem. What was being done to reduce recurrent droplet infection in the home, in schools and in institutions? Was the public being sufficiently educated in the use of handkerchiefs and masks? Other problems in this group were those of ventilation, and sterilization of air by sprays and ultra-violet light (cf. shelter problems). Endogenous infection raised the whole question of chronic disease of the upper respiratory tract in childhood. Was anything being done to deal with "tonsils and adenoids" from the preventive aspect? Sinus infection was also of great importance and there was a large body of evidence that spilling over of pus from the nasal sinuses was responsible for recurrent and chronic lung infection.

Regarding the problem of resistance to infection, was there enough exact knowledge upon which action could be based? What was the exact relation between rickets and bronchitis? Was it a vitamin deficiency (A or D)? or due to carbohydrate excess with the production of a fat, flabby, "catarrhal" child? Was it possible to be more specific about the influence of nutrition in promoting resistance to disease? There appeared to be a vicious circle—chronic infection → under-nutrition → chronic infection.

Acute lung infections which did not clear up properly or which recurred were frequently followed by chronic lung infections. Hence certain aspects of acute lung infection had to be considered. First came the occurrence of bronchitis and pneumonia associated with measles and pertussis. The problem seemed to be how to postpone these infectious diseases until after the age of infancy, possibly until after 5 years. Public health measures included the use of pertussis vaccines on a wide scale—in day nurseries and welfare clinics—and the use of blood (? blood banks) for modifying measles. Adequate convalescence after measles and pertussis was also very important. Secondly there was the problem of sulphapyridine in the treatment of pneumonia. Was there an increase in

unresolved pneumonia since the introduction of this drug—especially since borderline cases of severe bronchopneumonia were now being kept alive? Was there anything to be done about this? Thirdly there was the question of collapse of lung. This was probably far commoner than was generally supposed and might be due to sticky sputum produced by the injudicious use of atropine and belladonna and too little fluid in acute lung conditions. Finally the speaker dealt with active measures in prevention of chronic lung diseases, from the clinical standpoint. Tuberculosis must be excluded actively because far too many cases of collapse of the lung were disastrously treated by the passive measures available for tuberculosis. Hence every case of chronic lung infection should have a tuberculin patch test, followed by X-ray examination, contrast bronchogram (iodized oil) and bronchoscopy in that order if the severer types of chronic lung disease were to be prevented. Other measures were investigation of sinus disease, the treatment of diseased tonsils and respiratory exercises after pneumonia.

Dr. C. Elaine Field: A small but important branch of chronic chest disease comprises collapse of the lung and its frequent sequela, bronchiectasis. Within the last two years at Great Ormond Street and University College Hospital I have seen 82 cases. Of these, 39 were of massive collapse only, and 43 showed bronchiectasis in addition, as proved by bronchogram.

Ætiology:—Of the 39 cases of collapse, 31 cases gave a history of pneumonia or whooping-cough and in 23 of these the onset of their symptoms could be directly attributed to one or other disease. In the 43 cases of bronchiectasis, 41 gave a history of whooping-cough or pneumonia, and in only two cases the onset of symptoms could not be attributed to either of these diseases. From these figures and others in the literature, it is justifiable to say that pneumonia and whooping-cough are the chief predisposing diseases of collapse and bronchiectasis in children.

The theory of collapse.—It is well recognized that a plug of mucus will cause bronchial obstruction and massive collapse, but what happens for patchy collapse to occur: that is, blockage of the smaller bronchi and bronchioles? Lee Lander and Davidson suggest that the mucus plug gets sucked into the finer bronchioles. Fig. 1A shows a plug of mucus completely obstructing the left main bronchus. Collapse of the lung occurs distal to the plug. Increased negative intrapleural pressure resulting from the collapse produces a sucking action in the bronchi drawing the mucus into the finer bronchioles (fig. 1B and C). This procedure one imagines occurring in whooping-cough and some cases of pneumonia.

Andras visualizes a different procedure occurring in pneumonia—a failure of absorption of the exudate. Fig. 2A represents diagrammatically a consolidated alveolus and bronchiole. Resolution occurs in the alveolus (fig. 2B) but not in the bronchiole. As a result air and exudate are absorbed (fig. 2C) with resultant collapse of the alveolus. Both these authors suggest that most cases of bronchiectasis are preceded by collapse. Now Hedblom proved that in cases of collapse of the lung there is an increased negative intrapleural pressure, on an average, twice the normal but it may be twenty times the normal. Fig. 2D represents a normal lung and fig. 2E the post-pneumonic condition. A consolidated plug remains in one of the bronchioles so that the alveoli supplied by that bronchiole collapse; the other bronchiole has become patent following complete resolution. The increased negative intrapleural pressure fails to expand the collapsed alveoli and the pull is therefore referred directly to the wall of the bronchiole causing it to dilate with each inspiration. This predisposes to stagnation of secretion and infection with weakening of the wall of the bronchiole and permanent dilatation. But the increased negative intrapleural pressure over the patent bronchiole causes emphysema, a common association with collapse.

PREVENTION

Pneumonia.—Sulphonamides when given in full dosage for an adequate length of time have, I think, resulted in fewer cases of unresolved pneumonia.

Whooping-cough.—In 1935 the L.C.C. Annual Report estimated that 44% of London children develop the disease before their 5th birthday. According to Maclean's figures from St. Mary's Clinic, 90% of these children could have been protected by vaccination.

Collapse of the lung.—If this be due to tenacious mucus, it seems inadvisable to give drugs such as belladonna which make the mucus more sticky and adherent; expectorants such as potassium iodide might be given a trial. In cases where collapse of the lung has occurred immediate bronchoscopy should be instituted and all mucus sucked out followed by daily breathing exercises. Of the 39 cases of collapse previously mentioned, 26 have cleared up; 14 of these cleared up within one month of bronchoscopic suction. If at the end of a week X-ray showed the collapse persisting, a second bronchoscopic suction was performed. Hart advocates repeating this as many as ten times. A certain

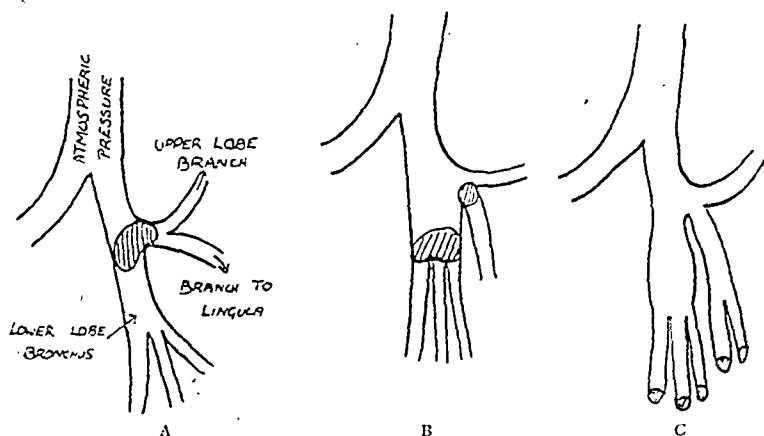
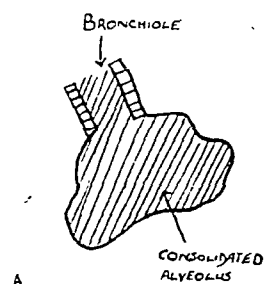


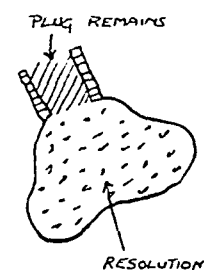
FIG. 1.—The mechanism of collapse. (copied by permission from "The Aetiology of Bronchiectasis," by F. P. Lee Lander and Maurice Davidson, *Brit. J. Radiol.* (1938), 11, 84.)

PNEUMONIC CONSOLIDATION



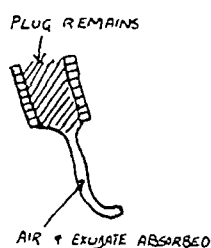
A.

IMPAIRED RESOLUTION

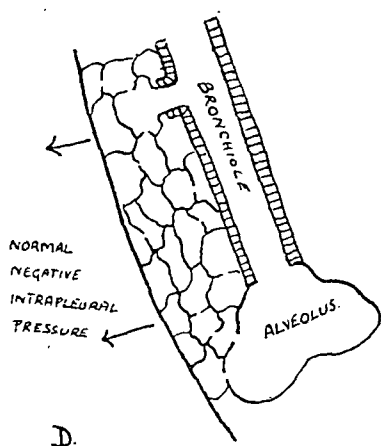


B.

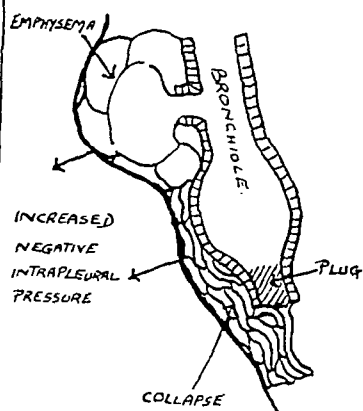
COLLAPSE.



C.



D.

NORMAL

E.

POST-PNEUMONIC

FIG. 2.—The mechanism of bronchiectasis.

proportion of these cases will clear up without bronchoscopic suction, the child coughing up the mucus; but it seems inadvisable to wait for this to happen as *time is vital*. Erb reports a case of a child who died four and a half weeks after the onset of whooping-cough. At autopsy the smaller bronchi were plugged with a stagnant exudate and microscopy showed invasion of the plug with young fibroblasts from the connective tissue of the bronchial wall—possibly already too late for bronchoscopic suction to be successful. Further microscopic work shows that granulation tissue develops in the interstitial framework of the lung and invades the collapsed alveoli, so that expansion cannot occur even if the obstruction is removed. How long, therefore, after the onset of collapse can successful expansion be obtained? This probably depends on the length of interval before infection occurs, and the type of infection; the more virulent, the more rapid the onset of permanent changes. *Massive or partial collapse should be treated as an acute emergency.*

Bronchiectasis.—Little can be done to prevent bronchiectasis developing in a collapsed lobe that has failed to expand. Postural drainage, breathing exercises, fresh air and treatment for infected nasal sinuses may retard the progress and inevitable result. Artificial pneumothorax may stop advance of the disease but it is not curative. The relationship of sinusitis to lung infections remains a vexed problem. In 31 cases of collapse of the lung only four had clear antra in the X-ray, and in 34 cases of bronchiectasis only three had clear antra. Treatment of sinusitis helps to prevent the progress of the lung infection and improves the general health of the child.

Bronchiectasis can develop with incredible speed in a young infant in the presence of a virulent infection. Erb reports a case of acute dilatation developing within ten days of the onset of pneumonia.

Lobectomy should be performed when possible, to prevent spill over of secretions into healthy parts of the lung, thus causing further collapse and bronchiectasis.

I would like to thank Dr. W. J. Pearson and Dr. W. G. Wyllie and the staff of Great Ormond Street and University College Hospitals for their help and co-operation.

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Dr. Nora I. Wattie: In Glasgow 3·4% of school entrants show evidence of chronic lung disease. The prevention of these chronic and often recurring disabilities is dependent upon the possibility of controlling the original causative infection which is pneumonia, either primary or as a complication of measles or whooping-cough.

With regard to measles, placental globulin has been successfully used in institutions in Glasgow to produce attenuated attacks. It might be possible to extend its use to home contacts, particularly under two years.

With regard to whooping-cough, the modern vaccines in adequate dosage are definitely of value, both in producing immunity and in attenuating the severity of the attack when infection has taken place. The practical difficulty with regard to more widespread use of pertussis vaccine to produce immunity is that mortality and lung complications are highest in the infant, and to affect these the vaccine must be given as early as 3 months of age. The anti-diphtheria campaign has got the idea of prophylaxis at 1 year into the minds of mothers, and the change to an earlier age with more chance of some reaction in the infant may be difficult. Combined immunization against both diphtheria and whooping-cough at 9 months-1 year should now be universally adopted. This combined immunization is insisted on as a condition of admission to war-time nurseries in Glasgow.

With regard to the pneumonias, the use of the sulphonamide preparation in adequate dosage has materially reduced the mortality rate and the number of cases with residual chronic infection. In the prevention of acute pneumonia in infants, however, education of the public on the dangers of droplet infection is important. Recent work carried out by Dr. Anderson of Knightswood Hospital, Glasgow, indicates that the type of pneumococcus present in pneumonia affecting infants under 1 year is that normally found in the upper respiratory tract. Housing and nutrition are also important aspects of the problem. Adequate provision of special convalescent facilities with skilled nursing is also urgently needed.

Section of Psychiatry

President—A. A. W. PETRIE, M.D., F.R.C.P.

[June 9, 1942]

DISCUSSION ON EFFECTS OF WAR-TIME INDUSTRIAL CONDITIONS ON MENTAL HEALTH

Dr. W. Blood: In the attempt to assess the state of the mental health of several thousand employees of both sexes working in large food factories, I have studied the figures relating to sickness absence for the years 1940 and 1941, the accident incidence for the same years, absenteeism, labour turnover and also everyday experience. The sickness records are based on the diagnosis as stated on the doctors' certificates and under the heading psychological causes are included nerves, nervous debility, neurosis, neurasthenia, anxiety, &c. Comparing the principal causes of sick absence in 1940 with 1941—the incidence of each illness being expressed as a percentage of the total illness—we get:

| 1940 | | 1941 | |
|-----------------------|------|-----------------------|------|
| Respiratory infection | 40% | Respiratory infection | 41% |
| Gastritis | 9.5% | Gastritis | 7.9% |
| Works accidents | 8.0% | Works accidents | 10% |
| Rheumatism | 6.7% | Rheumatism | 8% |
| Tonsillitis | 4.0% | Tonsillitis | 2.7% |
| Psychological | 2.7% | Psychological | 2.1% |

The particular points in these tables are that in 1940 2.7% of all sick leave was due to psychological illness, the comparable figure for 1941 was 2.1%. We are told, however, that a certain number of psychological illnesses masquerade under such labels as gastritis and rheumatism. The lost-time figure quoted is therefore almost certainly an understatement. The high figure in 1940 of 9.5% for gastritis has probably some psychological significance. The increase of works accidents in 1941, 10% as compared to 8% in 1940, has also possibly some psychological significance. It is not known how much, if any, of the gastritis (9.5% in 1940) and the rheumatism (8% in 1941) was really due to neurosis. With such knowledge one could judge the size of the problem much more clearly. If this problem is to be investigated it must be on cases of gastritis and rheumatism early, i.e. during the first two weeks of illness—if the results are to be valuable to industry—for this is the approximate duration of the vast majority of illnesses due to these complaints.

| | M. | F. |
|---------------------------------------------------------------------------------------------|----|------|
| From my 1941 records I find the ratio of male and female sick absence from all causes to be | 1 | 1.7 |
| From frank psychological causes | 1 | 4.0 |
| From gastritis | 1 | 1.2 |
| From rheumatism | 1 | 0.56 |

There is room for speculation here since if much psychological illness was masquerading under rheumatism and gastritis, one might reasonably expect the female part of the male-female ratio for these complaints to be higher.

In September 1940, when the blitz on London was at its height, from a group of factories employing over 2,000 men and women, only 22 were away ill with nerves, &c. Besides loss of time due to psychological illness, both obvious and obscure, to what extent is absenteeism from unknown causes due to mental ill-health, maladjustment or fatigue? I am sure from my own experience and from hearing the experiences of many other factory doctors that a fair proportion of absenteeism, otherwise unexplained, is due to mental ill-health (in its widest sense) or fatigue, and many cases could be quoted in support of this statement. When one considers the causes of extra strain thrown on the working population during the past two years, for example, the war, bombing, worry over relatives, children evacuated, the black-out, food difficulties, long hours, overtime and often a completely changed life as when a clerical worker or housewife goes into the factory to work, it seems obvious that some absenteeism must be due to mental ill-health or maladjustment or fatigue, often not recognized as such by the absentee. With appropriate organization in the factory, much can be done to smooth out individual psychological difficulties before they lead to sickness and absenteeism.

Accidents.—My figures for 1941 show an increase of 50 notifiable accidents over 1940. This increase of accidents is, I believe, common to industry. Much of the increase is due to inexperienced newcomers to industry—people who have not acquired the factory outlook nor the dexterity that is essential for some jobs. Furthermore the age of the factory entrant to-day is much in advance of that of the usual peace-time entrant, and these middle-aged people are harder to train. There is, however, reason to believe that some of the noted increase in accidents is due to maladjustment or fatigue, causing inattention or lack of co-ordination. One of the chief worries of the factory doctor is to see a man or woman, whom he knows to be fundamentally decent, failing by reason of the development of psychological symptoms to get back to work, after their accidental injury has completely recovered. Sometimes we are forced to witness psychological symptoms getting worse, although the patients are still attending hospital for their injuries. I should like to see a medical psychologist attached to every injury clinic, so that patients could be brought under psychological review at the earliest possible moment and those in need of treatment given it immediately.—instead, too frequently we find the trouble getting steadily worse and nobody bothers—it is nobody's business.

Mental ill-health, fatigue and maladjustment, as the cause of excessive labour turnover, inefficiency and unhappiness.—Before the introduction of the "essential work order" it was common experience that large numbers of workers were continually leaving one job to go to another—often so far as one could tell without good reason. Were they all seeking more pay, shorter hours, better conditions? I think not. Some I believe were fatigued, and others suffered from nervous strain—this expressed itself as a "fed-upness", an irritability and a desire for any kind of change. Much waste of time and loss of production was caused by this flitting from job to job.

What is the position to-day?—Most foremen and managers say that some of the workers are rather more "touchy" than they were pre-war. My own impression is that I meet more people who are tired and dissatisfied with their work. There are some who object to having their jobs changed, others who are always wanting a change and others who complain without just cause. There are to-day, I believe, a certain number of people (more than in pre-war days) suffering from a below-par mental state which is manifesting itself as a restlessness in some, an irritability or unhappiness or truculence in others, and if one recognizes the existence of this below-par state, many of the difficulties can be successfully dealt with. The doctor, the manager or the foreman must listen patiently to the workers' difficulties and grievances, real or imaginary. When they are real, help must be given, when they are imaginary, patient listening often effects the cure. There is need in industry to-day, as always, for patience, politeness and common sense. Sometimes it happens that a workman (or girl) who is sent to the doctor because he or she is "difficult", is physically ill. I can remember cases of tuberculosis, gastric and duodenal ulcers, who were first brought to notice by their difficult behaviour. To realize fully the effects of mental ill-health in industry, one must remember (a) the lost time due to frank psychological illness and absenteeism from underlying psychological causes, (b) the relation of mental ill-health and fatigue to accidents, both to the causation and to the prolongation of incapacity, (c) the prolongation of physical illness due to associated psychological factors, and finally (d) the inefficiency and waste of effort in having "a square peg in a round hole". Sometimes the cause or aggravation of mental ill-health lies in the works, where this is so it can be dealt with satisfactorily by a reasonable management. I allude to excessive hours, excessive overtime, under-pay, inequalities of pay for similar jobs, excessive night work, unhealthy or dangerous conditions of work and bad supervision. More often, however, mental ill-health emanates from sources outside of industry but many cases can be adjusted satisfactorily within industry.

What can be done to maintain mental health in the factory.—It is essential that wages, hours of work, security and conditions generally be satisfactory, and conform to the proved economic and physiological needs of the worker. If these "bread and butter" needs are satisfied I attach the greatest possible importance to (1) harmonious relationships within the works, and the full recognition of the worker as an individual, (2) to the development of the social group within departments, and affiliation to larger groups within the organization. In my own works we have been slowly evolving an organization, parts of which are still experimental, but which we hope will counteract some of the psychological difficulties previously experienced, and prevent others from developing. The organization consists of four main parts: (1) The staff record or engaging office; (2) the medical department; (3) welfare department; (4) the club. The applicant for employment is received courteously and particulars of his industrial experience, personal particulars, &c. are quickly

taken. He is then told about the job, the hours he will work, the wages he will receive and the holidays that he will be entitled to. He is then conducted to the medical department where he is examined to ascertain whether he is fit for the job (if not fit, the doctor offers suggestions for alternative work). The next move is to the welfare officer who informs him of the various welfare schemes, sports clubs, &c., also what is expected of him, e.g. punctuality, regularity, cleanliness and observance of the firm's rules. He is then told of what the firm will do for him in the way of sick pay, and various privileges, and it is impressed on him that if at some future date he has any kind of grievance he can see the welfare officer at any time, and if need be a director of the firm.

The applicant is then conducted to his department, and introduced to the superintendent or foreman and shown the job. Where training is necessary suitable arrangements are made. Before starting work the newcomer is introduced to one of the workers who shows the novice the ropes of his department and the cloakroom, canteens, lavatories, &c. Foremen and forewomen have special instructions to keep a friendly eye on the newcomer for the first week or two.

The follow-up.—During the first week or two the newcomer receives two or three visits from the welfare officer who originally interviewed him. This officer inquires how he is getting on with the job, &c., and also speaks to the foreman to get his opinion of the employee. Sometimes at this early stage it is obvious that the worker has been wrongly placed and transfers are arranged. I attach considerable importance to the follow-up, as a means of assisting the newcomer to find his feet; it also gives him a feeling that the firm is interested in his well-being. So much for the newcomer, but what of his future career? The welfare department, in close co-operation with the medical department, is responsible for the mental and physical health of the staff. Some of the methods adopted are: (a) Educational, lectures and discussions, in particular to superintendents and foremen and forewomen on such subjects as the Human Factor, Factory Hygiene, Accident Prevention and on the care of staff, with emphasis on the correct attitude to be adopted by supervision in dealing with staff problems; (b) music is relayed to most of our factories for periods of half an hour several times a day. Its value in allaying monotony is unquestionable, providing suitable music is chosen; (c) other duties of the welfare department are to give advice and assistance on any difficulties connected with work, financial troubles, home worries, love affairs, day nurseries, hospital savings associations, income tax, and, in conjunction with the medical department, convalescence, special diets and consultations with specialists are arranged where necessary, in full co-operation with the family doctor. There is no doubt that a well-run welfare department can assist in maintaining the mental health of the staff by relieving them (by advice and financial assistance when necessary) of troubles which, without help, might prove overpowering. Ultra-violet light in tonic doses has proved of considerable value in keeping night workers fit and cheerful.

Sports clubs and social activities play a large part in maintaining physical and mental health. They give opportunity for games in the fresh air, and for the formation of lasting friendships. In my own firm there are two full-time social organizers who arrange social functions, entertainments, sports and interdepartmental competitions.

Dr. Joan Harwood: I have been working in a factory in an area of Britain which is not purely industrial, and which for the first year of the war was not materially affected by the very great changes in industrial conditions which occurred in some parts of the country. Since then our staff in the factory has changed considerably: many have left, and some have been replaced by older workers on both the men's and the women's side. Anything like accurate analysis of sickness due to mental conditions is impossible.

At first thought, one might expect a large increase in the number of cases of mental disturbance, but this is offset by the fact that the war effort has provided large numbers of bored people with an interest, lonely people with companionship, and has relieved the economic anxieties of some, although it has increased those of others. Workers of the older generation particularly have been given an increased sense of personal value, and absorption in work gives people of all ages relief from the depression and tension resulting from separation from those they love.

The main factors which have a bearing on the mental health of the industrial worker are fatigue—in which I include boredom—anxiety and depression, and these are likely to be increased in war time. The effect of these naturally varies with the degree of exposure, and the particular chink in the individual's mental armour, and they would seem to be the chief exciting causes of conditions which appear on medical certificates as nervous debility, nervous exhaustion, nervous dyspepsia, and those two discussion-producing diagnoses, neurosis and neurasthenia. If we can do anything to mitigate these three elements in industry we shall have gone a long way towards forestalling trouble.

In the early part of the war, inadequate or hastily improvised methods of black-out were a source of depression. In some factories many windows were permanently blacked-out or bricked-up. The result was that, in the daytime, parts of the factory were gloomy, or at the best, people were working in artificial light all day. With improvement in methods of black-out and ventilation, at least one cause of depression was removed. Poor canteen and cloakroom arrangements, and dingy surroundings, have a similar effect. Women are depressed if they have to wear unsightly clothing, and this should be taken into account when protective clothing has to be provided. If they do not like the clothing, they will either refuse to wear it, or they will grumble and wear it in such a way that its purpose is defeated. Incidentally, it is probable that the morale of many women who have been out of work for years will be improved by the wearing of essential new clothes which they can now afford, being in a job. This will be more noticeable where the labour is drawn from what used to be known as distressed areas.

There are many factors which cause extra fatigue in war-time, and of these, long working hours and lack of sleep owing to air raids or alerts, are the most obvious. Long spells of night work are not tolerated well by women. In married women, or single ones with dependants, this is probably in great measure due to the home factor, which causes additional strain. There is a tendency to do too much housework, especially in the daytime after working a night shift. The inclination to worry about those at home, either children or elderly relatives, is increased during night work, especially when raids do occur.

Young girls, just starting on night and shift work, are bad offenders in the matter of sleep, as they quite understandably find it hard to give up frequent visits to the pictures and dance halls—in a way they are perhaps paying the price for the present-day standard of values, which has tended to make people more incapable of amusing themselves in creative recreation instead of passive entertainment. Incidentally, of course, women do not finish growth until they are about 23 years old, and are therefore up to that age least suitable for night work.

Dyspepsia is a common trouble on night and shift work, in both men and women. This is most noticeable in the maladjusted worker, when it may become a conscious or unconscious means of escape from having to do night work or shifts. Advice on the arrangements of the home routine may help the more normal worker. Single girls living alone often do not have time to shop for adequate or suitable food. They then become dyspeptic, run down or anemic, and tire easily.

Bad transport unfortunately still exists in many places, and is exasperating to the tired worker at the end of a heavy day or shift.

Repetitive work demanding skill and concentrated intelligent attention is fatiguing. A works manager of an aircraft factory told me that they had found that girls doing concentrated precision work at machines did it extremely well, and thoroughly enjoyed the work, but that at the end of a shift they were often in a state of tension. They were irritable and nervy, and quarrelled easily. Some burst into tears on the slightest provocation. The necessity for relaxation is obvious, particularly before the worker has a heavy meal. A drink immediately after work is helpful, and rhythmic exercise, such as walking or cycling home, acts as a sedative. I believe that if facilities for games, or keep-fit exercises, could be provided at factories or factory hostels, they would be of value. In arranging for the types of exercises to be used in such classes, the kind of work which is done in various parts of the factory should be borne in mind. For instance, monotonous and easy kinds of repetitive work might be relieved by vigorous exercises, but after work like the above the emphasis should be on relaxation. Slow rhythmic exercises, such as are found in Greek dancing, would be restful for these workers, especially if done to music.

Careful selection and placing of workers, from a medical as well as a psychological point of view, will cut down fatigue. A works doctor has an excellent opportunity of assessing the working value of an applicant at the initial examination. It is clear from the employment and medical history, the physique and mental attitude of the applicant, whether he or she is likely to show a record of bad timekeeping, or will not stand up to a job involving any particular strain. In workers of borderline physique, it may be advisable to consult the labour officer or manager; or get details of the medical history from the panel doctor in the case of an unwillingly conscripted worker.

In the factory with which I have been associated we have a psychologist, and tests have been designed to assess the aptitude of workers for various jobs. These tests follow the lines usually adopted by industrial psychologists. Sense of form, co-ordination, rhythm, mechanical aptitude, various types of memory, and other faculties can be measured, and the worker placed accordingly. The worker who will be most happy on simple repetitive work can be separated from those capable of, and requiring, more interesting work. The

clumsy worker, for instance, will not be tormented by having to do fine work requiring precise movements.

Even more essential is care in the choosing of executives. The petty tyrant with a lust for power, and the executive who shirks responsibility or lacks discipline, will obviously be a disruptive influence in the workroom. The one may at any time become entirely ruthless, and the other will lose the confidence and control of the workers. The ideal person is one who can lead without driving. In an old-established works the most satisfactory way is probably to choose from amongst known workers, using the psychological or some standard performance test as a balance.

Co-operation between the works doctor and personnel or welfare workers can be a vital link in dealing with people who are mentally upset or disturbed. The personnel worker is in a position to give the doctor valuable information concerning the worker, and can carry out suggestions for dealing with difficulties. Frequently their knowledge of individuals will help the doctor in handling the patient.

The menopausal woman, who constitutes quite a high proportion of the present industrial population, does at times present a difficult psychological problem. Often the executive woman has reached ripe experience when she is most valuable at her work, but she is less resilient than she was, and sometimes needs intervals for rest between spells of her work. In these days she often receives less tolerance and understanding from her colleagues than she should, at a time when she is often feeling that she is becoming a less charming and effective member of society. The average woman responds well to encouragement, and this, coupled with suitable treatment, will go a long way towards helping these cases.

Of the many minor, but nevertheless very potent, fears likely to arise in some factories in war-time, demotion or loss of job owing to the cutting down of work or re-organization is common. Older women as a rule dislike change of work, and anything which can be thought of to temper the wind will be of value. Younger ones mind this less, especially if they can be moved in a group with their friends; but they usually dislike being sent to work far away from their home towns, and illness is frequently a synonym for homesickness in girls with a small supply of backbone.

It has been suggested by some people that women will probably dislike work which produces destructive material. I do not think that the average young girl pictures the end-result of destruction, but the older worker, especially the older executive, often does. Curiously enough, working men very often dislike their womenfolk doing this type of work for this reason, but the sober necessity is philosophically realized by both.

Fear of danger is seldom present in the young worker, but parents are often afraid for them, and will try to prevent them going in for dangerous work. They sometimes succeed in instilling fear, in young girls especially, where it did not previously exist. The older worker, again from experience, has a more lively imagination and this is an advantage or disadvantage according to temperament. Thorough education and drilling in safety arrangements produce confidence, and morale is then high. This is well illustrated in the Royal Air Force. Higher pay for dangerous work is a help. People are more willing to bother with the necessity for taking precautions, if there is an additional financial incentive to do so. This is not merely a question of avarice: the higher rate of pay is a demonstration of the value and responsibility of the work performed, and raises the prestige of the worker both in the workroom and at home. This has also been proved in connexion with a type of war work which has a high dermatitis risk, and which requires almost niggling standards of cleanliness and care of the skin, if the workers are to be kept free from rash. Propaganda is essential, and the workers should be made to realize that their problem is the management's. Good pictorial posters with the minimum of reading matter are better than long notices. Meetings of workers' representatives, managerial staff, and the works' doctors, are helpful in spreading proper information and dispelling false rumours, and in securing the co-operation of the workers which is so essential.

Fear of any occupational disease can become a difficult labour problem if it is not handled well. Good facilities for rehabilitation of the sick or injured worker go far towards alleviating anxiety in industry, so that this too will play an important part in the fight against occupational risks.

Dr. H. M. Vernon (*in absentia* read by P. E. Vernon): In the last war the sickness and loss of efficiency experienced by munition workers was so considerable that Mr. Lloyd George, the Minister of Munitions, appointed a "Health of Munition Workers Committee" to investigate it. The Committee was appointed in September 1915, and it sat till the end of 1917. Special attention was paid to the health of the women, as they were much more adversely affected than the men. A total of

about 1,300 women working in eleven factories was examined during the first half of 1916, and a second set of similar size in the autumn of 1917. They were classified as regards health into three main groups, and it appeared that 58% of them were healthy, whilst 35% showed some signs of fatigue or ill-health, and 7% showed marked fatigue or ill-health. Many other women had dropped out altogether because of ill-health, and in consequence escaped examination. The most frequent defects noted in the women were probably of mental origin, for it was found that, on the average: 28.5% had frequent headaches; 20% were tired, nervous, or irritable; 23.5% had indigestion; 26% had disorders of menstruation; 20% had anæmia; 12.5% had rheumatism or muscular pains. No doubt many if not the majority of cases of indigestion were psychoneurotic in origin, and this may apply also to at least some of the menstrual and other disorders.

The fatigue of the women was shown indirectly by their accident rate. At a fuse factory of 10,000 workers I observed that the accidents treated at the ambulance room were nearly three times more numerous when a twelve-hour day was being worked than when a change over to a ten-hour day was made. During the twelve-hour day period some work was done on most Sundays, and the total hours came to about seventy-four per week, as compared with the sixty-three to fifty-five hours subsequently worked. The majority of the accidents were cuts to fingers and thumbs, but other types of accident such as muscular strains were equally affected by hours of work. The workers sometimes went to the ambulance room for a rest, and I found that in the twelve-hour period the women were treated for faintness nine times more frequently than the men, whilst they were given sal volatile as a restorative twenty-three times more frequently. When the hours of work were reduced to ten a day these cases greatly diminished, and the women were then treated only about three times more often than the men.

Observations made on the health of male munition workers during the last war showed very little evidence of mental effects. About 1,500 men, aged 41 and upwards, and 1,500 boys, aged 14 to 17, were medically examined, and of the men 17% were considered to be slightly below normal, whilst 5% were much below normal and 0.4% were in a bad state of health. The boys were much healthier than the men, the corresponding figures being 8% below normal, 0.9% much below, and 0.2% in bad health. These figures may to some extent reflect age differences, but it is likely that they also show the effects of working hours; for about 40% of the men, as contrasted with 2% of the boys were working over seventy hours a week. The men lost, on an average, eight days a year from sickness and accident, and the boys five days, but only 0.4 day of the men's time and 0.3 day of the boys' time were attributed to "nervous" causes. No doubt this apparent infrequency of mental ill-health was due largely to the neglect or ignorance of this aspect of industrial health which was usual at that time, but nevertheless it is in striking contrast to the observations made on women. The men were obviously much less fatigued by long hours of work than the women, and in the fuse factory investigation I found that their accidents during the twelve-hour day period were only slightly in excess of those incurred during the ten-hour day.

A somewhat similar difference between men and women has been observed during the present war. In Emergency Report No. 2 of the Industrial Health Research Board we are given some striking evidence of the effects on health of the strenuous production drive which occurred in munition factories immediately after the withdrawal of our troops from Dunkirk. At one factory, where the hours of work during June to August 1940 were from sixty-seven and a half to sixty and a half, the number of women absent because of nervous breakdowns and nervous debility increased from 11 per thousand per week during June to 18 per thousand in July and 23 per thousand in August. Hours of work were then reduced to fifty-seven a week, and the nervous cases fell at once to 14 per thousand and subsequently to 8 per thousand. During June to August the nervous cases were almost as numerous as those due to all other causes such as colds, influenza, gastric affections, and rheumatism. In contrast, at a factory where men were employed the nervous cases were only a fourth as numerous as those due to other causes, and in the months of June, July, and August, when the hours of work were seventy-one to sixty-six a week, they numbered only 4, 7 and 6 per thousand respectively. Subsequently they fell to 2 per thousand.

Section of Dermatology

President—H. C. SEMON, M.D.

[March 19, 1942]

Clinical Photography in Private Practice

By A. C. ROXBURGH, M.D., F.R.C.P.

THERE are various ways of taking clinical photographs in black and white. Either daylight, electric light (photoflood) or flashlight (photoflash bulb or flash powder) may be used, with either a half-plate camera, making contact prints, or a miniature camera, making enlargements up to half plate. Half-plate size is the most generally convenient size for the prints in either case. I always use a miniature camera and enlarge, the advantages being small size of apparatus, great depth of focus, allowing use of larger stop and therefore shorter exposure, and cheapness of materials. An even greater advantage perhaps is that it is easy to improve the composition of the picture when enlarging and also to darken or lighten parts which require correction by extra exposure or shading.

My difficulty is that I have no separate room available which can be kept always rigged. The apparatus must therefore be of the smallest and simplest so that it can be got out, arranged and the photograph taken during a half-hour consultation without delaying the next patient more than ten to fifteen minutes. As Twiston Davies has emphasized the only time to photograph an interesting patient is the first time one sees him, as a later appointment may not be kept or the condition may have changed.

Essential points are:

Lighting.—This should be arranged to show up raised lesions and skin texture in maximum relief, i.e. oblique lighting, not too diffused. Usually the beam of light should make an angle of about 45 degrees above and 45 degrees to one side of the line from patient to camera. A white towel hung on the shadow side of the patient just out of the field of view helps to reflect light into the shadows. I use either a photoflash bulb (Baby Sashalite) fired by dry battery or a photoflood lamp in reflector held in the hand and moved about until one is satisfied that the illumination and relief are as good as possible. If I am going to use a photoflash bulb I use an ordinary portable reading lamp first to find the best position for the light and then hold the flash in that position when taking the photograph. The light is usually held 2 to 3 ft. away from the patient.

Background and composition.—A plain background should be used. I use a plain bluish cloth screen over which I hang a dark blue rug if a darker background is required. All extraneous objects and bits of clothing should be carefully eliminated after study of the picture in the finder. This is not the place for a dissertation on the elements of pictorial composition but, although a record and not a picture is aimed at, good composition will make the photograph easier to look at and therefore more useful. An important point is that even in the case of a close-up view of a lesion enough of the patient should be included to make it obvious what part of the body is represented. In the case of extensive eruptions two photographs, one to show distribution and the other details of the lesion, should be taken.

Type of camera.—I use a Rolleiflex twin lens reflex taking a picture 2 by 2 in. If working at short range one must remember to allow for parallax, i.e. the finder lens is $1\frac{1}{4}$ in. above the taking lens. This camera allows of easy and accurate focusing and composition on a ground glass screen. One should always use a cable release for the shutter so as not to shake the camera and to allow of release while watching the patient. A lens hood should be used to keep glare out of the lens. "Proxar" supplementary lenses are slipped on for close work.

Film.—Orthochromatic film should be used, not panchromatic. The latter makes a red eruption look as light in colour as normal skin and therefore it hardly shows at all. If only panchromatic film is available a blue filter should be used. I now use Selochrome or Verichrome film, size 120. This gives twelve pictures 2 by 2 in. to each spool.

Exposure.—Using a Baby Sashalite flash bulb the stop required is from $f/4.5$ to $f/8$ according to depth of focus required and distance of light from patient. With a photoflood lamp generally about half a second at $f/5.6$ is correct.

Arrangement.—The camera rests on my desk, if necessary being raised on books. There is thus no risk of kicking away tripod legs. Hands to be photographed rest on desk on a piece of black velvet which is carried back over patient's chest, and head and shoulders if necessary, to block out extraneous objects. Legs and feet are photographed on a couch brought up near the desk. The face is taken by sitting the patient on a low stool near the desk. The trunk is taken with the patient on a chair near the desk.

Method of taking.—(1) Arrange patient and lighting and focus accurately. Select stop.

Using Photoflood

- (2) Set shutter to $\frac{1}{2}$ second exposure.
- (3) Leave room lighting on so long as none shines into the lens.
- (4) Hold light in best position with one hand.
- (5) Release shutter with other hand.

Using Flashlamp

- (2) Set shutter to "bulb" exposure.
- (3) Turn down main room lighting and leave only dim light in room.
- (4) Open lens with one hand, i.e. press release
- (5) Set off flash with other hand.
- (5a) Close lens, i.e. let go release.

(6) Change film.

(7) Repeat with slight variation of stop or lighting.

(8) Record name, age, and diseases of patient. Date, stop and exposure.

Development.—I use a fine grain developer, usually one containing "Meritol", and enlarge to half plate on glossy, contrasty paper such as Kodak Nikko.

[A number of photographs of various skin diseases, taken by the methods described, were then projected by epidiascope.]

As a matter of interest I am now going to show some colour photographs taken by Mr. F. G. Hennell by a new process of his own devising which produces colour prints of any required size, larger or smaller than the original and in any required number, but which are opaque and can only be projected by an epidiascope. These photographs are taken by flashlight in a one-shot, three-plate camera, and the colour separation negatives so produced are printed on to three stripping films. These films are separately toned by metallic salts and then squeezed down in register on any suitable paper, card or other base. Though none of these deals with dermatological subjects they do show the admirable results which can be obtained.

Discussion.—Dr. F. A. E. SILCOCK described and showed coloured photographs, sections and coloured photomicrographs of (1) Fox-Fordyce syndrome in a woman aged 29 years; (2) erythema elevatum diutinum on backs of both hands and wrist in a woman aged 30 years. Owing to present difficulties of bringing cases from a distance to this meeting he thought this method of demonstration might be very useful and convenient.

Dr. TWISTON DAVIES: How did Dr. Silcock take his colour photomicrographs and estimate the exposure required?

Dr. SILCOCK: The section is placed and focused under the microscope in the ordinary way, then the microscope with the slide in position is tilted into the horizontal position and the reflector adjusted to give good illumination. The miniature camera, a Zeiss Ikon, is brought up to the ocular of the microscope in the same horizontal plane till it just touches it firmly. All extraneous light is excluded by means of some suitable covering, e.g. a piece of dark cloth wrapped round the junction. Film used was Kodachrome A (for artificial light). One photoflood light was used and reflected as described above. The camera lens was set for infinity, the aperture used was $f/3.5$ and the exposure was one-fifth of a second. One-tenth of a second was also tried, under the same conditions, but was not sufficient. I hope to make further trials of this simple method of taking coloured photomicrographs, as it does not require any special apparatus.

[May 21, 1942]

Industrial Dermatitis

By SIBYL HORNER, M.B., D.P.H.

ALTHOUGH dermatitis in industry is not notifiable, the number of cases voluntarily reported to the Factory Department (now of the Ministry of Labour and National Service) has increased from 2,000 cases in 1938 to nearly 5,000 in 1940.

Dermatitis and ulceration of the skin produced by dust or liquids are conditions compensatable (by certificate from the Examining Surgeon) under the Workmen's Compensation Act. The number of such cases was 2,735 in 1938 and 6,196 in 1940. In the U.S.A. it is estimated that 65% of occupational diseases are industrial dermatoses. In one year 20,000 men lost an average of ten weeks' work through this cause. Preventive work in this country has made important advances of late years, but much remains to be done in the way of effective supervision in the factory. Dermatologists are, however, mainly concerned with recognition and treatment of industrial dermatitis.

Specialist treatment will in many cases reduce the period of absence from work and may possibly diminish the risk of recurrence.

Workers have, it should be remembered, a lurking fear that dermatitis is due to dirt and that it is contagious. The result is the free use of soap and water and antiseptics even when instructed to the contrary. This will delay a cure.

Recognition of industrial dermatitis lies in the clinical eye, in the wideness and accuracy of differential diagnosis, and especially in an accurate working history from the sufferer. If the cause does not appear to be occupational, household or avocational causes should be considered. The patient should be asked to give his own opinion as to its origin, or to demonstrate his movements at work. Friction, either of itself, or combined with an irritant is sometimes the clue.

The common causes of industrial dermatitis at the present time are oil, chemicals (including explosives), alkalis, solvents or degreasing agents. Cleansers, often alkaline or degreasing in character, are sometimes responsible for more dermatitis than the industrial materials handled. Dusts, sprays and vapours usually affect the face, exposed neck and flexures of the body before affecting the hands. Oil folliculitis is commonest on the forearms but by permeating through aprons, may produce a rash on the thighs. The hands are rarely affected and sore fingers in a machinist suggest friction in a bath of alkaline coolant. Some chemicals stain the skin, others cause excessive dryness and characteristic lesions on a broken surface, e.g. salt "holes", ulcers from bichromates and formaldehyde. Dermatitis frequently follows an injury (which may have become septic) or a burn.

Prevention of industrial dermatitis: Briefly the protective measures are: Selection, protection, inspection and cleanliness.

Selection: Choice of suitable personnel, to whom is explained the procedure of

Protection: In addition to efficient ventilation, splash guards and protective clothing, the exposed skin must be protected by a "barrier substance". A good barrier substance must be non-irritating and easily applied. It must be insoluble in the substance from which skin protection is required, and it should be easily removed after work. Where there is a danger of absorption through the skin, e.g. T.N.T., or aniline, barrier substances containing no fats should be used. No protection is complete without good facilities for washing with supervision to ensure the best use of these.

The use in industry of chlorinated naphthalene waxes produces a characteristic skin eruption, a non-infective folliculitis. This is well marked on the forearms in early cases, but is most typical on the face where it affects mainly the malar and mental regions. Pustulation may be present in late cases and occasionally there are general symptoms of malaise and depression with digestive disturbances. The skin affection seems to be due to the actual deposition on the skin of vapour from the heated wax rather than to internal absorption and subsequent excretion. In addition to this recognized wax rash, exposure to fumes during the heating of chlorinated naphthalene waxes has been followed by a few cases of serious liver damage some of which have had a fatal result.

The skin reaction to chromic acid and to the bichromates may not be the typical "chrome

ulceration" (which is notifiable), but dermatitis, indistinguishable from that of other irritants. Recently a number of such cases has occurred in anodizing, where only a weak solution of chromic acid is used by an electrolytic method. Here the two factors for resultant damage to the skin were present, i.e. trauma from unjigging articles coming from the acid bath, and contact with the residual irritant, leaking through faulty washers. Instead of "chrome ulceration" dermatitis was the result. This is worth bearing in mind where weak solutions of chromic acid or bichromates are encountered and there are opportunities for superficial damage to the skin.

A topical source of irritation of the skin and mucous membranes, by bichromates, probably intensified by ammonia, occurs at "chromating" baths, which are non-electrolytic but are heated to a high temperature with evolution of fumes which must be removed from workrooms if damage to personnel is to be avoided. The most common result at these baths is ulceration and perforation of the nasal septum, and the latter may occur after only a few weeks' exposure.

The effects, i.e. trophic changes preceded by cracking and dryness of the skin of the last phalanges of the fingers of beta radiation from the radioactive substances in the luminous compound used for painting dials and instruments should be looked for, as if this condition is detected in a luminizer there may be still sufficient time to prevent the more serious effects of gamma radiation. The typical skin changes, due to beta radiation, will not be confused with the drying effect of certain solvents used in luminizing paints.

There are several types of glue at present in use for aircraft. Some of these are acid in reaction while others are alkaline. Added hardeners for certain glues include alkalis and acids. All types of glue have been known to cause dermatitis, and the element of friction of the forearms with glue-contaminated benches and aprons is, I believe, a common factor for the production of the trauma which so often precedes dermatitis. It is, however, of the greatest importance that glue should not be allowed to harden on the skin. With the use of animal glues warts were not uncommon; again trauma, with the possibilities of infection is an aetiological factor.

Industrial methylated spirit is being supplanted to some extent by the use of methanol, methyl alcohol. The latter may be found to have an even more drying effect on the skin.

Hexamethylene tetramine, urotropine, which we have respected in small quantities as an accelerator in the rubber industry, may now have to be reckoned with in larger amounts in its newer uses.

Recently observed effects were considerable thickening and cracking of the skin with some loss of sensation in the finger tips. These changes were noticed chiefly on the palms of the hands where there was discoloration (tanning) of the skin. It is interesting to note that among a group of workers, a high percentage of whom showed some or all of these changes, it was the loss of sensation in the finger tips of which they complained. After the protective measures had been instituted good results were reported, with some relief from this distressing affection. Dermatologists can assist in the prevention of industrial dermatitis by teaching preventive methods both to their students and their industrial patients while their contribution to the national effort is evidenced by the good results of specialist treatment of this condition.

Discussion.—Dr. F. A. E. SILCOCK: In my opinion all dermatologists who report on cases of suspected occupational dermatitis should have personal practical knowledge of the exact industrial job at which the patient has been employed, and it has been my rule to see all manufacturing processes in actual working, so that a first-hand idea of working conditions and hazards should be gauged. Practical knowledge acquired in this manner may be very useful in several ways, e.g.:

- (1) One can estimate the risks of dermatitis.
- (2) In many cases suitable measures for preventing or dealing with dermatitis can be suggested.
- (3) It is obviously important to be able to state in your report that you have personally inspected the exact job on which a patient has been employed.
- (4) You will get an interesting and varied insight into many things, dermatological and otherwise, which will often be of great assistance to you if you can put such "tips" to use in everyday life. I have learnt a tremendous lot from visiting all kinds of industrial works, and have always found both employers and employees helpful and co-operative.

I will now take these points and briefly explain my meaning.

(1) I was asked to see a severe case of generalized dermatitis, affecting the entire body and limbs and even the conjunctivæ, in a munition worker. His job was that of degreasing shell-cases. He put dirty, greasy shell-cases on an antiquated three-tiered contraption which he then let down by a rope, with a bump, into a vat of boiling trichlorethylene for a short while, then he hauled them out again on this same apparatus. In doing so he got a spray both of hot trichlorethylene and its vapour, with the result mentioned.

(2) In this case, I suggested altering this manual method of dropping shell-cases into boiling trichlorethylene and substituting a travelling conveyor belt on which the cases could be placed. The belt then went on into the hot degreasing solution and emerged on the other side with the clean dry shell-cases, thus entirely preventing skin contact with liquid or vaporized trichlorethylene.

In a biscuit factory where operatives were having trouble from handling sugar for filling sweet biscuits, the installation was advised of automatic apparatus to supply protective emulsified cream for the operatives to smear over their fingers and hands after washing and before starting work. This apparently met with the desired effect as the trouble ceased.

In another factory, making electrical components, it was the practice to use an X-ray viewing box with a fluorescent screen, under which the part to be examined was placed so that the connexions of the metal wires could be seen and inspected. The operative was in the habit of inspecting several thousands of these parts daily and as sometimes a part when being inspected would drop into the X-ray viewing box, she would lift up the lead protective lid and manually remove the part whilst the X-ray tube was working. X-ray dermatitis of the fingers resulted. It was arranged that the electrical circuit was connected in series with the metal protecting lid and the X-ray tube, so that if the former was lifted up for any purpose it automatically switched off the current to the tube.

(3) With reference to the value of first-hand knowledge, a man employed in cutting out sole-leather had had his left thenar-eminence cut off in the machine; a surgeon had performed an excellent job by grafting a full thickness skin graft together with sub-cutaneous fat from the abdominal wall, but the man claimed that he could not carry on the same job again owing to the anæsthesia present over the newly acquired thumb pad. On investigation, I found that when cutting out the shaped sole-leather it is essential for the operative to hold a specially shaped cutting knife, which is the exact outline of the size of sole required, in both hands and run this over the leather worked upon; whilst doing so he had to feel with the thenar eminences of the hands the surface of the leather for inequalities; when he gets to the desired place he then causes this cutting shape to be pressed through the leather by a mechanical press with considerable force behind it. Only a man who had complete sensation in his fingers and thenar eminences could do this work satisfactorily as the leather must be cut to the best advantage or he will be no good as he would be too wasteful. I therefore gave the verdict in favour of the man.

(4) I have learnt a tremendous lot from visiting factories; I first saw emulsifying bases used to remove grease from soiled knitted fabric at a hosiery factory, hence my interest in triethanolamine, Lanette Wax S.X., &c., which I later advocated in dermatology. Trichlorethylene has been used for some time in factories to remove grease and I have used it for a considerable time to clean skin prior to operating on it, particularly if diathermy is to be used, as it is non-inflammable, an excellent degreaser, superior to ether, and has definite antiseptic properties as well. The higher sulphonated fatty alcohols, lauryl, stearyl and cetyl, are all fully fledged children in industry, whereas they are only now in their teething stage in dermatology, but I would like to forecast a future for them in this latter. Plastics are also another useful line in both industry and medicine.

Dr. W. H. F. OXLEY: As a general practitioner, I was hoping to have heard something that would have helped in deciding from the morphology of the lesion whether the disease is industrial or not.

I am interested in this because in wartime dermatologists and examining surgeons should get together and try to reduce the enormous amount of time lost to production owing to dermatological causes. I have learned much from what Dr. Horner has said which I shall hope to disseminate among those who are working in factories on the prevention of industrial diseases. There was a decrease in dermatitis for several years, but during the war the incidence has jumped up and we should do our best to prevent it in the interests of winning the war.

Dr. J. E. M. WIGLEY: In the most recent publication from America, *Industrial Dermatitis*, by Schwartz and Tulipan, of New York, the statement is made that the various protective applications of greases are a very temporary and not effective method of solving the problem of protection of the worker. I would like to hear whether Dr. Horner is satisfied that these degreasers are a better proposition than we are led to expect.

Dr. H. W. ALLEN: As a dermatologist I find myself sometimes at variance with the examining surgeon who gives a diagnosis which it is impossible to reverse. I suggest that the making of diagnoses be taken out of the hands of those occupied with the medical inspection of factories and put into the hands of dermatologists; the examining surgeon establishes his diagnosis without difficulty by rapid recourse to the confirmation of a medical referee, who may not be a dermatologist.

Dr. H. HALDIN-DAVIS: The most interesting series of cases I have seen among industrial dermatoses were a number of men affected with "perna disease", i.e. follicular dermatitis with comedones, who were using wires coated with trichloronaphthalene for making railway signal installations. As a rule this disease only affects men concerned in the actual manufacture of this compound.

I do not agree with the speaker who suggested the removal of the diagnosis of industrial dermatitis from the realm of the factory surgeon. His diagnosis is frequently appealed against and can be reversed quite easily. The law provides for the settlement of these cases very cheaply and expeditiously by a medical referee without the necessity of bringing the parties into open court and employing elaborate legal machinery.

Dr. H. W. ALLEN: Surely the diagnosis approved by a medical referee is irreversible. It is only when one goes to court that the diagnosis is altered by the opinion of an expert witness.

The PRESIDENT: As laid down in the Workmen's Compensation Act the certifying surgeon can make a diagnosis and give a certificate, but it can be reversed, and frequently is, by the medical referee. It is the latter's decision which cannot be reversed.

There are two defects which, in my view, need correction or amendment in that Act. The first is the long time that may elapse before the victim of a dermatosis receives any sort of monetary compensation, either from National Health Insurance funds or from an insurance company. I have come across cases where the disabled workman was actually penniless before the case was settled. The other is that the Workmen's Compensation Act makes no provision whatsoever for skilled or specialized treatment. If that were incorporated it would largely mitigate some of the hardships with which we are familiar to-day.

Dr. R. KLABER: I should like to ask a question about cutting oils. The ordinary lubricating oils only cause oil acne, and never anything like a true dermatitis, but cutting oils, which seem to be used increasingly, are causing much dermatitis. Are the responsible agents known? And is it possible to substitute one cutting oil for another?

With regard to respirator dermatitis, one wonders whether it is an accelerator or an anti-oxidant in the rubber which is at fault, and whether its use can be entirely avoided.

Bakelite substances seem to be specially tiresome in the form of liquid varnishes, and one would be interested to know why they are being used so much and whether they are always phenolic resins.

Dr. L. FORMAN: I agree with Dr. Silcock that dermatologists should acquaint themselves with the working conditions of the men who are sent to them as cases of industrial dermatitis. A knowledge of the processes involved and of the technical terms and "jargon" of each trade is essential.

I have had the opportunity of visiting two works recently. In a rope-making factory many of the workers had been off duty with boils. They handled fibre, at some stages impregnated with spindle oil. Those workers who took care to cover their legs and arms with thick garments, and particularly those who maintained a good standard of personal cleanliness, were comparatively free. Of the rest, all had varying degrees of oil acne and staphylococcal secondary infection. In a tannery, the men handled alkalis for the removal of hair, and strongly acid tanning solutions. All the men's hands showed a chronic dermatitis; some had more widespread changes.

Simple care and cleanliness of the skin will prevent a high proportion of dermatitis due to oils, dirt, alkalis, and acids.

In both the above works, facilities for maintaining a reasonable standard of cleanliness did not exist. There were a few dirty washbasins, no towels, no adequate supply of protective for the skin, and no changing rooms.

Even in contact with chemicals to which a specific sensitization would be expected, cleanliness may prevent this occurring.

During the last war there was a trinitrotoluene factory in Scotland where, during the whole period of its working, there was not a single case of T.N.T. poisoning. It was done by keeping the factory floor and benches scrupulously clean, and by giving each worker as she came into the works a clean towel, nailbrush, veil, and special gloves and boots for her own personal use; in other words, keeping the skin clean.

I have had seven cases of respirator dermatitis, a condition which is very easy to recognize and with which the War Office is familiar. No trouble is experienced when the respirator is changed, providing certain batches of rubber are avoided. The numbers stamped on masks made from these batches of rubber can be obtained from the War Office. It is interesting that the irritant is very easily soluble in saline, and patch tests on the forearm with pieces of moistened cotton-wool rubbed on the inside of the rubber part of the respirator, invariably gave positive results, namely, a vesicular or bullous dermatitis.

Dr. W. N. GOLDSMITH: One should make every effort to visit factories when possible, as otherwise even the expert dermatologist is likely to make mistakes in diagnosis. As a result of a recent visit to a cigarette factory and my request that they should send me *all* their cases of apparent tobacco dermatitis, and not merely the doubtful ones, I learnt for instance that the characteristic distribution was the bend of the elbows and the back of the neck. It hardly ever involved the hands, though the affected workers were usually "strippers". It attacks almost exclusively new workers after a few weeks and disappears rapidly if they stay away from work. They are then generally able to resume it with impunity.

Dr. SHERRY-DOTTERIDGE: I should like to ask Dr. Horner whether in this war cases of trinitrotoluene poisoning have increased or are very much less than in the last war and, if there is a decrease, can it be due to preventive cleanliness, or is there some secret which we have not been told?

I have been working in a rubber research works and have been rather fortunate in seeing some cases caused by irritating dust within twenty-four hours of a rash appearing. Research workers gave a history of itching and faint erythema, which passes off if washed immediately. Workers who do not take these steps go on to a more severe attack which clears up when given other work for a few days.

In two cases that went on to exfoliative dermatitis it was found that the patients had given themselves a good rub with sulphur ointment. That was only discovered after great difficulty, and I wonder whether most of the severe trade dermatitis seen in hospital might have been aggravated by home treatment.

Dr. W. GRIFFITH: My experience is that many factories have medical officers and a surgery with trained nurses, and it would obviously be a great boon if this system was extended. We regularly have nurses sent down to St. John's Hospital for Diseases of the Skin from the College of Nursing during their training as industrial nurses.

Some employers have informed me that doctors have told them that there is a way of de-sensitizing patients, or making them immune from dermatitis. I must confess I was rather sceptical. I would like to ask Dr. Horner whether she knows of any system of injection or otherwise which would make a patient immune from acquiring dermatitis.

Dr. T. O. GARLAND: I also consider that dermatologists should go more often into factories. Doctors should not be continually giving opinions on industrial diseases or accidents without seeing the site of the accident or disease. Our whole profession needs more direct linking with production. It should be appreciated that it is very difficult to-day for workers to get any accurate information on the dangers of the materials they handle. Our profession has a long-neglected duty to remedy this ignorance. Many employers can also be described as "unthinking". In this room recently I heard described an employer who asked that some ointment should be used in the prevention of dermatitis, which he claimed would also cure nystagmus. Doctors, too, are not guiltless. Some shop stewards recently told me of a doctor who had declared that their job of degreasing metal spars over an open bath of trichlorethylene held no possible dangers for them.

Dr. R. T. BRAIN: There is just one aspect which has not been touched upon: that is the use and nature of protectives. Are the protective agents proprietary preparations

or are there others in use which are not patented, and do they themselves irritate the skin? We are now very interested in the increasing use of emulsifying agents. Has Dr. Horner seen any ill-effects from these agents upon normal skin and could she tell us whether different preparations are used to protect from grease or from water?

Although cleanliness is desirable if the workman is using an irritant, yet in many other occupations a little wholesome dirt does less harm than frequent cleansing.

Dr. F. W. JACOBSON: Has Dr. Horner observed whether patients with a trifling degree of neuro-dermatitis, eczema or psoriasis, are more prone to develop industrial dermatitis? I am thinking of "atopic" dermatitis and am not quite clear whether it is only in the imagination of Coca and his pupils or a fact that a family history of hay-fever and chronic asthma predisposes to industrial dermatitis. Is care taken before workers are accepted in factories where there is special risk of dermatitis to examine and if necessary to weed out those who give a personal or family history of "atopic" dermatitis, asthma, hay-fever, or migraine?

The PRESIDENT: I think your question was answered by the word "selection" used by Dr. Horner.

Dr. JACOBSON: Does it go so far as the family history?

Dr. SILCOCK (in reply) said: A question was asked regarding the unfortunate man waiting for his compensation. In Leicester and neighbourhood I have got over that difficulty by arrangement with the Friendly Societies who advance the man his wages on his signing that he will refund if he does not get compensation.

With regard to the entry to factories. I have not had the least difficulty. Dr. Klaber asked about cutting oil: I see much dermatitis from it, but I have also seen people who have worked for many years without difficulty with sternal cutting oil, and when they are put on high-speed steel and use sulphurized cutting oil they get dermatitis and have to finish. Another irritant is the suds which are a mixture of caustic soda and soap.

Dr. HORNER in reply to the different queries put to her in the course of the meeting, said that entry to factories was only by authorized pass. For the prevention of dermatitis in industry a properly chosen and correctly applied barrier substance was effective.

The term "Selection" in wartime meant selection from among the personnel available.

Emulsions of oil in water and mineral oils in contact with the skin gave rise to different reactions.

There was at the Royal Society of Medicine in April, 1942, and recorded in the *Proceedings* a full discussion of the effects of T.N.T. (*Proc. Roy. Soc. Med.*, 34, 553, Sect. Therap., 19.)

Dr. Horner had noticed in many cases of dermatitis among strippers (tobacco) the same distribution as Dr. Goldsmith had observed, but a recurrence in these cases was not uncommon. She knew of nothing which gave immunity from a recurrence of dermatitis. However, the incidence of such recurrence could be lessened by early diagnosis, specialized treatment. A return to the original work should be permitted only after a period of convalescence of the skin. Of course, the co-operation of the person concerned was necessary to secure a good and lasting result.

Section of Orthopædics

President—C. LAMBRENUDI, F.R.C.S.

[February 21, 1942]

MEETING AT THE ROYAL NATIONAL ORTHOPÆDIC HOSPITAL

The Treatment of Spinal Injuries with Nervous Involvement

By K. I. NISSEN, F.R.C.S.

SINCE the onset of the war, some forty-five cases of traumatic paraplegia have been admitted to Stanmore. With the exception of ten gunshot wounds, the injuries have been of the type commonly met with in heavy industry. A number of patients, however, have lost their homes and near relatives, and have shown little desire to recover. Though the majority have come from the London area, only a handful have been transferred within the first week. As a result, few cases of the early recovery so common in general hospital practice have been seen. A number have had the bony deformity well corrected, but at considerable cost. Two such cases were received ten days after their air-raid injuries. Parts of their sodden plaster jackets had been removed to provide access for suprapubic drainage. Pressure sores covered the sacral and lumbar spines. One of these women had a urinary dermatitis extending up to the scalp and died the next day. The other is now able to walk but has had serious renal infection. These distressing cases emphasize the need for orthopædic treatment in harmony with the nursing care of the bladder, the bowels, and the back.

Before coming to the general care of paraplegic patients, brief mention may be made of those cases in which an immediate surgical procedure may be considered, as for instance, in gunshot wounds and in fractures of the laminae. Enthusiasm for operation is tempered by the fact that, whatever the subsequent treatment, no cord lesion which is still complete after forty-eight hours shows a useful degree of recovery. This holds true whether a bony lesion is present or not, and is also true when depressed fractures of the laminae or small fragments of metal invite operation. The prognosis in severe lesions of the cauda equina can seldom be given so early, but no case of successful suture has yet been recorded in this country.

Of eight gunshot wounds with severe paralysis, two had missiles removed from the neural canal. In one case the cord was severely bruised while in the other it was divided. Both cases died within a week. The other six were left alone and all have survived without local complications from retention of the metallic fragments.

The question of operation again arises when fracture-dislocation of the lumbar spine is suspected. Two joints and four articular processes are involved, and the expected combinations of dislocation with or without fracture of the denticles are actually found. Critical X-rays are necessary to exclude unilateral or bilateral fracture of the articular processes in which there may be no posterior obstacle to reduction by closed manipulation. On the other hand these simple manœuvres alone are useless in bilateral dislocations with intact and interlocked facets. Hyperextension may elevate the anterosuperior fragment of the body of the inferior vertebra, but with separation. When the jacket is discontinued,

or even sooner, marked wedging recurs with angulation of the theca. It is commonly believed that hyperextension of an unreduced fracture-dislocation may increase the paralysis immediately. Cases occur, however, where the paralysis ultimately resolves despite this treatment, and there is no doubt that reduction improves the stability of the spine rather than the immediate prognosis of the paralysis. Post-mortem specimens also show that reduced posterior intervertebral joints may ossify firmly, giving the effect of a localized spinal fusion.

The mechanism to be used in reduction is logical. The locked position is one of recoil. When recoil is only partial, the widely separated articular processes are plainly visible in the X-ray films and suggest how considerable the displacement may be at the time of impact. A cautious reversal of the mechanism of injury may therefore be employed in reduction and the spine well flexed over a broken operating table after exposure of the region under general anaesthesia. It may be possible then carefully to lever back the intact articular processes, while backward traction on the superior spinous process is maintained. If an articular facet still hinders reduction, as little as possible should be removed. Complete excision of a facet seriously impairs the posterior stability of the spine, and if the spinal jacket cannot be maintained for any reason the deformity is liable to recur.

Reduction may not occur even when any posterior bony obstacle has been removed. Hyperextension has not been found effective at this stage. The posterior border of the superior body only hinges well forward on the upper table of the lower vertebra. The displacement between the bodies may be reduced by strong pressure in opposite directions on the upper and lower segments with the spine well flexed. Only when reduction has been seen should hyperextension be applied to maintain it. Local anaesthesia may serve well for operative exposure but is not suitable for subsequent manipulations.

Only five cases of fractured cervical spine with paraplegia have been treated. Respiration is almost entirely diaphragmatic and a mechanical respirator should be available. Cases of high cord involvement suffer respiratory distress when prone, and the face down position for transport is contra-indicated. In general no harm has come from transport with the patient on his back except during the evacuation from Dunkirk.

Whether operation is performed or not, the patient usually has to receive immobilization of the fracture, compatible with full nursing care over a period of months. Many are treated in a plaster jacket. The level of complete anaesthesia is most important in determining whether a jacket, with its risk of pressure sores, should be applied. When the level extends higher than the groin, the risk is so great that it exceeds the doubtful value of correction and of the patient's mobility. Jackets were applied very carefully in three cases with complete anaesthesia to a level between the pubis and umbilicus, but in spite of frequent turning superficial sores developed in the usual places. After six weeks deep sloughs down to the ischial tuberosities suddenly appeared and the jackets had to be abandoned in favour of plaster beds. The retention of posterior column sensibility, however, reduces the tendency to pressure sores so very considerably that reduction and the application of a jacket may be regarded as safe. Post-mortem specimens of compression fractures show that the greatest thecal deformity occurs at a dense transverse ridge formed by the collapsed annulus fibrosus bulging backwards into the neural canal. Hyperextension affords an opportunity for restoration of the disc space which should on no account be missed when the cord is only partly damaged.

The padding of plaster jackets is important. Small pieces of adhesive felt are entirely inadequate and frequently shift off the bony prominences, while bulky padding makes for uneven creases and ridges. A stockinet vest, with a half thickness of white wool from the usual blue roll over the iliac crest and spines and a full thickness of white wool down the spinous processes, is generally satisfactory. Many jackets applied elsewhere showed marked transverse ridging from the hasty application of the jacket before full hyperextension had occurred. The strain on the arms and shoulders during reduction by Watson-Jones' two-table method can be much reduced by using pulleys and a sling, which should ride high in the axilla and so not interfere with the application of the sternal plate. The side of the patient's bed makes a convenient lower table, and if the sling is applied with the patient lying prone, he may be slung into position like a piece of cargo with the greatest of ease. The sling is invaluable when injuries to the upper limb and shoulder girdle are present.

The pubic part of the average plaster jacket does not extend downwards actually over the surface of the pubic bone. This is probably a legacy from the illustrations of Böhler and Schneck in which the diagrams indicate the need for support over the pubis, but photographs of patients show the jackets trimmed at the level of the pubic crest.

This downward extension is most important when suprapubic cystostomy has been performed or is required. A window extending from the pubic crest to the umbilicus can be made and its reinforced margins protected with vaseline gauze. The operation can be performed without soiling the cast, and with reasonable care the jacket may last two or even three months without becoming sodden from leakage of urine. Such patients may get up in a chair or attempt walking with the catheter spigoted.

Persistent vomiting and abdominal distension may occur after the application of a jacket to any case of spinal injury. In uncomplicated fractures it is common after the application of a second plaster jacket, particularly when the patient has not been doing abdominal exercises. Again it may occur when reduction has been delayed several days in a patient confined to bed since the accident. The relative frequency in cases with nervous involvement is much greater. Poor tone in the abdominal muscles is common to all these patients and is probably a much more important factor than injections of morphia or omnopon. The vomiting usually occurs some hours after reduction, but may be precipitated (after a week or more) by some minor upset such as an indigestible meal. Minor degrees of vomiting may be overlooked; but if it is very severe with marked dehydration and wasting, intermittent gastric syphonage, continuous intravenous salines and removal of the jacket are then indicated. In cases which are likely to develop this complication the surgeon should be content with a lesser degree of hyperextension.

Patients unsuitable for a plaster jacket usually have serious cord involvement at or above the 9th dorsal vertebra and may have complications such as pressure sores or urinary sepsis as well. For these difficult cases the full length plaster bed has been found most satisfactory, especially for the first six weeks. Its development proceeded naturally from the satisfactory nursing of Pott's paraplegia in plaster beds. The short beds described in most works on fractures have some serious disadvantages for heavy cases. They are not mounted on blocks and hence are easily soiled by the incontinent patient. If made in hyperextension they are prone to cause pressure sores after two or three weeks when the spinal muscles commence to waste. When used in conjunction with Braun's splints, the skeletal traction interferes with the accurate fitting of the cast and with the daily routine of full passive movement of all joints.

The value of evenly distributed pressure in the prophylaxis and treatment of pressure sores is well recognized, but this ideal is very difficult to obtain. The usual technique of making plaster beds has been modified considerably, but has already been described in the *Proceedings* (*Proc. Roy. Soc. Med.*, 34, 457 [Sect. Surg., 27]).

A full-length bed is very comfortable. The nursing care could hardly be simpler. A bedpan is constantly in position, and with the exceptions of dribbling incontinence in women, extensive pressure sores and very hot weather, the bed keeps clean and sweet. The gutters for the lower limbs avoid deformity and allow ready access for physical treatment. Patients with sores are turned out once a week. They are washed with soap and water, given an exposure to ultra-violet light, dressed with dry gauze and returned to their freshly lined beds. Sores respond well to this simple and economical treatment. Transport is greatly facilitated and the constant level is an aid to the smooth working of tidal drainage.

The disadvantages of a good full-length bed are few, the chief one being that the supine attitude favours urinary calculi. Flexor spasms make any form of nursing difficult and this is no exception. Rapid wasting in flaccid paraplegia may require early renewal of the bed. These beds, however, require some skill to make and as small errors in technique can make them thoroughly unsatisfactory they are not likely to win general favour. A water bed or a sorbo mattress are satisfactory alternatives as soon as excretion is under control.

Complete relief from pressure on the sacrum may be obtained by transfixing the iliac crests with Steinmann's pins or Kirschner wires and applying vertical traction. This has been done in only one case, but a sacral sore 3 in. in diameter and over an inch deep healed in six weeks. The pin holes may, however, form tender adherent scars.

The treatment of the bladder still remains a vexed question. Manual expression and the allowing of overflow incontinence seem to be out of favour. Cases admitted with indwelling catheters have seldom been free from sepsis, which indeed can be expected to be severe after a fortnight. Yet Frank Kidd, using a few simple precautions, found this form of drainage very successful in the last war.

Tidal drainage is a variation on the theme of the indwelling catheter. We have used the simple methods of Lawrie and Nathan and of Belliss rather than Munro's complicated apparatus, but with little real success. (Since this paper was written, Stewart's modification of the Belliss apparatus, incorporating a cystometer, has been used and found very

satisfactory.] Unfortunately the opportunity to use tidal drainage from the beginning has not yet arisen in this Centre. The longest time it has been used in a patient starting free from sepsis is fifty-eight days, with evidence of infection developing over the last week. Even four to six weeks should afford an opportunity for cases of transient paralysis to regain control or for reflex micturition to commence. We have had no cases of tidal drainage with early recovery, and the patients who have developed reflex micturition have been women. They finally had suprapubic drainage, which in women is easier to manage than truly reflex micturition. No case admitted with urinary sepsis has cleared up with tidal drainage. The method will receive enthusiastic support only in those centres with an expeditious system of transfer and meticulous general supervision.

Suprapubic drainage is the last resort in cases with urinary sepsis, and delay may be fatal. It is a simple and satisfactory treatment for cases which in time recover control, for the fistula soon closes when the tube is withdrawn. On the other hand it destroys any chance of reflex micturition in permanent paraplegia. Neurologists deplore this, but many urologists maintain that suprapubic drainage is preferable especially in women. The tube should be inserted midway between the pubis and umbilicus so as to give a sloping track. The fistula then does not become adherent to the pubis and leakage is minimal. A plaster jacket can be applied if necessary. As an initial treatment suprapubic drainage should certainly be used when the conditions of nursing care and transport are gravely disturbed. It is too frequently postponed when sepsis has arisen.

Hopelessly paralysed cases are all too frequent. Flexor spasms can give rise to grave discomfort. Flaccid cases are so comfortable in comparison that the destruction of cord function below the level of the lesion, even at the cost of reflex micturition, should be considered. In some cases of paraplegia the misery of the patient is such that one cannot forget the injunction "Thou shalt not kill; but need'st not strive officiously to keep alive".

Some Minor Fractures in the Hand

By V. H. ELLIS, F.R.C.S.

FRACTURE OF THE CUNEIFORM OR TRIQUETRAL BONE

Greening (1942) found 20 fractures of the cuneiform in 580 fractures of the wrist, and in the present series 9 have been found in 1,000 consecutive but unclassified fractures, i.e. about 1%. It is not an unimportant fracture, as the disability is considerable both in severity and duration. Although the cuneiform bone of the wrist may be comminuted by a fall on the hand, as in one case in this series, more commonly a fragment is torn from the dorsum of the bone, forming one of the class of sprain fractures. Two strong ligaments are attached to the bone, one from the semilunar, the other from the styloid process of the ulna; fractures, therefore, result from perilunar dislocation of the carpus and from forcible palmar flexion of the wrist.

Diagnosis.—The sprain fracture is not visible in an antero-posterior X-ray of the wrist, and although it is clearly shown in the lateral view the fragment may appear to be a portion of the semilunar. The site of tenderness on the ulnar side of the carpus is adequate evidence of its origin and this is confirmed in a slightly oblique X-ray. The wrist is frequently swollen, and movements, particularly palmar flexion, are restricted by pain.

Mechanism.—The mechanism of production is uncertain. In all cases it was due to a fall upon the hand, but in none was it associated with a Colles's fracture (except of the other wrist). In one case there was a spiral fracture of the 4th metacarpal bone and in another there had been a Smith's fracture of the injured wrist two years previously. Probably therefore forced flexion of the wrist is responsible.

Treatment.—Neglect of treatment, or simple strapping as for a sprain, is apt to produce a disability sufficient to prevent heavy manual labour for two to three months and perhaps longer. Immobilization in a plaster of the scaphoid type, but with some ulnar deviation, seems to be the best form of treatment. This may allow continuity of work. The plaster should be maintained for five weeks and be followed by exercises to restore movement. Union does not always occur and the fragment may sclerose (fig. 1), but this does not seem to prevent full painless function.

Results.—Of this series of 9 cases: One comminuted fracture was in plaster two months and discharged with full function in four months; eight cases immobilized for an average of four and a half weeks were discharged after an average of eight weeks; two had a disability preventing return to original heavy work.



FIG. 1.—Fracture of carpal cuneiform.

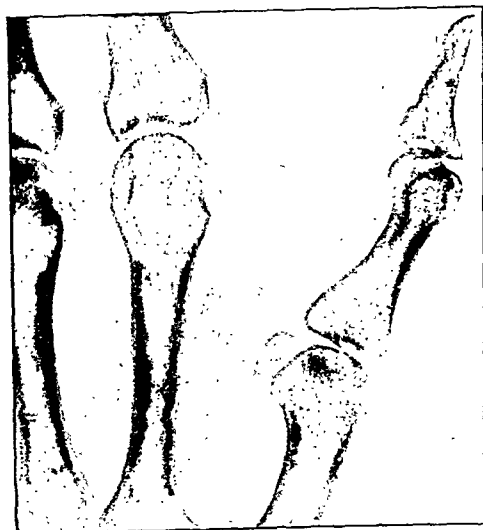


FIG. 2.—Fracture of sesamoid.

FRACTURE OF A SESAMOID BONE OF THE THUMB

In Scobie's case (1941), it was presumed that the bone was crushed by direct violence. In the writer's case the patient fell dislocating the thumb. She reduced the dislocation herself but there was subsequent swelling and tenderness over the ulnar sesamoid. This bone was apparently transversely fractured by traction (fig. 2). There are therefore probably two forms of fracture comparable to those found in the patella. The thumb was splinted in flexion on Kramer wire for six weeks and the patient was discharged to her original domestic work after two months.

References.—GREENING, W. P. (1942), *Brit. M. J.* (i), 221. SCOBIE, W. H. (1941), *ibid.* (ii), 912.

[April 11, 1942]

MEETING AT ST. NICHOLAS' ORTHOPÆDIC HOSPITAL

Practical Points in Connexion with Amputations

By GEORGE PERKINS, M.C.

THESE remarks apply only to final amputations done through uninfected tissues with a reasonable certainty of primary healing.

SITES OF ELECTION

Skilful limb-makers can fit artificial limbs to stumps of any length or shape, but they tell us that certain limbs look better and function better than other limbs, and that they can only fit these good limbs to stumps of a certain length; and in the matter of amputations surgeons must resign themselves to take orders from the experienced limb-makers. There is no doubt about their experience: at the limb-fitting centre at Roehampton for example they have fitted 40,000 leg amputees since the last war, and have already in this war supplied 1,123 limbs. Their conclusions are roughly these:

(1) End-bearing stumps do not last. The majority of pensioners of the Great War with Syme and transcondylar amputations—amputations designed to take end-bearing—have required reamputation at a higher level. It should be noted, however, that this conclusion is not accepted by our Canadian cousins, who consider that in the lower limb the Syme and the Stokes-Gritti amputations are to be preferred to all others. It may be that the Canadian limb-makers produce a better limb than we in this country; the fact remains that the limbs evolved by British limb-makers for amputations at these two levels are unsatisfactory.

(2) The shorter the stump, the less trouble it gives. The troubles are mostly circulatory. The end of a long stump becomes cold, blue and congested, and finally ulcerates; and stumps a trifle shorter suffer in proportion to their length.

- (3) The stump must be long enough to remain within the socket during movement.
- (4) The stump must be long enough to contain the insertion of the muscles activating the joint above the amputation.
- (5) The stump must not have a bulbous extremity; otherwise it will not fit inside a conical socket.

Applying these theoretical conclusions, the limb-makers tell us that there are two sites of election in the arm and two in the leg. In the leg, one is above and one below the knee—the above-knee amputation and the below-knee amputation. In the arm one is above the elbow and the other below, the above-elbow and below-elbow amputation.

Above-knee amputation.—The ideal length for an above-knee stump is between 10 in. and 12 in., depending on the height of the patient. The measurement is made from the top of the great trochanter to the end of the femur.

A stump shorter than this is not so good, because, the main adductor muscle being inserted all the way down the shaft of the femur, a short femoral stump is deficient in adductor power and the unopposed abductor muscles hold the artificial leg abducted.

A short stump is difficult to retain inside the socket when the patient flexes his hip actively, especially if the subcutaneous tissues are bulky; and it is often necessary to fit a shoulder-controlled limb instead of a stump-controlled limb. Shoulder-control and stump-control are two terms used by limb-makers and require definition. There are two ways of activating an artificial leg. In one, the patient lifts his limb off the floor by tip-toeing on the other foot. Once the limb is off the ground, the patient by shrugging his shoulders applies tension to shoulder braces attached to the limb and thereby flexes the hip-joint. The flexor muscles of the hip are inert and the stump therefore does not tend to leave the socket. This method—shoulder-control—produces an ungainly gait, and new amputees are being fitted whenever possible with stump-controlled limbs. In the stump-control method the hip is moved actively by its own muscles; whenever the limb is being advanced the hip flexors go into action, and if the stump is short the stump tends to come forward out of the socket.

When the stump is less than 6 in. it is not practical to fit an above-knee limb, and the patient has to put up with a tilting-table limb. This limb has an artificial hip as well as an artificial knee. But although the patient's hip is not used the limb-makers like left 4 in. of the shaft of the femur measured from the top of the trochanter. This small piece of femur when flexed to a right angle provides a boss of bone anteriorly (the end of the stump) and a boss of bone laterally (the great trochanter) round which to mould the pelvic socket. These bosses prevent the socket from rotating on the pelvis.

In an above-knee limb weight is transmitted through the tuber ischii.

Below-knee amputation.—The ideal length of a below-knee stump is $5\frac{1}{2}$ in., measured from the inner joint line to the end of the tibia.

All the muscles that control the knee are inserted high on the tibia, so that from the point of view of muscle control a below-knee stump need not be more than 2 in. in length. But, unless the stump measures at least 4 in. it will not remain inside the socket when the knee flexes. And, on the assumption that the shorter the stump the less trouble it gives, it would appear that 4 in. of tibia should be the aim. Two other considerations, however, affect the decision: The Royal Warrant lays down that the pension for a man with a stump of less than 4 in. is higher than when the stump is longer than 4 in. This dates from the time when it was thought that 7 in. was the ideal length. Therefore to avoid argument, it is better for the stump to be longer than 4 in. The second and practical consideration is that a below-knee amputation is difficult to perform satisfactorily; so often the skin gapes and the wound heals by granulation tissue leaving a wide, poorly nourished scar. It is better therefore to start with a tibia longer than necessary so that if a trimming operation has to be done later a portion of the bone can be sacrificed to enable the skin edges to be sutured without tension.

For these reasons the ideal length for a below-knee amputation is declared to be $5\frac{1}{2}$ in.

It is not universally appreciated that the limb-makers have made a change in the site of election, and the general belief is that a satisfactory below-knee limb cannot be fitted to a stump shorter than 7 in. As a matter of fact the limb-fitting surgeons at Roehampton prefer a 4 in. tibial stump to one of $5\frac{1}{2}$ in.

Not only has there been a change in the ideal site for amputation, but also in the method of weight-bearing. Hitherto it has been customary for a below-knee amputee to take weight under the expanded upper end of the tibia and the head of the fibula. Experience has shown that these bearing points after a few years develop painful bursae, and more and more below-knee stumps are being fitted from the beginning with a long thigh corset taking a bearing under the tuber ischii.

Below-elbow amputation.—The ideal length for a below-elbow stump is 7 in. measured from the tip of the olecranon to the end of the ulna.

In theory it would seem advisable to retain the lower radio-ulnar joint so as to preserve active rotation of the forearm. In practice, however, if the socket is loose enough

to accommodate the change of shape in the forearm during rotation it is too loose to fit the stump. An amputee cannot rotate his forearm when wearing a limb although he may be able to do so when not wearing a limb.

The muscles controlling the elbow are inserted high in the forearm so that the factor of muscle control is of no importance in determining the length of a below-elbow stump.

Nor does a long below-elbow stump suffer to the same extent from vascular disturbances as a long below-knee stump; so that it would seem immaterial what length is chosen. The advantage, however, of having a shortish stump is that appliances can be brought nearer the elbow and thereby can be better controlled. The shorter the stump the better, from this point of view, but the stump must be long enough to get a good purchase on the socket of the limb. Therefore a length of 7 in. is chosen as the ideal. The length should not be less than 4 in. because a stump shorter than this cannot be retained in the socket when the elbow is flexed.

Above-elbow amputation.—The ideal length for an above-elbow stump is 8 in., measured from the point of the acromion process. The only consideration in deciding the length of an above-elbow stump is that the bone section should be made above where the shaft begins to widen. An amputation through or close to the elbow presents a bulbous bony extremity, and the club-shaped stump will not fit into a conical metal socket. The socket has in consequence to be made of split leather which is laced up after the stump is laid in it; such a socket is heavy, clumsy and hot. A length of at least 6 in. is needed to retain the stump inside the socket. The extra 2 in. suffices to hold the stump securely. More is not needed, and a total length of 8 in. ensures that the bone is divided through the narrow part of the shaft and leaves sufficient room for the fitting of the artificial elbow at its natural level.

SITUATION OF THE SCAR

Formerly, when end-bearing was the vogue, great pains were taken to avoid a terminal scar. By cutting unequal flaps the scar was located an inch or more away from the end, usually on the posterior surface. In the modern artificial limb the socket does not make contact with the end of the stump, and a terminal scar far from being objectionable is an advantage, since in this situation it is shielded from pressure.

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The difficulty is how to ensure that the scar shall not adhere to the muscles. I endeavour to do this by cutting through the deep fascia at the same level as the skin and reflecting it back with the skin; and at the end of the amputation I cover in the raw bone and the raw muscles by sewing over them the deep fascia.

Muscles.—For many years now limb-makers have been asking surgeons not to cover the end of the bone with muscle, since this leaves a baggy uncontrollable mass which can only with difficulty be inserted into the conical socket. The muscles should be cut

- (3) The stump must be long enough to remain within the socket during movement.
- (4) The stump must be long enough to contain the insertion of the muscles activating the joint above the amputation.
- (5) The stump must not have a bulbous extremity; otherwise it will not fit inside a conical socket.

Applying these theoretical conclusions, the limb-makers tell us that there are two sites of election in the arm and two in the leg. In the leg, one is above and one below the knee—the above-knee amputation and the below-knee amputation. In the arm one is above the elbow and the other below, the above-elbow and below-elbow amputation.

Above-knee amputation.—The ideal length for an above-knee stump is between 10 in. and 12 in., depending on the height of the patient. The measurement is made from the top of the great trochanter to the end of the femur.

A stump shorter than this is not so good, because, the main adductor muscle being inserted all the way down the shaft of the femur, a short femoral stump is deficient in adductor power and the unopposed abductor muscles hold the artificial leg abducted.

A short stump is difficult to retain inside the socket when the patient flexes his hip actively, especially if the subcutaneous tissues are bulky; and it is often necessary to fit a shoulder-controlled limb instead of a stump-controlled limb. Shoulder-control and stump-control are two terms used by limb-makers and require definition. There are two ways of activating an artificial leg. In one, the patient lifts his limb off the floor by tip-toeing on the other foot. Once the limb is off the ground, the patient by shrugging his shoulders applies tension to shoulder braces attached to the limb and thereby flexes the hip-joint. The flexor muscles of the hip are inert and the stump therefore does not tend to leave the socket. This method—shoulder-control—produces an ungainly gait, and new amputees are being fitted whenever possible with stump-controlled limbs. In the stump-control method the hip is moved actively by its own muscles; whenever the limb is being advanced the hip flexors go into action, and if the stump is short the stump tends to come forward out of the socket.

When the stump is less than 6 in. it is not practical to fit an above-knee limb, and the patient has to put up with a tilting-table limb. This limb has an artificial hip as well as an artificial knee. But although the patient's hip is not used the limb-makers like left 4 in. of the shaft of the femur measured from the top of the trochanter. This small piece of femur when flexed to a right angle provides a boss of bone anteriorly (the end of the stump) and a boss of bone laterally (the great trochanter) round which to mould the pelvic socket. These bosses prevent the socket from rotating on the pelvis.

In an above-knee limb weight is transmitted through the tuber ischii.

Below-knee amputation.—The ideal length of a below-knee stump is $5\frac{1}{2}$ in., measured from the inner joint line to the end of the tibia.

All the muscles that control the knee are inserted high on the tibia, so that from the point of view of muscle control a below-knee stump need not be more than 2 in. in length. But, unless the stump measures at least 4 in. it will not remain inside the socket when the knee flexes. And, on the assumption that the shorter the stump the less trouble it gives, it would appear that 4 in. of tibia should be the aim. Two other considerations, however, affect the decision: The Royal Warrant lays down that the pension for a man with a stump of less than 4 in. is higher than when the stump is longer than 4 in. This dates from the time when it was thought that 7 in. was the ideal length. Therefore to avoid argument, it is better for the stump to be longer than 4 in. The second and practical consideration is that a below-knee amputation is difficult to perform satisfactorily; so often the skin gapes and the wound heals by granulation tissue leaving a wide, poorly nourished scar. It is better therefore to start with a tibia longer than necessary so that if a trimming operation has to be done later a portion of the bone can be sacrificed to enable the skin edges to be sutured without tension.

For these reasons the ideal length for a below-knee amputation is declared to be $5\frac{1}{2}$ in.

It is not universally appreciated that the limb-makers have made a change in the site of election, and the general belief is that a satisfactory below-knee limb cannot be fitted to a stump shorter than 7 in. As a matter of fact the limb-fitting surgeons at Roehampton prefer a 4 in. tibial stump to one of $5\frac{1}{2}$ in.

Not only has there been a change in the ideal site for amputation, but also in the method of weight-bearing. Hitherto it has been customary for a below-knee amputee to take weight under the expanded upper end of the tibia and the head of the fibula. Experience has shown that these bearing points after a few years develop painful burse, and more and more below-knee stumps are being fitted from the beginning with a long thigh corset taking a bearing under the tuber ischii.

Below-elbow amputation.—The ideal length for a below-elbow stump is 7 in. measured from the tip of the olecranon to the end of the ulna.

In theory it would seem advisable to retain the lower radio-ulnar joint so as to preserve active rotation of the forearm. In practice, however, if the socket is loose enough

to accommodate the change of shape in the forearm during rotation it is too loose to fit the stump. An amputee cannot rotate his forearm when wearing a limb although he may be able to do so when not wearing a limb.

The muscles controlling the elbow are inserted high in the forearm so that the factor of muscle control is of no importance in determining the length of a below-elbow stump.

Nor does a long below-elbow stump suffer to the same extent from vascular disturbances as a long below-knee stump; so that it would seem immaterial what length is chosen. The advantage, however, of having a shortish stump is that appliances can be brought nearer the elbow and thereby can be better controlled. The shorter the stump the better, from this point of view, but the stump must be long enough to get a good purchase on the socket of the limb. Therefore a length of 7 in. is chosen as the ideal. The length should not be less than 4 in. because a stump shorter than this cannot be retained in the socket when the elbow is flexed.

Above-elbow amputation.—The ideal length for an above-elbow stump is 8 in., measured from the point of the acromion process. The only consideration in deciding the length of an above-elbow stump is that the bone section should be made above where the shaft begins to widen. An amputation through or close to the elbow presents a bulbous bony extremity, and the club-shaped stump will not fit into a conical metal socket. The socket has in consequence to be made of split leather which is laced up after the stump is laid in it; such a socket is heavy, clumsy and hot. A length of at least 6 in. is needed to retain the stump inside the socket. The extra 2 in. suffices to hold the stump securely. More is not needed, and a total length of 8 in. ensures that the bone is divided through the narrow part of the shaft and leaves sufficient room for the fitting of the artificial elbow at its natural level.

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through at the same level as the bone. It would seem unnecessary to mention this were it not that one still sees stumps in which the muscles have been left long and sewn over the end of the bone.

There is, however, one point of interest regarding the muscles. In using an above-knee limb the adductor muscles are of great importance, and for some time I have been searching for a method of attaching the huge belly of the adductor magnus to the femur in order to preserve its length and therefore its power. As it is, the muscle retracts and, not having a lower fixation to bone, is of little use as an adductor. So far I have not been successful, and I wish some of you would consider the problem when next doing an above-knee amputation.

Nerves.—Neuromata form inevitably whenever nerves are cut across. Neuromata elsewhere in the body are usually not tender; in a stump they may be. The possible causes for the tenderness are: (1) Pressure from the socket of the limb; (2) irritation from neighbouring fibrous tissue; the result of past sepsis or of maltreatment of the nerve at the time of the amputation; and (3) repeated traction from piston action when the skin is adherent to the underlying muscles. By reviewing the possible causes one is in a better position to counter them.

It is, I believe, the usual practice to cut the nerves shorter than the muscles. This dates from the time when end-bearing was the aim, the idea being to remove the neuromata from the end of the stump. But in the modern limb the end of the stump is not in contact with the socket; and I hold therefore that it is better not to shorten the nerves, since they are more liable to be pressed on by the socket if shortened than if left long. Many surgeons, besides pulling on the nerves to shorten them, clamp and ligature them and sometimes in addition inject them with alcohol. All these proceedings are traumatic and likely to increase the amount of scar around the nerves. I believe it best to leave the nerves alone; if the surgeon does not even know where they are, so much the better. The third cause for tenderness—traction from piston action—can be avoided by getting a non-adherent scar.

Tender neuromata are less common than they were. In the past year at Roehampton I have only once operated for a tender neuroma; whereas I am under the impression that at Shepherd's Bush, after the last war, a week rarely passed without one or more operations on neuromata. I have been told that it is too early yet to expect them in this war. Feeling as I do that an adherent skin scar is the most potent causal factor, I believe that, should operation become necessary, it is better to remove a portion of the main trunk of the nerve well above the tender area rather than excise the neuroma itself, for the end of the nerve will certainly become adherent to the operation wound if the neuroma has been approached through scar tissue.

Hæmostasis.—In amputations as much care should be paid to controlling hæmorrhages as in operations inside the skull or the abdomen. It is a tedious business to check innumerable small bleeding points, for no sooner have a score or so been clamped and tied (or seared with diathermy) than a score of others appear. Nevertheless time spent in securing hæmostasis is time well spent, especially in a below-knee amputation. Pulling apart of the skin edges, sloughing of the edges, and sepsis—the three bugbears that ruin an amputation—rarely occur if a hæmatoma is avoided.

As well as taking infinite pains to ligate all bleeding points however small, it is advisable to apply pressure to the end of the stump. I use two long strips of elastoplast over a moderate amount of dressing. This seems to me more effective than a pressure bandage applied over a large mass of wool.

Drainage.—Coming to the vexed question of drainage: I feel safer when I have drained, yet I cannot remember having seen a drain do any good. Should hæmorrhage occur, a drain does not function, for the blood clots and does not flow out. My present practice is not to drain unless I am apprehensive of sepsis, and then to leave the drain in several days so as to facilitate a channel for the exit of pus should it form.

POST-OPERATIVE TREATMENT

The task of the surgeon ought not to finish with the healing of the wound. It is his further duty to prepare the stump for the artificial limb, and also to prepare the patient for using the limb—two distinct processes.

The stump in its immediate post-operative state is too large and is the wrong shape; it is required to convert a wide cylinder into a narrow cone. Time alone will effect this, but the process can be hastened (a) by bandaging or (b) by the wearing of a temporary prosthesis. The limb-fitting surgeons at Roehampton prefer the first to the second method. The bandage method has the advantage that it can be started as soon as the wound has healed. Crêpe bandages are used, and two are sewn end to end to form one long bandage. They should be 6 in. wide for an above-knee stump and 4 in. for all other stumps. The principle of the method is to compress the stump from below up.

the maximum pressure being exerted at the distal end. The bandage is applied much as one applies an ordinary stump bandage; for the pressure to be effective, however, the bandage must first be rolled very tightly and then applied under the greatest possible tension. The bandage needs reapplying several times a day. One of the disadvantages of the pylon method is that the socket, as ordinarily made, fits the shape of the stump as it is and not as one hopes it to be, so that although the stump shrinks it does not shrink to the right shape.

The first post-operative task, therefore, is the preparation of the stump; the second is the preparation of the patient. After an amputation the muscles of the stump, deprived of their natural function, waste and lose their physiological connexion with the central nervous system. Moreover the joint above the amputation is apt to be held flexed and to become fixed in that position. The muscles must be strengthened, and employed in order that the amputee does not forget their existence. I consider this treatment—rehabilitation it may be called—most important. The patients are taught to exercise their stump muscles against the resistance of a weight working over a pulley. In an above-knee amputation, particular attention is paid to the hip extensors and the hip adductors; in a below-knee amputation, to the quadriceps. Also, the patient is made to move the joint above the amputation fully in all directions. Massage is deprecated, partly because it irritates the cut nerves, but chiefly from a psychological standpoint; the patient should learn to do something for himself and not rely on something done for him.

The patient is encouraged to move his stump voluntarily as soon as he will, and not to hold it immobile and inert; and of course the stump should not be propped up on a pillow. As soon as the wound has healed, the patient is sent to the gymnasium where in concert with others he exercises the stump muscles, and after the exercise he is shown by the masseur how to bandage the stump correctly.

The sooner a limb can be fitted the better, since it shortens the time during which the crippled limb is out of action. It is usually possible to fit an artificial leg in three months from the healing of the wound, and an artificial arm in six weeks. Shrinkage may not be complete by this time and the original socket may ultimately be too big, but it is better to spend extra money on a new socket than to delay the fitting of the limb.

Teaching the patient to use his limb.—When the patient has at last been fitted with his limb it is the surgeon's business to supervise his first attempts at walking. At present this is being done by the fitter, who cannot be expected to know much about the mechanics of walking; and the patient so frequently acquires faulty habits in learning to walk. The patient should be returned to us when he is "passed out" with his limb, for we do know that it is absurd to try and walk without being able to stand on one leg, and we do know what muscles the patient should activate. The aim is to get the amputee walking without a limp—an aim easy to achieve if the patient is properly taught.

Cineplastic amputations.—The young surgeon with an ingenious mind is tempted to try his hand at cineplastic amputations. He should remember, however, that (1) the muscle activators have neither sufficient range nor sufficient power to be of practical use; (2) the limb-makers cannot make suitable limbs for cineplastic amputees; (3) all the British patients who had cineplastic amputations after the last war have been reamputated; (4) the Italian surgeons who introduced and popularized the method have given it up.

AMPUTATIONS IN CHILDREN

It is customary to fit children with peg-legs, and to withhold artificial limbs until they have ceased to grow. This is done on the score of expense and because it is supposed that the limb-makers cannot make limbs for children. At Roehampton the limb-makers fit children from the age of 3. The limbs are made telescopic and can be lengthened as the child grows. Psychologically it is important to make a child like other children, and a child fitted with an artificial limb walks perfectly. Moreover once the peg-leg gait is acquired it is difficult to eradicate. Therefore every effort should be made to give children proper artificial limbs.

THE DIFFICULTY OF THE BELOW-KNEE AMPUTATION

Amputations vary in their ease of performance. Both arm amputations are easy to do and may be relegated to a house surgeon, the above-knee amputation is relatively easy and is well within the compass of a registrar, the below-knee amputation is difficult and should only be done by the expert.

Long-arm retractor.—I have found a long-arm retractor useful. It is three feet long and hinged so that it fits inside a sterilizer. The sharp right-angled prong is inserted into the medulla of the bone and an orderly, standing at the head of the patient, holds the handle and raises the limb to the vertical and so exposes to view the raw surfaces. In my experience the retractor does not cause bleeding from the medulla.

The Prophylaxis and Treatment of the Stiff Knee Following Fracture of the Femur. (*Abridged*)

By ROBERT H. YOUNG, F.R.C.S.

THE problem of the fractured femur is the problem of the stiff knee. The fracture itself presents little difficulty, with the exception of those fractures occurring in the supracondylar region, and the upper third of the shaft of the femur. In war time, the problem of the stiff knee is of the utmost importance. A soldier who has had a fracture of the femur is unlikely to return to duty, not because of the mal-union or non-union of the fracture, but because of the disability resulting from the stiffness of the knee-joint.

For most occupations the industrial worker requires a range of at least 90 degrees, and for some he requires a range considerably greater than this in order to do work efficiently. Much time has therefore to be spent on treatment directed towards unstiffening the knee long after the fracture has healed.

The three main factors in the production of the stiff knee are disuse, immobilization, and sepsis. Immobilization of the knee-joint and sepsis may be unavoidable, particularly in certain severe open fractures with loss of bone substance. But in our experience disuse is the commonest and most important cause, and the one most easily prevented. Unless movement of the knee is begun early, within the first six weeks, such stiffness will result that a full range of movement is unlikely ever to be regained, and a useful range will only be obtained after many months of strenuous treatment.

In brief, the method we use is as follows: That part of the sectional mattress of the Pearson bed under the leg is removed. The slings supporting the leg below the knee are then removed. The leg is counterpoised by a weight of about 2 lb. suspended by a sling passing under the leg just above the ankle. The patient then actively bends the knee against the resistance of the counterpoising weight.

Previously, flexion of the knee was obtained by the use of the hip flexors and the aid of gravity. By the present method, however, flexion is obtained by an active contraction of the hamstrings. This automatically produces an active relaxation of the quadriceps, and a greater range of movement is possible. The patient performs these exercises twice a day in addition to quadriceps drill and other exercises to the leg, which are carried out at least three times a day.

We have been able to analyse the results obtained in 34 patients with fractures of the femur. 18 patients were treated by early knee movement, and 16 without early knee movement. Of the 18 patients treated with early knee movement, 5 had transtrochanteric fractures, and an average of 12 degrees of knee flexion at three months; 9 had fractures of the shaft and an average of 90 degrees of knee flexion at 6½ months; 4 had T-shaped fractures into the knee-joint and an average of 115 degrees of knee flexion at 6 months. In sharp contrast, the 16 patients treated without early knee movement, had an average of 35 degrees of knee flexion at 12 months.

A further analysis of the results of early knee movement showed that, if about 35 degrees of knee flexion could be obtained in the first six weeks, subsequent immobilization of the knee by means of a caliper or a plaster spica did not prevent the early return of normal movement. Moreover, normal movement was regained by exercises without the employment of manipulation under anaesthetic, or forcing passive movement.

The treatment of the established stiff knee by exercises, faradism and manipulation under anaesthetic has proved disappointing in our hands. One of the reasons for this is the contracture of the quadriceps muscle. Manipulation, since it is the application of a sudden force, cannot overcome this. Excision of the patella is obviously unsound and will inevitably fail. We have endeavoured to overcome quadriceps shortening by forced passive stretching without anaesthesia, using the application of a large force over a long period of time. The method we use is as follows: The patient lies face downwards on a table or suitable couch. A halter is then passed round the leg just above the ankle, and traction is maintained by a system of pulleys at right angles to the leg. A weight greater than the quadriceps can counteract is used—usually 20-35 lb., and the pull maintained for about fifteen minutes at a time.

We have been able to compare the results of treatment of the established stiff knee in 13 patients. Five patients were treated by faradism, exercises and manipulation under anaesthesia. Eight patients were treated in the same way, with the addition of a forced passive stretching. In the first group, two patients obtained a range of 45 degrees at 18 and 16 months respectively; two a range of 30 degrees at 13 and 10 months; one a range of 10 degrees at 26 months. In the second group, two obtained a full range at 10 and 12 months; one a range of 110 degrees at 18 months; three a range of 90 degrees at 16, 10 and 9 months respectively; two others obtained a range of 45 degrees and 30 degrees at 20 and 12 months respectively and are still under treatment.

Four cases illustrating the main points were demonstrated.

Section of Radiology

President—M. H. JUPE, D.M.R.E.

[June 19, 1942]

DISCUSSION ON THE EFFECTS OF OCCUPATIONAL EXPOSURE TO X-RAYS AND RADIOACTIVE SUBSTANCES

Dr. John R. Nuttall (*abridged*): The development of the use of X-rays and radioactive substances in industry and medicine was accompanied by tragic loss of health and of life. As a result of much careful investigation the International Protection Committee has improved working conditions in the medical field so that workers to-day enjoy a high degree of safety.

Unfortunately, since the outbreak of the present war, evidence has been accumulating that the stage is set for a re-enactment of the tragedies of the pioneer period. X-ray apparatus and luminous paint are again being used in industry. In medical practice there has been wide distribution of portable X-ray diagnostic plants and radiotherapy is being done in small temporary units where the staff is continuously too near the sources of radiation. We should therefore review our knowledge of the dangers, and should consider the means of combating them.

Serious upset of health may arise from the local effects of radiation upon the skin and upon the blood, and also from the ingestion of radioactive substances into the alimentary tract, the inspiration of radon, and damage to the reproductive organs. By far the most important are the blood changes.

Ingestion is unlikely in hospital work because radium there is in sealed containers. It may occur, however, if instruments contaminated with radon are introduced into the mouth.

Inspiration of radon does not appear to present a problem in hospital.

With reasonable care there is no danger of sterility. Menstruation is unaffected. Probably radiation work should be avoided in early pregnancy. On cessation of exposure early reactions of the skin recover but late skin effects remain and may lead to epithelioma.

I should like to mention an observation I have not seen described—the curious phenomenon of acute radiation tiredness. Briefly, after a day's work, normal in ordinary physical and mental effort but during which there has been comparatively large exposure to radiation, the worker complains of undue physical and mental fatigue and irritability. The development of unexpected fatigue is considered in the Manchester Radium Institute to indicate that too much radiation has been received and to call for investigation of exposure. My impression, however, is that it is not constant in all workers, and that it is modified by conditions of temperature and ventilation in the work-rooms.

D. R. Goodfellow has shown that there is only one sign of early over-exposure which is common to all individuals. This is an absolute and progressive leukopenia due to neutropenia, which, if unchecked, will reach a dangerously low level. Individuals vary in their susceptibility to radiation, the more sensitive ones exhibiting absolute lymphocytosis together with absolute neutropenia. In severe cases the curves may intersect.

The old Manchester Radium Institute was small and badly protected, there were several cases of skin damage, and at least one member of the medical staff abandoned radium work with serious blood changes showing the prodromal signs of aplastic anaemia. The average total W.B.C. count of all workers fell to 4,300 with individual low counts of 3,000. Minor infections were frequent and several workers required leave of absence on account of their blood condition. In 1933 the Institute moved to new quarters in which the protection was tested and found to be efficient. Within six months the average total W.B.C. count had risen to 5,500. A rising number of patients involved more treatments being carried out by each member of the staff, and two years later the average total W.B.C. count was 4,400. Additions to staff and re-organization of duties were followed by a rapid rise to an average W.B.C. count of 6,000. At this level it has remained.

This history is instructive. That lack of protection is followed by serious damage to health is a long-established fact, but that "efficient" protection is not sufficient is perhaps less widely appreciated. The policy of short-term service in radiation work is widely employed, but is only applicable to semi-technical workers and nursing staff. Rotation of duties so that an individual is exposed to radiation for a few weeks at a time, alternating

this with some safe duty, may be applied more widely. Coupled with rotation is the conception of dilution of the exposure by spreading the work amongst a large staff, and it is my impression that rotation and dilution are of just as great importance as distance and lead shields. Whilst considering dilution it is interesting to observe that when each doctor was carrying out an average of six radium treatments involving exposure per week the white cell counts were bad. When additions to staff lowered the average weekly treatments to four and a half the average total W.B.C. count rose to 6,000.

This level of about 6,000 W.B.C. is important. Although it is lower than the usually accepted normal (and certainly represents an initial drop in individuals after employment) it does seem to be unattended by ill-effects, to be capable of maintenance while an average amount of work is being done and not to be accompanied by susceptibility to minor infections.

Workers in the X-ray therapy department using completely protected tubes operating at 240 kv. and in the diagnostic department have shown no particular blood changes as a result of their employment. Their working hours, and the protection of the plant conform fully with the Protection Committee's Regulations.

It is obvious that modern technical improvements have made it possible for X-ray work to be safe for all except the culpably careless. It is difficult to see how further technical developments alone can perform a similar service for radium workers other than those engaged in beam therapy. There is room for further regulations with regard to the clinical use of radium, taking into account the finding that up to a point there is comparative safety, but that very little increase in exposure beyond that point is followed by definite leukopenia. In the staffing of new, or enlarged, departments under the Cancer Bill the value of dilution must be taken into account if other centres are not to experience the dangers which befell the Manchester Radium Institute during the period of rapid growth following its removal to new, and apparently ideal, quarters.

Dr. J. C. Mottram : *Mutations produced by radiation in relation to mankind.*—Radiation changes the hereditary function of cells in two ways, by causing chromosomal aberrations and by producing gene mutations. In considering action on men and women, both these should be taken into account, especially as there is some overlap; for instance, small deletions of chromosomes are difficult to distinguish from gene mutations.

Gene mutations were known long before it was discovered by Muller that X-rays produce mutations. They occur spontaneously, their cause being unknown; further, mutations produced by X-rays do not differ in any way from spontaneous mutations. The vast majority of mutations of radiation were previously known. The common spontaneous mutations are commonly produced by radiation, and the uncommon ones rarely. It is indeed a fair statement to make that radiations do no more than greatly increase the incidence of spontaneous mutations.

In the first place, dosage must be considered.

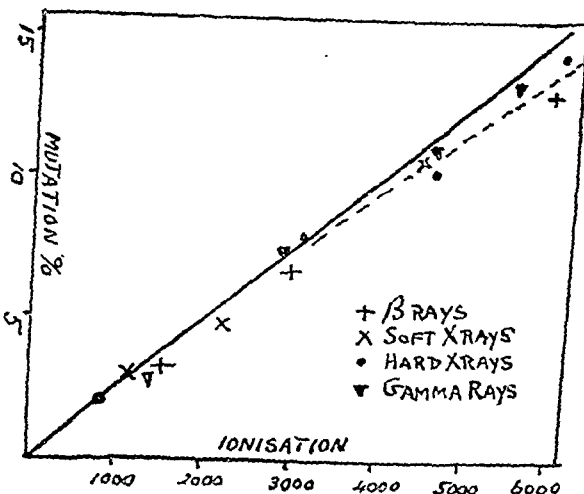


FIG. 1.

Here is seen a linear relationship between ionization and mutation percentage. This is quite different from most biological effects of radiation, such as erythema where a considerable dose is required before any effect is produced. Here the smallest dose will produce

a mutation if, by chance, a cluster of ions is released in a gene. This is important; it means that one can only escape this effect by complete protection from radiation.

We are really concerned with the lowest part of this chart where there are no readings; it has, however, been established that the linear relation extends down to very small doses; there is some falling off from the linear relation at high doses because some clusters of ions will wastefully produce two lethal mutations in the same chromosome.

The chart also shows that there is no wave-length dependence: soft X-rays and gamma rays are equally efficient.

TABLE I.

| Mg. Ra. | Hours expos. | r units | Chromosomes | % mutations |
|---------|--------------|---------|-------------|-------------|
| 300 | 1 | 6315 | 637 | 4.71 |
| 4 | 37½ | 6315 | 636 | 4.71 |
| 2 | 75 | 6315 | 626 | 4.57 |
| 300 | 1 | 12630 | 626 | 9.74 |
| 4 | 75 | 12631 | 622 | 9.64 |
| 2 | 150 | 12627 | 619 | 9.53 |
| 4 | 150 | 25263 | 366 | 20.22 |

Since the relationship is linear and independent of wave-length, it would be expected that there would also be no dependence on intensity. Table I shows this to be the case. It follows that one cannot escape this effect of radiation by keeping the intensity very low. Therefore persons exposed to radiation will show in their descendants, on an average, more mutations than normal persons; they can keep this effect low by protection, but only by complete protection can they entirely eliminate it.

It has been calculated by Pickhan that if the spontaneous rate were due to radiation, a dose of 10-12 r would be required; the spontaneous rate for the fruit fly has been found to be from 1-3 mutations per 100 ova or sperm. It will probably be a little higher in man as there are more genes at stake, so I am taking the figure 3%.

If 10-12 r represents the spontaneous rate, it is obvious the X-ray photography where the ovary might receive 2 r, is of no importance. In screen examinations, the ovary might get 30 r, which would about double the spontaneous mutation rate. It will be seen later that this too is of little importance. The dose required to produce temporary sterilization is about 300 r; this would raise the mutations to 33 per 100 ova or sperm, a limit which, as will be seen, is to be avoided.

Occasionally in treatment the ovaries will receive larger doses, in which case the patient should be warned of the danger to subsequent children. About 1,000 r causes permanent sterility.

For properly protected radium and X-ray workers the dose is small, 1-2 r per week, but this will accumulate to, say, 100 r per year, and 300 r in three years. The life period of sperm is short and a dangerous dose will not accumulate, but this is not the case for ova where a dose of 300 r in three years would, in my opinion, be undesirable. Perhaps there is not this difference since spermatogonia are long-lived.

TABLE II.

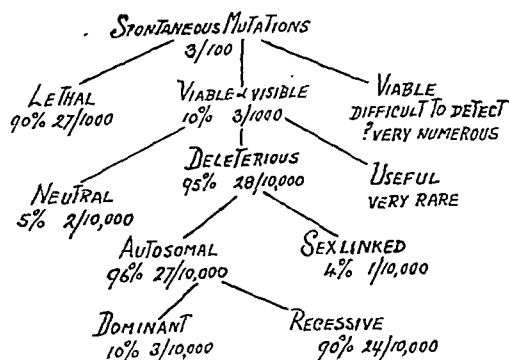


Table II deals with mutations in detail; they are divided into lethal and viable. Some of the viable are visible and easy to detect, but an unknown number are small and difficult to recognize. The figures in the table indicate only approximately the percentages.

Lethal mutations form about 90% of the spontaneous, and will also form 90% of those due to radiation. They will be either dominant or recessive. If dominant, it would only mean, for the person exposed to radiation, that occasionally an embryo would die in early pregnancy; there would still be plenty of normal ova and sperm to make use of. If recessive, the same would hold for some of the radiated person's descendants; how distant, I shall indicate (*see deleterious recessives*). Lethal mutations are therefore of very little importance.

Turning to viable mutations, it is seen that some are neutral, 5%, a very few useful and the vast majority, 95%, deleterious. The useful and the neutral do not, of course, concern us. The deleterious mutations are divided into sex-linked 4% and autosomal 96% (*i.e.* pertaining to all chromosomes other than the sex chromosomes).

The autosomal are again divided into dominant and recessive. Now, from the point of view of a radiated person, these dominant deleterious mutations are important, as they will appear in his children and half the children of an affected child and so on. Likewise the sex-linked mutations will appear in the sons of his daughters, in all, about 4 per 10,000 eggs or sperm for the spontaneous rate; this would be raised to 44 per 10,000 by an exposure of 300 r. I do not think the risk of having one child in 230 bearing a deleterious trait to pass on to descendants should be disregarded from the point of view of the children of radiated persons or of the human race. It is, however, a matter of opinion. The spontaneous rate is 1:2,700.

As regards recessive deleterious mutations, these are much more numerous but will not appear in the children of the radiated person, only in his descendants, and only occasionally in his near descendants, should cousins or other near relatives marry. Muller has calculated that in man, the average time before a new recessive gene would meet a like one and thus manifest itself would be from 750-3,000 years. These figures become 5,000 years for a mutated gene to meet another descended from the original mutated gene. I think that these figures for recessives show that we need not take them into account.

Turning lastly to the small viable mutations: little is known of these, probably they are very numerous, playing an important part in general health, susceptibility to disease, mental well-being, &c. It is known that all living processes are under the influence of heredity as well as of environment; it may be that further knowledge here will necessitate a reconsideration of radiation from that point of view.

In conclusion, it is my opinion that a few hundred r to ova or sperm is a risk to be avoided from the point of view of producing dominant deleterious mutations. I draw attention to the fact that female radium and X-ray workers can easily accumulate such a dose over a number of years.

Professor Sidney Russ restricted his remarks to the harmful effects of radioactive substances. It seemed that the chief dangers in handling arose in chemical and technical work; the danger to people using radium in medical work was avoidable by the practical adoption of straightforward rules of safety. But this was a much more difficult matter when one had to deal with the naked salts of radium; here the chemist had occasionally to run risks, glass tubes containing two to three hundred milligrams of mature radium salts had to be opened and the contents put into solution. A meticulous regard to personal safety should be supported by a working policy which forbade such mass operations to be performed at all frequently by the same person.

The technical operations with radium were mainly those in which the radium salt was mixed with a fluorescent substance and used as an illuminating paint. The operatives, known as luminisers, had received special consideration for their safety. An Order (1942) called the Factories (Luminising, "Health and Safety Provisions") Order had recently come into force. Provided the management at a factory made it their business to see that the excellent provisions for safety in this Order were carried out, there need be little fear of damage to the health of their employees. Even so, in most cases the air in a radioactive laboratory or workshop would contain radon, the occupants would breathe it and during the working day their atmosphere would be a very slightly radioactive one. Some recent measurements proved that an operative under these conditions showed measurable radon in the expired air. Controls upon the workshop air showed less than this amount, and the question arose, whether the radon in the expired air was due to radium in the operative concerned. This was a grave issue, and it showed the need for great care in recommendations about the limits of safety. It might be necessary to suggest one limit for radon in the

air and a different limit for the amount of radium in the body which we might have to tolerate—everyone may have some, bearing in mind the ubiquitous character of this very rare substance, so this should prevent fantastic limits being put forward.

What was the threshold of safety of radon in the air? Professor Russ quoted two medical opinions which had been expressed about these safety limits. The first was a recent private communication from the States: "Medical research workers have determined that a tolerance dosage of radium has been received when a breath sample from the exposed person contains 10^{-11} curie per litre of air." The second was in a paper by Read and Mottram (*Brit. J. Radiol.*, 1939, 12, 54): "... the tolerance concentration (of radon) would seem to be about 10^{-11} curie/c.c." Note that one is a thousandfold the other. One referred to the safety limit of the amount of radium in the body, the other to the concentration of radon that can be safely tolerated in the atmosphere. Professor Russ gave a detailed criticism of the latter, and concluded that for safety it should be recommended that a concentration of radon of 10^{-10} curie per litre in the air for operatives should not be exceeded.

The "tolerance dosage of radium" ("quantity" seemed a better word) was a difficult matter. If we found the suggested safety limit of radon per litre in the expired air (i.e., 10^{-10} curie) and we could prove that this radon was due to radium in the body, then what was this quantity of radium? To deduce this we required to know what percentage of the radon produced per second by such radium was to be found in the expired air. Estimations of this percentage inevitably varied. Read gave the figure 50% for radium which had been recently ingested. Evans had proved a range decreasing from 40% to 2% as the time the radium has been in the body increases. If we took Read's figure for the one type of case and 10% for the other, calculation showed that the quantity of radium in the body would range from 4 to 80 micrograms. These figures at once suggested that, if 10^{-10} curie per litre in the expired air was found by separate tests to be due to the presence of radium in the body, then the operative should be taken off all radioactive work.

If a recommendation were framed in this way we were not committing ourselves to a radium figure that we had no accurate means of estimating, with the advantage that the same figure, viz. 10^{-10} curie per litre of expired air, might serve as a danger signal for an atmosphere of radon and as a much more dangerous sign of a deposit of radium in the body.

Additional data were given about the concentrations of radium and radon occurring in Nature.

Dr. Janet M. Vaughan: *The effect of occupational exposure to X-rays and radioactive substances upon hæmopoiesis* (Abridged).—First, it is necessary to distinguish between the two substances, since radium is more likely to be dangerous on account of the greater penetrating power of the rays. Past records are difficult to interpret, because the distinction is rarely made. Secondly, the possible effect of defective hygienic surroundings other than rays must be taken into account. Thirdly, it is essential to have records of the blood-count in workers before they take up X-ray or radium work, as anæmia may be due to other causes.

Review of the available evidence suggests that external radiations from X-ray or radium are without effect on the red cells or hæmoglobin of workers starting with a healthy blood picture (Aubertin, 1912; Pfahler, 1922; Portis, 1925; Lavedan, 1927; Mottram, 1932; Kaplan and Rubinfeld, 1936; Whitby, 1936. See Table I). It appears also, that provided reasonable precautions are taken, X-rays and probably external radiation with radium are without effect upon the white cell count, provided the worker has a normal count in the first instance (Pfahler, 1922; Portis, 1925; Lavedan, 1927; Kaplan and Rubinfeld, 1936; Whitby, 1936). Certain workers have described an increase in eosinophils and sometimes of monocytes, associated with a leucopenia due to a decrease in polymorphs. (Table II.)

TABLE I.—EFFECT OF X-RAYS UPON RED CELLS AND HÆMOGLOBIN.

| Date | Author | Effects observed |
|------|----------------------------|-----------------------------------------------------------|
| 1912 | AUBERTIN | ... no effect |
| 1912 | BÉCLÈRE | ... decrease |
| 1914 | GUDZENT AND HALBERSTÄEDTER | ... (1) decrease ... (2) no effect |
| 1922 | PFÄHLER | ... no effect |
| 1925 | PORTIS | ... no effect |
| 1927 | LAVEDAN | ... (1) increase ... (2) no effect |
| 1932 | MOTTRAM | ... (1) increase ... (2) decrease ... (3) no effect |
| 1936 | KAPLAN AND RUBENFELD | ... no effect |
| 1936 | WHITBY | ... no effect |

TABLE II.—EFFECT OF X-RAYS AND RADIUM UPON LEUCOCYTES.

| Date | Author | Effects observed on | | | | |
|------|--------------------------|---------------------|-------------|------------|-------------------|-----------|
| | | Total | Eosinophils | Polymorphs | Lymphocytes | Monocytes |
| 1912 | AUBERTIN | (1) increase | increase | increase | | |
| 1912 | BÉCLÈRE | (2) decrease | increase | decrease | | increase |
| 1914 | GUDZENT & HALBERSTÄEDTER | decrease | | decrease | | increase |
| 1919 | RUSS | | | | decrease | |
| 1923 | PFÄHLER | decrease | increase | decrease | relative increase | |
| 1925 | PORTIS | decrease | | decrease | relative increase | |
| 1927 | LAVEDAN | decrease | increase | decrease | | increase |
| 1936 | KAPLAN & RUBENFELD | decrease | | decrease | relative increase | |
| 1936 | WHITBY | decrease | | decrease | | |

Internal radiation with radioactive substances, however, presents a severe industrial hazard and is of particular importance in war time, when the use of instruments with luminous dials is common. Such radioactive substances may be either ingested, as when painters lick their brushes (Martland, 1931; Rajensky, 1939), or inhaled in the form of dust or emanation. The former is more common. Radioactive substances are then absorbed from the intestine and deposited to a large extent in the bones, where they continue to emit alpha rays described by Martland (1931) as "the most potent and destructive agent known to science". The presence of as little as a microgram (Rajensky, 1939) may result in a severe and fatal anaemia. This anaemia, with one doubtful exception, is megalocytic and hyperchromic in type (Martland, 1931). At autopsy, an active regenerating marrow similar to that found in Addisonian pernicious anaemia is present. Certain cases have developed fatal symptoms six to eight years after their last exposure. In this late form death is usually due to sarcoma of the bones, but anaemia may be present and is then of the same megalocytic hyperchromic type.

In order to protect workers handling luminizing paint, a new order has just been introduced called the Factories (Luminising, "Health and Safety Provisions") Order.

Deaths from anaemia following inhalation of radioactive substances have been recorded (Martland, 1931), but are not as common as those following ingestion.

The question of the occurrence of leukaemia in X-ray and radium workers has been much discussed (Nielson, 1932; Rolleston, 1930; Colwell and Russ, 1934; Maingot, Girard and Bousser, 1938; Weitz, 1938). Both myeloid and lymphatic leukaemia are said to have occurred in X-ray and radium workers as the result of their occupation. When the large number of such workers is remembered, the evidence that rays were responsible for the blood dyscrasia is not altogether convincing. It has been possible to trace less than twenty such cases with adequate records.

It is therefore concluded: (1) External radiations from X-ray or radium do not represent an occupational hazard to workers starting with a normal blood picture provided normal and adequate protective precautions are observed. (2) Severe and fatal blood dyscrasias may result from handling radioactive substances, the most common being a megalocytic hyperchromic type of anaemia, dependent upon internal radiation by alpha particles following ingestion by mouth. (3) Individual idiosyncrasy is probably of some importance in the development of blood changes, following exposure to X-ray and radioactive substances, as it is with other occupational blood dyscrasias.

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Section of Psychiatry

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[February 18, 1942]

DISCUSSION ON THE ASSESSMENT OF CRIMINAL RESPONSIBILITY IN THE ARMED FORCES

Lieut.-Col. T. M. Backhouse: The question of the mental responsibility of any person who is accused of a crime is a question of fact to be decided neither by doctor nor lawyer but to be decided, as every other question of fact in a criminal court, by a jury of non-experts representing the ordinary opinion of the country and coming to their decision with such assistance as they may be able to obtain from the medical specialist on the one hand and the legal specialist on the other. Thus, the medical witness may give the greatest assistance to the jury by offering them what is his expert opinion as to the probable state of the person accused at the time he did the act of which he is accused, but still the jury have to find the facts. Equally, the lawyer, in the person of the Judge, Prosecutor, or Defending Counsel, will tell the jury what is the abstract law and assist the jury in applying the law to the concrete facts of the case they are trying, but it remains the responsibility of the jury to decide the question of fact as to whether or not the accused should be held responsible to the State for his action, bearing in mind in reaching their conclusion both the medical evidence which they have heard and the direction on law which they have heard. Any departure from this principle would strike at the very foundation of English Criminal Law.

Shortly, once a person is alleged to have committed a crime the law insists that it is a matter for organized society, in the form of a court, to decide what steps are necessary to protect society, whether the alleged offender be mentally affected or not, and that this cannot be left to the medical practitioner or the lawyer alone.

In civil courts the position is clearly defined and may be taken to be understood by the professional lawyer who guides or assists the court, but considerable difficulties have been encountered by military courts, which usually have no special legal knowledge nor the assistance of a lawyer, in deciding how to reconcile the medical views with the legal. In the civil courts if a person is accused of a crime, after a preliminary investigation by a magistrate he is then committed for trial and tried, if at all, either by a court of quarter sessions or an assize court, either of which would be presided over by a very experienced lawyer who would direct the court on law and in each of which the decisions are made by a jury; and from these courts there is an appeal to the court of criminal appeal. Army procedure differs in a number of ways. If a soldier is accused of an offence there is a preliminary investigation by his commanding officer and a summary of evidence, which corresponds to the depositions in a civil court, is taken. If the commanding officer decides that there is a case to be tried he remands the soldier for trial by court-martial and forwards the charge, together with the summary of evidence, to a senior officer, usually his Brigade Commander, who then decides whether or not he will convene a court to try the soldier. If he decides that the soldier should be tried, he then convenes a court-martial for the trial. In the great majority of cases this court will be composed of three regimental officers with no legal experience and with no legal assistance and this court sits both as judge and as jury. The decisions of this court, however—unless they find the accused not guilty—have no legal effect until they have been considered by a confirming officer, who is normally the same person as the convening officer, and he can refuse confirmation—which would act as an acquittal—or he can reduce any sentence, or remit it altogether, and he can if he so wishes direct that the sentence shall not begin to operate until it has been reviewed again by a superior military authority. After the confirming officer has dealt with the case the proceedings are then reviewed for the first time by a lawyer, a member of the Judge Advocate General's Department, who will advise the superior military authority to whom the proceedings are next referred, on any legal point which may arise, and the superior military authority, usually a Divisional or Corps Commander, may quash the finding of guilty if he thinks fit, or may remit or reduce any sentence or may suspend the sentence. The proceedings are again reviewed by the Judge Advocate General when they reach the War Office for final disposal.

Every person accused of a crime is presumed by law to be sane and legally responsible

for his actions until the contrary is proved by evidence, but the law recognizes three classes of persons as not mentally normal, and each class is carefully defined and in the civil courts a procedure is laid down for their discovery and treatment. All other persons are treated as being fully responsible to society for their actions. The classification is:

(a) Persons unfit to stand trial owing to their being found to be insane after their committal for trial or at the time of their trial. (b) Persons who are insane at the time of the commission of the alleged offence. (c) Mental defectives within the meaning of the Mental Deficiency Acts.

Persons unfit to stand trial owing to mental disability may be subdivided into: (i) Persons committed for trial who before they can be tried are certified to be insane. The Criminal Lunatics Acts, 1884, empower the Secretary of State to remove such person to a lunatic asylum and detain him as a criminal lunatic until he is either committed to prison or discharged. (ii) Persons brought before a court and before any plea is recorded found to be insane by a jury empanelled for the purpose. The issue which the jury is directed to try is as follows: "whether the person is of sufficient intellect to comprehend the course of the proceedings of the trial so as to make a proper defence and challenge a juror to whom he might wish to object and to understand the details of the evidence." In the event of the accused being so found he is in fact treated as a criminal lunatic and the trial of course does not proceed.

Persons found to be insane at the time of the commission of the alleged offence.—If a person is found to be fit to plead the trial will proceed normally but the defence can raise the issue that the accused, although he committed the act or omission constituting the offence, was insane so as not to be responsible according to law for his actions at the time. In this case: "the jury ought to be told that every man is presumed to be sane and responsible for his crimes until the contrary be proved to their satisfaction, and that to establish a defence on the grounds of insanity it must be clearly proved that at the time of the commission of the act the party accused was labouring under such defect of reason from disease of the mind as not to know the nature and quality of the act he was doing or if he did know it that he did not know he was doing what was wrong."

If the jury accept this view they will return a special verdict to the effect that the accused is guilty of the act or omission charged against him but was insane at the time when he did the act or made the omission, and the court will order the accused to be kept in custody as a criminal lunatic until His Majesty's pleasure shall be known.

Mental defectives.—The legal definition of a mental defective is contained in the Mental Deficiency Acts and includes idiots, imbeciles, feeble-minded persons and moral defectives. For the purpose of the above definition mental defectiveness means a condition of arrested or incomplete development of mind, existing before the age of 18 years, whether arising from inherent causes or induced by disease or injury.

Mental deficiency not amounting to legal insanity is not regarded as an excuse for crime but only as a matter to be considered in the question of the subsequent treatment of an offender after he has been found guilty. A mental defective is tried in the same way as any other prisoner but if the court before whom any person is charged with a criminal offence punishable, in the case of an adult, with penal servitude or imprisonment, is satisfied on medical evidence that he is defective within the meaning of the Act, the court may either postpone passing sentence and direct that a petition be presented under the Act, or, in lieu of passing sentence, itself make an order committing the accused person to an institution for defectives or to be placed under the guardianship of some person. A duty is placed on the prosecution to bring before the court such evidence as to an accused person's mental condition as may be available if it appears to them that any person charged with an offence is a defective.

Where the greatest difficulty arises, I think, is in the difference of the meaning of mental defective to a lawyer and to a psychiatrist. I have argued this question with several psychiatrists, and am satisfied that it is the different usage of this phrase that leads to the greatest confusion. To a lawyer there are only three classes of persons, sane, mental defective and insane, and if a mental defective in the medical sense is so defective as to fall within the legal definition of insanity, to a lawyer he ceases to be a mental defective although to a doctor he remains one. This has led to endless argument but I think that if it is once realized that to a lawyer a mental defective simply means a person who falls within the definition of mental defective contained in the Mental Deficiency Acts, and who is not so defective as to fall within the classification of insanity, the position becomes clear, and during the rest of this paper I use the phrase "mental defective" purely in its legal sense.

To a certain extent the considerations affecting the decisions as to whether a person should or should not be tried by court-martial are dissimilar from those to be considered

in deciding whether a civilian should be tried before the civil courts. A military court is concerned with the maintenance of military discipline and a military convening officer is entitled to take into account many questions of convenience, expedience and expense which are not the concern of the civil authorities under similar circumstances, and to say, if he considers that it is unnecessary for the purpose of military discipline, that he will not waste the time of military personnel in trying a soldier even though he be guilty of a civil offence.

Military procedure in respect of persons unfit to plead or insane at the time of the commission of the offence is precisely similar to civil procedure. Mental defectives, however, are not dealt with at all under either the Army Act or the Rules of Procedure, but as I have already explained there are two further stages in military procedure which are missing in civil procedure, namely, confirmation and review with the powers of remission and suspension inherent therein and of course the final power of discharge from the Army.

It will be seen from the above that in law only insanity is the concern of the court before the question of guilt is determined. Mental deficiency is never a defence but merely a matter to be taken into account in deciding on the treatment of a delinquent after conviction. The following principles therefore follow: (i) Both insanity and mental deficiency are matters which concern the convening officer when deciding whether or not to convene a court-martial. (ii) Insanity only and not mental deficiency concerns the court when deciding whether a soldier is fit to stand his trial; (iii) Insanity only concerns the court prior to a finding of guilty; (iv) Mental deficiency may concern the court, subject to what is suggested hereafter, when determining the sentence of the court; (v) Mental deficiency is always the concern of the confirming officer when considering the subsequent treatment of a soldier found guilty by court-martial.

A recent Army Council Instruction lays down that the unit medical officer must give a certificate as to both the physical condition of the soldier and also his mental condition before application for trial is made, and if either he or the soldier's commanding officer considers that the soldier's mental condition should be investigated, he will obtain a psychiatrist's report. The psychiatrist's report goes to the convening officer with the application for trial, and in this report is set out the answers to a series of questions giving his definite opinion as to whether a soldier is fit to plead within the meaning of the legal definition, whether he was, in his opinion, at the time of the commission of the act insane within the definition laid down in the McNaughton Rules, and further, even though he thinks he is both fit for trial and sane at the time of the commission of the offence, his views on his general mental condition, and the effect which might be expected to result from trial and/or punishment, and lastly his fitness to continue to serve in the Army.

If there is any suggestion that the soldier is insane or mentally defective, the case together with the report is submitted to an officer of the Judge Advocate General's Department, and after receiving his advice the convening officer can then decide whether in the light of the information disclosed in the report it is in his view necessary for the purpose of discipline to convene a court-martial. If a soldier is reported to be insane, plainly no court-martial would be convened until he had been before a medical board, and the question of his insanity decided by the board. If he is reported to be mentally defective it would then be a matter for the convening officer to decide whether to apply for his discharge under the provisions of King's Regulations 390 or whether it is desirable that he should be tried and the question of discharge considered at a later stage.

If trial is once ordered by the convening officer and at the trial either the prosecutor or the defending officer raises the issue that the accused is unfit to plead, evidence is called and the court hears and decides the issue on the evidence brought before it and it should not accept reports or other inadmissible evidence.

If the defending officer raises the issue of insanity at the time of the commission of the offence the court should proceed strictly on the evidence and should not accept any medical reports or other documents inadmissible in evidence, bearing in mind that it is for the defence to prove the insanity.

In the event of a finding of guilty the court should accept, if the defence wish to put it forward, any evidence of mental deficiency and for this purpose they may properly accept a report from a psychiatrist *de bene esse* and take this evidence into consideration together with other factors in deciding upon their sentence.

When the proceedings are forwarded to the confirming officer, this officer then considers, taking into account the medical reports before him, whether or not he should recommend a suspension of the sentence pending an application for the accused's discharge, or whether he should allow the soldier to proceed to the detention barracks in the ordinary way, drawing the attention of the commandant to the soldier's mental condition in accordance with King's Regulations 691 (c) and notifying this

for his actions until the contrary is proved by evidence, but the law recognizes three classes of persons as not mentally normal, and each class is carefully defined and in the civil courts a procedure is laid down for their discovery and treatment. All other persons are treated as being fully responsible to society for their actions. The classification is:

(a) Persons unfit to stand trial owing to their being found to be insane after their committal for trial or at the time of their trial. (b) Persons who are insane at the time of the commission of the alleged offence. (c) Mental defectives within the meaning of the Mental Deficiency Acts.

Persons unfit to stand trial owing to mental disability may be subdivided into: (i) Persons committed for trial who before they can be tried are certified to be insane. The Criminal Lunatics Acts, 1884, empower the Secretary of State to remove such person to a lunatic asylum and detain him as a criminal lunatic until he is either committed to prison or discharged. (ii) Persons brought before a court and before any plea is recorded found to be insane by a jury empanelled for the purpose. The issue which the jury is directed to try is as follows: "whether the person is of sufficient intellect to comprehend the course of the proceedings of the trial so as to make a proper defence and challenge a juror to whom he might wish to object and to understand the details of the evidence." In the event of the accused being so found he is in fact treated as a criminal lunatic and the trial of course does not proceed.

Persons found to be insane at the time of the commission of the alleged offence.—If a person is found to be fit to plead the trial will proceed normally but the defence can raise the issue that the accused, although he committed the act or omission constituting the offence, was insane so as not to be responsible according to law for his actions at the time. In this case: "the jury ought to be told that every man is presumed to be sane and responsible for his crimes until the contrary be proved to their satisfaction, and that to establish a defence on the grounds of insanity it must be clearly proved that at the time of the commission of the act the party accused was labouring under such defect of reason from disease of the mind as not to know the nature and quality of the act he was doing or if he did know it that he did not know he was doing what was wrong."

If the jury accept this view they will return a special verdict to the effect that the accused is guilty of the act or omission charged against him but was insane at the time when he did the act or made the omission, and the court will order the accused to be kept in custody as a criminal lunatic until His Majesty's pleasure shall be known.

Mental defectives.—The legal definition of a mental defective is contained in the Mental Deficiency Acts and includes idiots, imbeciles, feeble-minded persons and moral defectives. For the purpose of the above definition mental defectiveness means a condition of arrested or incomplete development of mind, existing before the age of 18 years, whether arising from inherent causes or induced by disease or injury.

Mental deficiency not amounting to legal insanity is not regarded as an excuse for crime but only as a matter to be considered in the question of the subsequent treatment of an offender *after* he has been found guilty. A mental defective is tried in the same way as any other prisoner but if the court before whom any person is charged with a criminal offence punishable, in the case of an adult, with penal servitude or imprisonment, is satisfied on medical evidence that he is defective within the meaning of the Act, the court may either postpone passing sentence and direct that a petition be presented under the Act, or, in lieu of passing sentence, itself make an order committing the accused person to an institution for defectives or to be placed under the guardianship of some person. A duty is placed on the prosecution to bring before the court such evidence as to an accused person's mental condition as may be available if it appears to them that any person charged with an offence is a defective.

Where the greatest difficulty arises, I think, is in the difference of the meaning of mental defective to a lawyer and to a psychiatrist. I have argued this question with several psychiatrists, and am satisfied that it is the different usage of this phrase that leads to the greatest confusion. To a lawyer there are only three classes of persons, sane, mental defective and insane, and if a mental defective in the medical sense is so defective as to fall within the legal definition of insanity, to a lawyer he ceases to be a mental defective although to a doctor he remains one. This has led to endless argument but I think that if it is once realized that to a lawyer a mental defective simply means a person who falls within the definition of mental defective contained in the Mental Deficiency Acts, and who is not so defective as to fall within the classification of insanity, the position becomes clear, and during the rest of this paper I use the phrase "mental defective" purely in its legal sense.

To a certain extent the considerations affecting the decisions as to whether a person should or should not be tried by court-martial are dissimilar from those to be considered

in deciding whether a civilian should be tried before the civil courts. A military court is concerned with the maintenance of military discipline and a military convening officer is entitled to take into account many questions of convenience, expedience and expense which are not the concern of the civil authorities under similar circumstances, and to say, if he considers that it is unnecessary for the purpose of military discipline, that he will not waste the time of military personnel in trying a soldier even though he be guilty of a civil offence.

Military procedure in respect of persons unfit to plead or insane at the time of the commission of the offence is precisely similar to civil procedure. Mental defectives, however, are not dealt with at all under either the Army Act or the Rules of Procedure, but as I have already explained there are two further stages in military procedure which are missing in civil procedure, namely, confirmation and review with the powers of remission and suspension inherent therein and of course the final power of discharge from the Army.

It will be seen from the above that in law only insanity is the concern of the court before the question of guilt is determined. Mental deficiency is never a defence but merely a matter to be taken into account in deciding on the treatment of a delinquent after conviction. The following principles therefore follow: (i) Both insanity and mental deficiency are matters which concern the convening officer when deciding whether or not to convene a court-martial. (ii) Insanity only and not mental deficiency concerns the court when deciding whether a soldier is fit to stand his trial; (iii) Insanity only concerns the court prior to a finding of guilty; (iv) Mental deficiency may concern the court, subject to what is suggested hereafter, when determining the sentence of the court; (v) Mental deficiency is always the concern of the confirming officer when considering the subsequent treatment of a soldier found guilty by court-martial.

A recent Army Council Instruction lays down that the unit medical officer must give a certificate as to both the physical condition of the soldier and also his mental condition before application for trial is made, and if either he or the soldier's commanding officer considers that the soldier's mental condition should be investigated, he will obtain a psychiatrist's report. The psychiatrist's report goes to the convening officer with the application for trial, and in this report is set out the answers to a series of questions giving his definite opinion as to whether a soldier is fit to plead within the meaning of the legal definition, whether he was, in his opinion, at the time of the commission of the act insane within the definition laid down in the McNaughton Rules, and further, even though he thinks he is both fit for trial and sane at the time of the commission of the offence, his views on his general mental condition, and the effect which might be expected to result from trial and/or punishment, and lastly his fitness to continue to serve in the Army.

If there is any suggestion that the soldier is insane or mentally defective, the case together with the report is submitted to an officer of the Judge Advocate General's Department, and after receiving his advice the convening officer can then decide whether in the light of the information disclosed in the report it is in his view necessary for the purpose of discipline to convene a court-martial. If a soldier is reported to be insane, plainly no court-martial would be convened until he had been before a medical board, and the question of his insanity decided by the board. If he is reported to be mentally defective it would then be a matter for the convening officer to decide whether to apply for his discharge under the provisions of King's Regulations 390 or whether it is desirable that he should be tried and the question of discharge considered at a later stage.

If trial is once ordered by the convening officer and at the trial either the prosecutor or the defending officer raises the issue that the accused is unfit to plead, evidence is called and the court hears and decides the issue on the evidence brought before it and it should not accept reports or other inadmissible evidence.

If the defending officer raises the issue of insanity at the time of the commission of the offence the court should proceed strictly on the evidence and should not accept any medical reports or other documents inadmissible in evidence, bearing in mind that it is for the defence to prove the insanity.

In the event of a finding of guilty the court should accept, if the defence wish to put it forward, any evidence of mental deficiency and for this purpose they may properly accept a report from a psychiatrist *de bene esse* and take this evidence into consideration together with other factors in deciding upon their sentence.

When the proceedings are forwarded to the confirming officer, this officer then considers, taking into account the medical reports before him, whether or not he should recommend a suspension of the sentence pending an application for the accused's discharge, or whether he should allow the soldier to proceed to the detention barracks in the ordinary way, drawing the attention of the commandant to the soldier's mental condition in accordance with King's Regulations 691 (c) and notifying this

action in the covering minute attached to the proceedings in accordance with King's Regulations 702.

The whole essence of the procedure is to ensure as far as possible (a) that the question of a soldier's fitness for trial is decided by the convening officer with the assistance of the psychiatric specialist and the legal specialist before the court is convened at all; (b) that the question of the soldier's responsibility for the crime is tried on proper evidence at the trial and that only genuine legal insanity is allowed to interfere with a finding of guilty or not guilty; (c) that any question of mental irresponsibility short of legal insanity is properly considered not in the atmosphere of a court-martial but after the trial by the confirming officer again with the assistance of the legal and psychiatric specialists before the soldier in fact receives any punishment.

Another problem is the question of the disposal of a mentally defective soldier who is not certifiably insane. At present he is merely discharged from the Army and left to his own devices, and as often as not re-enlists. I understand that conversations are taking place between the War Office and the Home Office or the Board of Control to deal with this aspect of the problem.

Lawyers have always been ready and willing to have a revision of the McNaughton Rules, and it is the medical profession who has failed to agree upon any new definition. In practice the present definition appears to work very well, and it is left in every case in civil law to a jury of ordinary men, in military law to a court of ordinary military officers, to apply their common sense to the problem of responsibility. In my own experience it is seldom that a man who is really not responsible for his actions is convicted of any offence and indeed it is much more often that a man who is really responsible escapes.

Surgeon Lieutenant-Commander E. W. Anderson, R.N.V.R.: *The assessment of responsibility in naval offenders.*—As psychiatrist to a Royal Naval Barracks it has been my duty for the past year to examine all offenders where doubt exists that the individual was not responsible for his offence in virtue of mental abnormality. Sometimes this disorder is so apparent that the case is referred by the authorities before disciplinary action is taken, in others the plea of mental disorder is raised by the individual himself when charged. In the latter case the man is informed that he may have the benefit of a psychiatric opinion. In some cases the plea is, often obviously, a mere pretext and the alternative of a period in cells is preferred to exposure as a humbug and the possible gibes of his mates. Optimism, however, is a striking characteristic of the naval rating and a number of bogus cases reach me in the hope that punishment may at least be mitigated. The psychiatrist has no harder task, nor any greater responsibility than the assessment of the imputability of offenders whether Service or civilian. The degree of care and thoroughness must be as great in each. There are, however, certain circumstances which would appear to modify the doctor's attitude to the Service case, an attitude which may again be slightly modified according to whether the offence occurs in peace or in war. Many civilian practitioners called up for service especially those accustomed to private practice, do not appreciate that the only concern of the Service with regard to the medical treatment of an individual, is "can this man be made fit for service or not?". In the latter case he is of no further interest to the Service and must be invalidated. In warfare, especially total warfare, this attitude is heavily underlined. Under present conditions, especially in the Services, the individual is of much less account than the community. That it is this doctrine we are supposed to be resisting with all our strength has been a frequently remarked paradox.

This position of the individual is of significance with reference to "Crime". We must be clear for practical purposes whether responsibility exists in a given case, or is diminished as the result of a morbid mental state. We are thus concerned with what is called psychological responsibility. Any definition of imputability, as Kinberg (1941) has recently pointed out in a distinguished analysis, is unsatisfactory. We are accordingly thrown back for guidance on a detailed and intimate knowledge of psychiatry. The greater our knowledge of psychiatry and psychopathology, and, one might add, the more we know of men and women, the more likely are we to give a correct judgment.

Certain differences in general between Service and civil crime must be considered. The former concerns as a rule offences which are on a different plane from many of those on which we are asked to advise in civil practice. Thus desertion is a very different matter from rape or crimes of violence. Desertion appears a technical offence and in peace time might seem to be entirely so, but since the function of a fighting service is the preparation for war, and the maintenance of a strict discipline is necessary to this end, desertion, even in peace time, cannot be regarded merely as a technical offence. Even in peace, the penalty must be severe, but in war, where the whole community is in danger it acquires a greater moral significance, both on account of its possibly graver consequences and because of the increased sensitiveness of public opinion towards any purposive hindrance

to the war effort. In war, the orientation not merely of the executive, but also of the psychiatrist where crime is concerned, must be persistently towards the community and the individual's claims must be assessed in this light. In assessing the responsibility in the individual case, the corrigibility of the offender and the deterrence of others are questions which seem to have a sharper and more immediate significance than in civil practice. True, civil punishment has both a corrective and a deterrent function. It is, however, more general and abstract in its operation. In a ship or establishment, a number of men are grouped together in close contact and knit by firm discipline. If a man offends and is punished all the others will hear the details of the offence and the punishment awarded. Any laxity in dealing with offenders is likely to be followed by an increase in crime. Whether or not an individual punished for embezzlement in Plymouth will deter another about to attempt this in Aberdeen is doubtful. One offender punished for desertion in a ship may deter 50 others. In general, Service punishment would appear to be successful in its aims.

It is therefore essential that the psychiatrist should not create a breach in this system, that he should not form a weak link in the chain, in particular out of consideration for the welfare of the individual, as against the community. He must decide in any given case (1) whether, even if psychiatric disorder exists, punishment is likely to deter him from repeating the offence; (2) what the effect on general discipline is likely to be if this man escapes punishment; (3) is punishment likely to make his state worse and thus make him less likely to be an efficient rating, or will it have lasting evil consequences to himself? Clearly in the case of a self-reproachful depression the first requirement might be satisfied; the effect on general discipline of the punishment of an obviously sick man is likely, if anything, to be bad, and with regard to (3) it is at least not likely to improve his state. This is an extreme and absurd instance.

It is, however, otherwise with many psychopaths. Many of their mates regard them as responsible and failure to punish them would have in time an adverse effect on morale as well as bringing psychiatry into contempt and disrepute among all ranks. The psychiatrist in a barracks should enjoy widespread confidence, and his work should be free from the suspicion of sentimentality and hocus-pocus. Punishment in many of these cases is likely to succeed in the first aim, i.e. deterrence, and the individual's condition is not likely to be made worse by punishment. I am strongly of the opinion that, in the case of many hysterical offenders punishment should precede treatment. In this way, the ground is cleared for future therapy and one motive for the reaction is removed.

To some of us there appears to have been an unduly tolerant attitude to some forms of psychopathy in recent years, due perhaps to the flabby and uncritical acceptance at face value of certain psychopathological doctrines and perhaps also to a greater slackness in our moral fibre in the inter-war years. In psychotherapy the individual's comfort seems to be given more thought than that of his entourage which is often regarded with indifference. Thus it has been sought to take the "stigma" from hysteria. That this may be undesirable, for example, in certain cases of "effort syndrome" has been shown by Paul Wood (1941). The Service psychiatrist will do well to see to it that the way of the hysteric shall be hard and the profit be withdrawn from psychopathy. In one year of 662 patients were seen in the Psychiatric Department. Of these, 56, or approximately 8.5% were offenders. The series included two officers. 76% of the offences concerned desertion and leave breaking. Six showed no psychiatric disorder.

Hysterical and affective reactions were associated with desertion more frequently than other forms, but the rather low incidence of hysterical states and reactions was surprising, for the hysterical deserter is usually regarded as the prototype of the psychopathic Service offender. Affective states were, however, actually as common. In 28 (50%) of the cases, no interference with punishment was recommended. In another 5 it was recommended that punishment might be modified.

A useful case, illustrating some points in the assessment of responsibility is that of a man, a supply P.O. aged 25, who was charged with having created a disturbance, and of having damaged certain property. The previous evening he had drunk 4 to 5 pints of beer between 5.30 and 10 p.m. It was stated that he had conversed normally at 10 p.m. He then went to the clothing store where he slept and after that did not remember anything of what happened. He said "my mind sort of went blank". He recalled that, soon afterwards, he had seen the damage he had done and remembered the escort arriving to take him to cells. He said "everything was quite ordinary afterwards". He understood perfectly what he had to do and say. He was not charged with drunkenness. He had no memory of the period between entering the store and seeing the damage he had done. He stated that the amount of alcohol was for him not excessive. He gave a history of one previous episode of violence after drinking during which he had

assaulted a policeman. The quantity of alcohol was said to have been greater on this occasion. As a child he had shown several psychopathic traits. His father had been a periodic drinker and his mother had had a depressive illness after his father's death. This man was recommended for invaliding which was carried out. It was considered that punishment in this case would have little deterrent effect since it was improbable he would abstain, and that having regard to his history and the likelihood of similar behaviour again perhaps after a small dose of alcohol, he was unlikely to be suitable for further service. The effect of his invaliding on morale was probably good, since the man was a senior rating and was deeply perturbed at the decision. His mates of equal seniority with good prospects in the Service probably also regarded the matter with concern. Also in view of the relatively small amount of alcohol taken and the unpredictability for him of its effects, he could not be regarded as responsible for having offended through his own negligence.

The hysterical amnesic reactions as a rule, are of the thinnest, and it is usually possible, by the ordinary methods of the interview, to demonstrate in nearly all cases their partial character. This amnesia is of course a situational reaction and as such implies some degree of realization of wrongdoing. The patients very often remember a good deal of what they did during the alleged blank period. One's impression is that these amnesiac are mostly more spurious than those met in civilian practice. They demonstrate, often nakedly, the shifting character of the boundary line between conscious deception, i.e. malingering, and deception which is less conscious, if this phrase may be permitted. This is most evident in hysterical pseudo-dementia. These individuals often show an extreme capacity for self-deception and considerably less for the deception of others. Such people are nearly always second-rate personalities, often of poor intelligence with unsatisfactory work records, a poor "health conscience", and a lifelong tendency to evasion, but who have, up to date, shown no psychopathic episodes. They are shiftless and take the short view. They readily fall to the temptation to quit. Desertion is for them the easiest of all offences. In civil life, if they have shirked their responsibilities, work or family, the penalty for them has often been not so severe or at least immediately not so painful. In short they have got away with it, at least for a time. Many first offenders in the Service, particularly new recruits, are unaware of the rigours of the punishment which awaits them for desertion. Some are, indeed, outraged at the use of the word to describe their absence. Many of these when punished receive for the first time in their lives an immediately unpleasant and uncomfortable award and because they are so egocentric and susceptible to such discomforts, the effect of punishment is at least well worth trying. The problem of the habitual offender is different. In many cases it was unfortunate that they should have been accepted for the Service. Being in, however, there was nothing else to be done. Invaliding was undesirable since this was often just what they wanted and would react unfavourably on morale. Other modes of getting rid of them were equally undesirable. The problem of disposal will be referred to later.

Hysterical pseudo-dementia in my experience is rare in civil life. In eleven years I remember seeing only 4 cases. I have occasionally seen in civil life delinquent mental defectives who showed a pseudo-demential colouring to their oligophrenia. During two years in the Navy I have seen at least twice as many cases. Two of these have been recorded elsewhere (Anderson, 1941; Anderson and Mallinson, 1941). Despite careful study of this reaction I have been unable to make any essentially new observations. Most of them correspond to the classical descriptions, e.g. that of Wernicke, and Stertz (1910). They have occurred in people of low intelligence and social level who in several cases gave a history of head injury. The theoretical problems raised by this reaction are important and fascinating, but it is with its practical and medico-legal aspects we are concerned here. I mentioned earlier that the estimation of the degree to which conscious participation enters into the genesis of these states was extremely difficult. It is practically impossible. One may think that this individual's reaction has more humbug about it than the other, but no more than that can be said. With most of them this element of humbug was strong. Most psychiatrists of experience would agree that feigned insanity is a rarity. I cannot recall ever having seen a case of pure simulation. Where the suspicion of simulation was strong I have never felt able to make a charge of malingering. This view has weighty support, e.g. by Kraepelin, Bleuler and Bumke. Bumke (1936) for example states: "The differentiation of psychogenic from exaggerated or pretended symptoms is fundamentally impossible." He goes on to say that the psychological mechanism involved in each is the same and further that even demonstrable simulation does not rule out hysteria. He admits that this conclusion involves a "painful renunciation". Bleuler (1937) states that those who simulate mental disorder are nearly all psychopaths and include some who actually are mentally disordered.

I could give many examples of simulation which illustrate this. Bleuler also reminds us that the mere demonstration of simulation does not in itself indicate that the individual is sane or imputable. He again stresses the impossibility of drawing a dividing line between simulation and disease.

The mentally defective in the Navy form a small group. As to the question of responsibility it is altogether too simple to suppose that because an individual has a mental age of less than 10, i.e. a Binet I.Q. of less than 70, the standard by which, in practice, mental defect on the cognitive side is diagnosed, such an individual is thereby irresponsible. The question is complex; in civil life, the law is not clear on the point. Those charged with carrying out the provisions of the Mental Deficiency Acts for local authorities usually advise one of the modes of disposal under these Acts, for such an individual convicted of an offence. In the majority of cases, this is the soundest and wisest procedure. I have, however, known judges to sentence an individual with a mental age of 8 or 9 to prison, the legal decision pivoting, of course, on the question of responsibility. In one case I have in mind I think that the judge was right. When we turn to Service practice it may be stated as a fact that not a few individuals with a mental age of no higher than 8 are trainable for certain branches, e.g. seamen, and some have served at sea evidently without attracting attention. If there are no associated psychopathic features, many such might make more satisfactory ratings than some of their more intelligent brethren. When, after a period of service such an individual offends, his irresponsibility cannot be assumed without more ado, merely on the grounds of his mental age. It may very well be that, on the long view, such an individual is a risk and should properly be got rid of by invaliding from a Service which makes such exacting demands on the individual as does the Navy, but unless he shows inefficiency either sustained or intermittent, to such a degree that there are frequent or dangerous lapses, I would hesitate before recommending that he should be invalided. If therefore it is decided to do nothing against retaining him, and even in certain cases where it is the question of his responsibility and ultimate punishment for an offence must be as carefully weighed as with any other offender referred to you. In some cases this elementary knowledge of right and wrong is present, and they have demonstrated in the past their capacity to obey, even under temptation to do otherwise. If this is so, and no psychopathic features, e.g. morbid anxiety, exist, their responsibility for the offence is very likely great enough to merit punishment. Naturally, it is impossible to lay down general guiding principles. It may very well be felt that sufficient opportunity to offend has not existed up to date, and that the individual concerned has been finally faced with a set of circumstances beyond his intellectual grasp and has taken the short way out. If this is so, then he has proved himself, in virtue of his mental defect, incapable of effective service, and since a similar set of circumstances may easily occur again, the proper course is to invalid him. This is the more usual train of events, but I must make it clear there are exceptions. Even when it is decided to recommend a defective offender for invaliding, you may in virtue of his history to date including the offence, i.e. on a basis of fact, have arrived at the opinion that he is responsible. On the other hand, your belief that he is unlikely to be of further use to the Service is more tenuously grounded, i.e. is essentially a prediction. That again the influence on morale has a strongly determining part in the decision to punish is inescapable.

Lastly mention may be made of the affective changes which are reported in those who have been exposed to cataclysmic happenings, e.g. earthquakes (Bälz, Stierlin), and heavy prolonged enemy action. Thus, Larkworthy (1941) points out that in such circumstances men may be absent from their place of duty or slow in obeying orders and thus be unjustly blamed. In such cases, a remarkable apathy is the change described (affective stupor). The men tend to sit about and to take no interest or part in their surroundings. I have described a similar apathy in those who had been exposed to blast, where it seems it may sometimes last for months.

On the question of disposal of the habitual offender, punishment has proved ineffective in these cases and they are a continual liability or even menace. As far as this Service is concerned up till a month or two ago, the practical alternatives were invaliding, an obviously undesirable course, discharged "Unsuitable" which is regarded with disfavour as a way out, or discharged "Services no longer required" (SNLR). This method is a drastic one since it constitutes a life-sentence. Such a man becomes unemployable by any reputable employer for the rest of his life, and is marked ineligible for unemployment benefit. Thus the difficulties in the way of his rehabilitation are considerable. That, on occasions, it has its advantages in respect of certain types of offender is not denied, and it is not suggested that the category should be abolished because, amongst other things, its existence has a probable deterrent effect. But many habitual offenders may be, and perhaps more often than we think are, corrigible. It seems unjust therefore not to offer such men the opportunity to mend their ways. For this purpose a Labour

Camp has been instituted to which the apparently incorrigible offender and many others, not always offenders, may be sent. The man will go there as a naval rating and be subject to ordinary naval discipline and will work for the common good. The camp has a naval medical officer with psychiatric experience. Since the conditions at this camp are designedly strenuous, it may be expected that a number will, after a longer or shorter period there, prefer to do their duty in the normal way. It seems to me this institution must necessarily have a penal character for which no justification need be offered since judicial punishment at the present day, and increasingly so, has also a therapeutic aspect, perhaps drastic, but by and large, effective. It is in short "corrective". It is too early yet to have ascertained even preliminary results from this interesting experiment, which may well offer guidance after the war in the problem of the disposal of certain types of psychopath.

At present some offenders sentenced to longer terms of detention must do their punishment in civilian gaols. This is an unfortunate state of affairs which has already drawn protest from Lady Astor in a recent letter to *The Times*. The naval correspondent of that newspaper replied shortly afterwards stating that separate detention quarters for naval offenders were being provided. That Service offenders are out of place in an ordinary civilian gaol was also remarked to me recently by the senior medical officer of one of our larger prisons.

In this paper it has been sought to show that in the assessment of the responsibility of naval offenders there is an inevitable shifting of the stress from the individual to the community, yet the rights of the individual are as carefully regarded as is compatible with the necessarily restrictive framework of present-day conditions. The general background of this approach has been described and certain principles which have at least guided me have been outlined. No doubt, individual temperament and character condition to some extent our general attitude to the problems, what must guide our particular approach to such cases is an objective scientific psychiatry, our knowledge of which in many instances will inevitably not be deep enough. This knowledge is the decisive weapon for our task. This is essentially the view of Kinberg, who adds in the article already mentioned that "no jurisprudential philosophical speculations on so-called imputability are needed . . . the conception of imputability is a false abstraction, an inanimate conception, incapable of development which must be cancelled".

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Major E. A. Bennet, R.A.M.C.: In the career of an alleged culprit there are several points at which it may be questioned whether or not he is to be held mentally accountable for his actions. Three of these are of special importance:

(a) When a commanding officer has to decide whether or not he will apply to the convening officer for the trial of a soldier under his command by Field General Court-Martial. (b) When a convening officer decides whether a person should or should not be tried by court-martial. (c) When an accused person is before a military court.

The assessment of mental responsibility appears to be governed by different criteria according to the stage reached in the proceedings. Once a prisoner is before a court a rigid principle in assessment of responsibility must be followed. But this need not apply at the earlier stages. Therefore it will be convenient to consider the question of the assessment of mental responsibility (1) before and (2) after a soldier appears before a military court.

(1) Before a soldier is sent for a court-martial, the assessment of mental responsibility for his act or omission can be considered on a wide, common sense basis. It is recognized that many offenders can be dealt with apart from a court-martial, in a manner which cannot injure military discipline.

Persons suffering from certain degrees of mental defect are likely to fall under this heading. It has been found "that a disproportionately large fraction of the 'population' of military prisons and detention barracks is composed of men below average intelligence and that there is a disturbing incidence of men who are dull and backward."

It is known that many of these persons have fallen into delinquency because they have been given work beyond their capacity and, in consequence, they have become discouraged and emotionally disturbed.

A recent Army Council Instruction outlines the procedure which should be adopted in dealing with delinquents in the dull and backward group. This procedure is apparently working very well. In the future, it is hoped, no dull and backward person "shall escape appropriate classification" (*ibid.*) and investigation.

The assessment of mental responsibility in this group is in the hands of a commanding officer or of a convening officer. Their decision, inevitably, is influenced by the result of selection tests, if known, and by the report of a military psychiatrist.

Another section of persons also falls into this pre-court-martial group, namely those thought to be suffering from psychoneurotic ailment. Unfortunately there is no procedure to deal with this group. A procedure similar to that now used for the dull and backward would be valuable. The vast majority of psychoneurotics should be sent for trial. But there are many exceptions. The sorting of this group is a difficult matter requiring common sense, psychiatric experience, and an appreciation of the part the disposal of such a delinquent may play in the discipline and morale of his unit.

When a careful psychiatric investigation, which should consider appropriate corroborative material, reveals longstanding hysterical ailment in a person who puts forward loss of memory with wandering as an excuse for his misdeed, then it would seem that it is a waste of public money to make application for a court-martial. With similar reservations, an act committed under the unconscious motivation in an obsessional neurosis might call for treatment in hospital rather than for a court-martial. Other conditions which so affect the mind as to reduce responsibility to partial responsibility, are found in confusional states, in schizoid episodes, and in certain classes of persons afflicted with recurrent endogenous depression. The use of the term "partial responsibility" is not unknown in the criminal courts in Scotland. There would seem to be excellent reasons, from a psychiatric point of view, for the use of this concept in the assessment of mental responsibility in the armed forces.

(2) The situation is entirely different once a soldier appears before a court-martial. Provided he is fit to plead and to stand his trial, and assuming that he knew what he was doing at the relevant time, and knew that it was wrong, then he will be convicted provided a case be proved against him.

The "test" here stated, and immortalized in the McNaughton rules, leaves a court no option in the assessment of mental responsibility. There is no halfway house between full responsibility and a criminal lunatic asylum.

That this "test" is not uniformly satisfactory in practice is borne out by the spate of cases in which the interpretation of the McNaughton rules has been disputed.

The McNaughton rules were drawn up in 1843, when psychology, as we know it to-day, had not been born. Consequently we get in the rules certain notions which no one now accepts—such as the concept of partial insanity, reminiscent of the long abandoned faculty psychology. The law cannot operate in academic detachment and apart from human beings. Bad psychiatry, such as that on which the rules are based, must assuredly result in bad law. Efforts have been made from time to time to amend the rules. The most recent attempt appears to have been in 1922; and it is possible that it would have succeeded but for the fact that the proposed alteration failed to obtain official medical support.

The criterion used by a court in deciding upon the degree of mental impairment which frees a prisoner from criminal responsibility is, briefly, whether the prisoner possesses or lacks volition. Would the accused have acted as he did under the eye of the A.P.M.? Psychiatrists argue that there are persons of unsound mental condition who do in fact commit acts, many of them criminal acts, which they detest. They do these acts under urges which are outside their conscious control, and they act, knowing that what they do is wrong, legally and morally. Persons with an obsessional neurosis would fall in this category. The presence of a policeman at their elbow might or might not modify their technique. But it would be simplifying the matter to an absurd degree to argue from this that they possessed the power to avoid the act. The court, representing society, might feel that society had to be protected from such persons and might in consequence commit them to prison. On the other hand, if it could be shown that the person did in fact lack volition, it might be thought that it was a travesty of justice to commit the offender to prison.

Psychiatry in 1843 took no account of mental activity outside consciousness. But psychiatry to-day is well aware of the value of this concept. In fact it is one of the main foundations of modern psychological knowledge. In assessing responsibility, therefore, it would seem that a court should be in a position to consider the view accepted by psychiatrists—that it is possible to distinguish between impulses which are not resisted and those which are irresistible. Clearly it is essential to make this distinction, for every crime, and indeed every act, is done under some impulse and the law sets out to compel people to resist certain impulses.

An act lacking intention could not be described as criminal. Consequently if it could be shown that the act of a person suffering from an obsessional neurosis was committed under the compulsion of an overriding motive, of which the person was unconscious and therefore powerless to resist, that act could not be said to be the result of conscious volition. An individual might be aware that his act was improper and even criminal, and yet it might be that he was powerless to resist the impulse to commit it. It is here contended that such an act lacks volition and for this reason it should not be punishable. The question of the responsibility of a person for an act would be assessed in the particular case by the court. The court being in possession of medical and other reports must decide the question of responsibility in precisely the same way that it decides any other fact and the law assumes responsibility until it is disproved.

Yet if the information given in psychiatric reports indicated that the prisoner, although not certainly insane, lacked the power of control over his conduct, it is not possible, in the present state of the law, for a military court to act on such a report. They must proceed according to the rules laid down ninety-nine years ago.

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Section of Epidemiology and State Medicine

President—E. H. R. HARRIES, M.D.

[May 22, 1942]

Clinical, Epidemiological and Experimental Observations on an Acute Myalgia of the Neck and Shoulders; Its Possible Relation to Certain Cases of Generalized Fibrositis

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Introduction.—An epidemic of "benign myalgia of the neck" was reported in America in 1935 (Massell and Solomon). The disease was described as typical "stiff neck", or acute torticollis of short duration; although in some instances symptoms persisted for several weeks or tended to recur. Four outbreaks of this disease were studied in England during the winter of 1941-42; the clinical, epidemiological and experimental findings are reported here.

Clinical observations.—Analysis of 125 case records of acute myalgia of the neck and shoulders leads to the definition of certain clinical types of the disease. In most instances the duration of symptoms is short and recovery apparently is complete although the persons are liable to recurrences weeks or months later. In a smaller proportion of cases, perhaps 15%, some aching and stiffness persist for weeks, although not enough to cause interference with daily routine. Finally, an occasional patient has become seriously incapacitated; myalgic areas have appeared in other parts of the body and after several months a disease typical of generalized fibrositis has developed. The case-histories of two patients are given in detail to illustrate courses which the disease may follow.

Description of a case of myalgia in its commonest form.—A 25-year-old male chef felt perfectly well until the morning of October 18, 1941, when he noticed a slight pain in the left side of his neck. Within two hours the pain had become so severe that he was forced to stop work and lie down. He was then fairly comfortable, but movement of the head, particularly turning to the right, caused a sharp pain. When seen he held his head very rigidly, turning his eyes or moving his whole body in order to look to one side. On palpation of the neck an area of extreme tenderness was found in the left trapezius muscle about the middle of its anterior free margin. No spasm was detected, and physical examination was otherwise negative. There was no elevation of temperature. Total leucocytes numbered 8,300, of which 38% were lymphocytes. Erythrocyte sedimentation (100 mm. tube) was 4 mm. in 60 minutes. On the next day his condition was about the same; but by the third day he had much less pain and was able to move his head fairly comfortably. On the fourth day he was symptom free. For the next five weeks he remained well. On December 2, without warning, the pain returned and he remained in bed for four days. This again was followed by apparently complete recovery. He went to America in December and has since written that he has had two further attacks similar to those described.

Description of an acute myalgia progressing to generalized fibrositis.—A female laboratory technician, aged 23, awakened on October 15, 1941, with a severe sharp pain in the right side of the neck. This caused her to move her neck and head as little as possible. Examination revealed exquisite tenderness in the portion of the right trapezius muscle adjoining the clavicle; no spasm was felt. On the following day neither the location nor the character of the pain had altered, and she remained in bed. Temperature was normal. Leucocyte count was 9,500, of which 38% were lymphocytes. On the next day it was 11,400, with 34% lymphocytes. Erythrocyte sedimentation was 12 mm. in 60 minutes. The patient remained in bed for five days with persistence of the pain in the neck, although she gradually lost the typical "stiff neck" attitude. On the seventh day of illness the pain disappeared. Ten days later, following a bicycle ride, the symptoms returned. The same area of tenderness in the right trapezius was found to be present. After another six days in bed she resumed her work apparently recovered except for some stiffness in the neck and a "tired feeling" which tended to develop in the shoulders and arms at the end of the day. On November 12, for the third time, the pain in the neck became severe and she entered the hospital as a patient. This time the pain was more extensive, involving the right side of the neck, the right arm, forearm and hand. Areas of muscle tenderness were found in the right trapezius, the right deltoid, and the extensors of the right hand. The pain was referred to areas more diffuse than the corresponding areas of tenderness. A few days later she complained of pain in the right side of the chest; this was not aggravated by coughing or deep breathing. A small point of tenderness was found in the muscles of one intercostal space. During the succeeding weeks the patient often complained of an unpleasant numbness or "pins and needles" sensation in the palm of the right hand. Other myalgic areas appeared from time to time in the upper portion of the right rectus abdominis, the left trapezius and deltoid, along the medial margins of the scapula, and in the hamstring muscles just above the knees. The patient remained in the hospital for thirty-one days. During the first half of that period her temperature occasionally reached 99° F., but thereafter was never above normal. Leucocyte count on November 2 was 7,600, with 24% lymphocytes; on November 18 8,000, with 16% lymphocytes; on December 12 6,700, with 41% lymphocytes. Erythrocyte sedimentation on December 12 was 10 mm. in one hour. She resumed work on December 23, but during the five months since then there has seldom been a time when she was completely free of pain. She complained of occipital headaches, most pronounced in the morning. She has frequently been forced to stop work for an hour or so, and indeed, on four occasions has been unable to work for periods of two or three days. Since November 12 the clinical picture has gradually become indistinguishable from that of generalized fibrositis. A striking effect of this illness has been a personality change from a natural cheerfulness to spells of depression and discouragement.

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by patients. The lack of *systemic disturbance* was often a striking clinical feature. In some cases, however, the patients felt generally ill. One of these had such severe headache that an organic disease of the central nervous system was suspected, and a lumbar puncture was done; the diagnosis became evident later when myalgic areas appeared. No complications such as arthritis were observed. The condition is probably never fatal. *Personality changes*: Periods of mental depression were invariably noted in the severe cases. Discouragement, weeping, lack of co-operation, and sometimes sullenness were manifestations. This behaviour, coupled with such indefinite complaints as numbness in the hands, and with little objective evidence of disease, could easily lead to a mistaken diagnosis of psychoneurosis.

Laboratory data.—The *leucocyte count* was ordinarily within normal limits; there was often a moderate lymphocytosis. *Erythrocyte sedimentation rate* was always normal. *Throat cultures* showed only normal bacterial flora. Several *blood cultures* were made in the more severe cases and were invariably sterile.

Relation to fibrositis.—In 5 of the 125 cases detected in the surveys (2 from the hospital, 2 from the factory and 1 from the A.T.S. unit) the disease has followed a protracted course, and the patients now appear to have rather severe, generalized fibrositis.

Association of acute myalgia with common cold.—51 of the 125 cases of myalgia (41%) gave a history of common cold at the time of the onset of myalgia. This appears to be more than a chance association, but its significance is uncertain. It is worth noting that the symptoms of both conditions are aggravated by exposure.

EPIDEMIOLOGICAL OBSERVATIONS

In October 1941, several cases of "stiff neck" occurred among members of the staff of the American Red Cross-Harvard Field Hospital Unit. All persons in the community were, therefore, canvassed in order to determine the prevalence of the condition. This revealed that a considerable number of persons had suffered an attack of myalgia in the neck or shoulder regions within the preceding two-month period. Similar cases of myalgia were reported in a nearby factory and also among a Unit of the Auxiliary Territorial Service billeted in the neighbourhood. Systematic inquiries disclosed a recent high incidence of acute myalgia in these two groups. A fourth survey was made among a detachment of soldiers. The incidence of myalgia in that community was considerably less than that found in the other groups.

In order to evaluate the apparent high incidence of myalgia in the first three groups of people, the follow-up surveys were made for comparison. In each community more than half of the persons interviewed in the follow-up survey had been interviewed in the original one. Living and working conditions were similar throughout both periods. All studies were completed between October 1941 and April 1942. The weather was consistently cool or cold.

METHOD OF SURVEY

All persons in the communities involved were interviewed by trained public health nurses. The nurses had been instructed in the nature of the disease and in the purpose of the inquiry and had been given special charts for recording their findings. Many persons who had been recently affected with myalgia were also examined by a physician. Questionable cases were excluded, and in the follow-up surveys persons still having symptoms which had begun during the original surveys were omitted. The data are, of course, subject to the usual limitations of the survey method, since reliance must necessarily be placed on the patients' recollection of symptoms. Recent attacks were more clearly remembered than those six or eight weeks past. There was a noticeable tendency for dates of onset to be grouped around the "first", "tenth", or "fifteenth" of a month. Nevertheless the total picture is believed to be fair and comparison of findings in two surveys of a given community is reasonable.

Findings of surveys.—A graphic representation of the case incidence in the first three communities is shown in fig. 2. A total of 84 cases occurred during the original survey periods compared with 41 cases in the later ones. Details of the conditions in these communities are given below:

Auxiliary Territorial Service Unit.—This was a group of young women whose duties were mainly clerical. They lived in a former boys' school, and in seven neighbouring residences. There was relatively little contact between residents of the different houses. The two surveys in this group represent essentially a continuous record, since the interval between the end of the first and the beginning of the second was only two weeks. In the October-December survey 24 cases were found among 177 women, an attack rate of 13.6%. There was some grouping of cases in the first week of December. During the second two-month period only seven cases were identified among 165 women, an attack rate of only 4.2%. During the first survey there was a considerable difference in incidence of the condition among occupants of different dormitories. Table III shows the rates in the

CHARACTERISTICS OF THE DISEASE

The frequency of various characteristics of myalgia and of its association with the common cold is shown in Table I. The site of the pain was in the region of the trapezius muscle

TABLE I.—CLINICAL FEATURES: ANALYSIS OF 125 CASE-HISTORIES.

| | Actual number of cases | Per cent. |
|---------------------------------|------------------------|-----------|
| Pain in neck or shoulders ... | 125 | 100.0 |
| A. Sharp | 32 | 25.6 |
| B. Ache | 73 | 58.4 |
| C. Stiff | 20 | 16.0 |
| Pain on turning head ... | 83 | 66.4 |
| Tender areas | 69 | 55.2 |
| Headache | 60 | 48.0 |
| General discomfort ... | 42 | 33.6 |
| Upper respiratory infection ... | 51 | 40.8 |

in nearly every instance. In some cases painful areas were also present in the deltoid or in the muscles attached to the scapula; the sternocleidomastoid was rarely affected. The common locations of pain are shown in fig. 1, in order of frequency. Often pain was

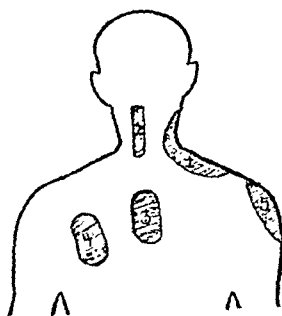


FIG. 1.—Usual locations of myalgia in order of frequency.

more widely distributed than is indicated in this diagram. The two sides of the body were equally involved. Only 55% of persons complained of *tenderness*; however, all patients seen in the acute stage have had definite areas of tenderness and we believe, if looked for, this sign would be regularly present. In many cases "*nodules*" could be felt in the affected muscles. These often could be made to disappear under the influence of heat and massage, and were probably caused by localized muscle spasms. Others however, were more persistent and two consultants who had had wide experience with rheumatic disease, had no hesitation in pronouncing them typical "*fibrositic nodules*". Pain on turning the head depended on the location of the myalgia. It was usually present when the painful area was along the anterior free border of the trapezius muscle (Area 1, on fig. 1). Headache, present in about half the cases, was in some instances the chief complaint. It was usually in the occipital region and was associated with myalgic areas near the cranial attachments of the trapezius muscle. General discomfort, described as an "*aching sensation*", was experienced by one-third of the patients. Fever was not a characteristic of this disease, although in some of the severe cases rises of temperature to 100° F. were occasionally noted. Duration of symptoms, shown in Table II, was less

TABLE II.—DURATION OF SYMPTOMS.

| Duration (days) | Cases | Per cent. |
|-----------------|-------|-----------|
| 1—4 | 52 | 55.3 |
| 5—9 | 16 | 17.0 |
| 10—14 | 11 | 11.7 |
| 15 or more | 15 | 16.0 |
| Total | 94 | 100.0 |

than five days in most cases. In a few instances, as previously stated, symptoms lasted for several months. Interference with normal activity occurred in 44% of the 125 cases, but usually lasted only a day or two. Two of the five patients with severe symptoms have been obliged to give up their work. In addition to these, several other persons continue to have symptoms from time to time and are occasionally partially incapacitated. Recurrences: A characteristic feature of this form of myalgia was its tendency to recur after varying intervals of time. In severe cases this tendency was manifested by a series of exacerbations. No constant inciting factor has been ascertained, although chilling, unusual physical exercise such as cycling, and menstruation have all been held responsible

four largest: 4.3% and 3.1% in two dormitories and only 0.9% in the other two. The distribution of the seven cases found in the follow-up period was such that the rate was not above 0.9% in any dormitory. Because there had been some changing of residences the attack rates quoted were computed on the basis of person-weeks in each residence. Cases were allotted to the dormitory in which the affected person had spent the preceding ten days.

Factory.—A description of this factory and an analysis of the original survey have already been reported (Beeson and Scott, 1941). At that time 29 cases were found among 163 people interviewed, an attack rate of 17.8%. The second survey was made after an interval of thirteen weeks and covered the months of March and April 1942. Out of 179 persons interviewed only 14 cases were found. Thus the attack rate had fallen from 17.8 to 7.8%.

Hospital Unit.—The first survey in this community covered the period between September 17 and November 17. Among 198 persons interviewed there had been 31 cases of myalgia. The follow-up survey disclosed that in March and April there were 20 cases among 149 persons. Thus the attack rate in the whole community had changed only from 15.7% to 13.4%. On further analysis, however, it was found that the incidence of disease fluctuated considerably among the different groups of personnel. In the autumn period the attack rate among the 72 persons who lived and took all their meals in the hospital (doctors, nurses, technicians and orderlies) had been 23.6%, while in the spring this had fallen to 11.6% among 95 residents. In the non-resident group (clerks, domestic helpers, construction workers) the rate was only 11.6% among 126 persons in the autumn, but rose to 27.3% among 33 non-residents in the spring. The findings indicate that the greatest prevalence of the disease shifted during the winter from the resident group to the non-resident group. (Omitted from the first survey were 24 persons who took part in the transmission experiments.)

Soldiers.—Cases of myalgia had been reported among some soldiers in a tank training regiment. A survey was made, covering the months of October and November 1941. Only 17 cases were identified among 278 men, an attack rate of 6.1%. A second survey in this group could not be done. The data obtained in this community are not, therefore, included in the remainder of this report.

History of previous attacks of stiff neck.—All of the 1,031 persons interviewed in the three communities were asked whether or not they had suffered from "stiff neck" in the past. Of the 125 persons who had recently suffered myalgia, 62 or 49.6%, gave a history of previous attacks. This is at variance with the findings of Massell and Solomon (1935), who stated that none of their patients remembered having suffered from this condition before. Of the 906 persons who had not had myalgia within the periods of our surveys, only 201, or 22.2% remembered having had a "stiff neck" in the past. This difference between persons who were recently affected and those who were not, suggests that there may be individual differences in susceptibility to the condition.

Attack rate according to age.—Ages of persons in the surveyed population ranged from 15 to 70 years, the majority being young adults. The attack rates were highest in adults of middle age (Table IV), being lower in young adults and declining progressively with advanced age.

TABLE IV.—ATTACK RATE ACCORDING TO AGE.

| Age-group (years) | Number of persons | Cases | Attack rate % |
|----------------------|----------------------|-------|------------------|
| 15 — 19 | 149 | 16 | 10.7 |
| 20 — 29 | 478 | 52 | 10.9 |
| 30 — 39 | 193 | 29 | 15.0 |
| 40 — 49 | 131 | 19 | 14.5 |
| 50 — 59 | 46 | 6 | 13.0 |
| 60 — 69 | 34 | 3 | 8.8 |
| Total | 1,031 | 125 | 12.1 |

Sex incidence.—Our data are not suitable for analysis in regard to sex incidence because the proportion of males was small and the average age of males was considerably higher than that of females. Nevertheless it may be significant that the attack rate was low in the group of soldiers surveyed, that it was lower in male factory workers than in female factory workers, and that it was low among the construction men in the hospital community. Because of the usual mildness of the disease it is possible that men accustomed to hard manual labour would be less apt to remember an attack of myalgia than would women.

Communicability.—The fluctuating incidence of this condition and the variation in incidence in different groups within the same community are characteristic of an infectious disease. In these studies no evidence of transfer by contact with affected persons was obtained. Many persons who were close associates of patients did not develop symptoms. Conversely, the disease developed several times in hospital patients who had been on isolation precautions for other conditions, and who apparently had no contact with an attendant who had symptoms of myalgia. There is nothing to suggest that the causative

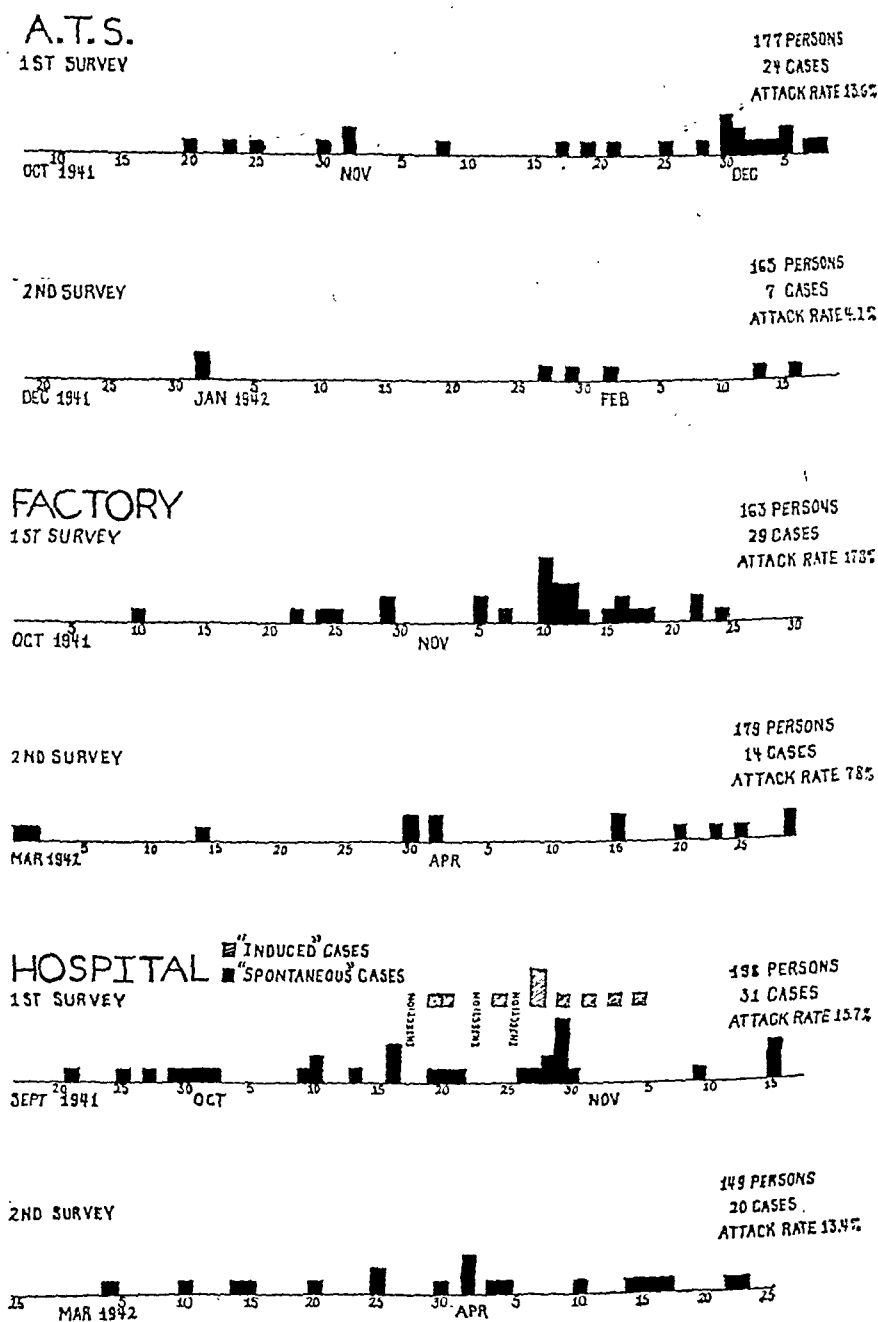


FIG. 2.

TABLE III.—INCIDENCE IN DIFFERENT DORMITORIES—A.T.S. UNIT.

| Dormitory | Person-weeks | Cases | Rate % |
|-----------|--------------|-------|--------|
| 1 | 347 | 15 | 4.3 |
| 2 | 218 | 2 | 0.9 |
| 3 | 210 | 2 | 0.9 |
| 4 | 127 | 4 | 3.1 |
| 5 | 53 | 0 | |

Since myalgia was currently prevalent in the population from which these volunteers were drawn it was impossible to conclude that the disease resulted from the inoculations. Consequently four attempts at transmission were made in communities some distance away and free from spontaneous cases. Blood from patient C4 was used for the first of these attempts. The blood was taken on October 27, the day of onset of symptoms, but was not used until October 30. During the interval three means of preservation were employed: part was stored at 7° C.; a second part was frozen at -10° C.; the third portion was dried by the Cryochem process, the last two procedures causing hæmolytic. Fifteen middle-aged women (Group D) volunteered for the tests. The two hæmolytic portions were given intramuscularly to two groups of five persons, in amounts of 3 c.c. The remaining five received intravenously, 1 c.c. of the blood which had been kept at 7° C. No case of myalgia resulted.

A second trial (Group E) with four of the same volunteers was made six days later using freshly drawn defibrinated blood from patient C5; this had been transported in ice and was administered intravenously in amounts of 5 c.c. within six hours. None of them developed myalgia.

A third trial (Group F) was made on November 20, with five of the original Group D. The blood was obtained from patient A4, who was suffering a severe exacerbation of her disease. The blood was treated as before and 5 c.c. were given intravenously to each, but no case of myalgia resulted.

A fourth trial was made on March 1, with four middle-aged men (Group G, not shown in figure). Blood from a patient in the fifth day of myalgia, treated as in the last two trials was used. 6 c.c. were given intravenously to each of the volunteers but no case of myalgia resulted.

Comment.—The transmission experiment using filtered or centrifuged washings from nose and throat was not impressive, since only one of six persons contracted myalgia, a frequency about the same as that of the spontaneous disease at the time. The results of the first experiments with blood were much more suggestive. Ten of 18 persons, in three successive generations, developed myalgia. This was an attack rate of 55.5% in a period of seventeen days. When the incidence of the spontaneous disease among the 166 susceptibles¹ of the Hospital Unit was calculated for the same period of time it was found to be thirteen cases, or 7.8% (observed difference = 48%. Standard error diff. = 12. Quot. = 4). The incubation periods in seven of the ten experimental patients were from two to four days. In the other three cases the onset was seven, ten, and sixteen days after inoculation. In Group C the results were particularly impressive, since all had myalgia within the succeeding ten days. The experimental disease was identical with that observed in spontaneous cases, except that the proportion of severe and prolonged illnesses was greater. Three of the experimental patients—A4, B4 and C4—have had clinical courses which resembled that of Case 2, previously described. The unusual severity could be due to heavy dosage, or to the great virulence of an infectious agent; this increased virulence could have been due to the intravenous route of inoculation or to rapid passage through a susceptible host.

Certain facts may be cited in explanation of the failure to transmit the disease in other communities. *Group D:* The blood had been stored for three days before use. Under these conditions an infectious agent might no longer be active. *Group E:* Only four persons were concerned, and the blood was from a patient whose symptoms subsided on the following day. Fortuitously in Groups A, B, and C, the donors had been patients in whom the subsequent disease was severe. *Groups F and G:* The bloods used were from patients who had been ill for thirty-two and five days, respectively. It is distinctly possible that an infectious agent might have left the blood in this time.

Attempted Transmission to other Hosts

All efforts to establish the disease in animals, and in the developing chick embryo, were unsuccessful. Portions of the same materials used for volunteers were employed as follows:

| | | | | Mice | Guinea-pigs | Rabbits |
|--------------------------|-----|-----|-----|-------------------|-------------------|-------------------------|
| Nose and throat washings | ... | ... | ... | Intranasally | Intranasally | Intranasally |
| Blood | ... | ... | ... | Intracerebrally | Intramuscularly | Intracerebrally |
| | | | | Intraperitoneally | Intraperitoneally | Intravenously |
| | | | | | Subcutaneously | On the scarified cornea |
| | | | | | Under foot pads | |

Developing eggs were inoculated with blood on the chorio-allantoic membrane and into the allantoic sac. Many "blind" passages were made by these routes and also by the intra-amniotic route, with consistently negative results.

Comment.—No evidence of illness or of pathological change was observed in these

¹ Susceptibles equal total population minus inoculated persons minus those having had the disease in previous month, or $193 - 18 - 14 = 165$.

agent is conveyed by food. The epidemiological characteristics of this disease resemble those of diseases which are spread through the agency of latent or sub-clinical cases. The probable existence of such cases is indicated by the mildness of many manifest cases and by the frequency with which symptoms recur after free intervals.

EXPERIMENTAL OBSERVATIONS

Experiments were performed in an effort to determine the transmissibility of the disease to human and other hosts. Materials used were: (1) washings from the nose and throat, and (2) whole blood.

Transmission to human volunteers.—(1) *Washings from nose and throat:* Normal persons were treated with washings from the noses and throats of patients with myalgia. The washings were first rendered bacteriologically sterile by Berkefeld filtration, or were grossly freed of bacteria by angle centrifugation. The materials were sprayed into the noses and throats of volunteer subjects twice a day for five days. One of six persons so treated developed myalgia thirteen days after the last spraying. This was an incidence no greater than that in the community at large and, therefore, probably lacked significance. Twelve other persons—Groups A and B to be described—were sprayed, in addition to receiving blood. Four of them developed myalgia. Since the attack rate among persons of Group C, who received only blood, was still greater the evidence did not suggest that washings contained an infectious agent.

(2) *Whole blood:* The plan of these experiments is illustrated in fig. 3. In all experiments blood was drawn with an all-glass syringe and defibrinated with glass beads using

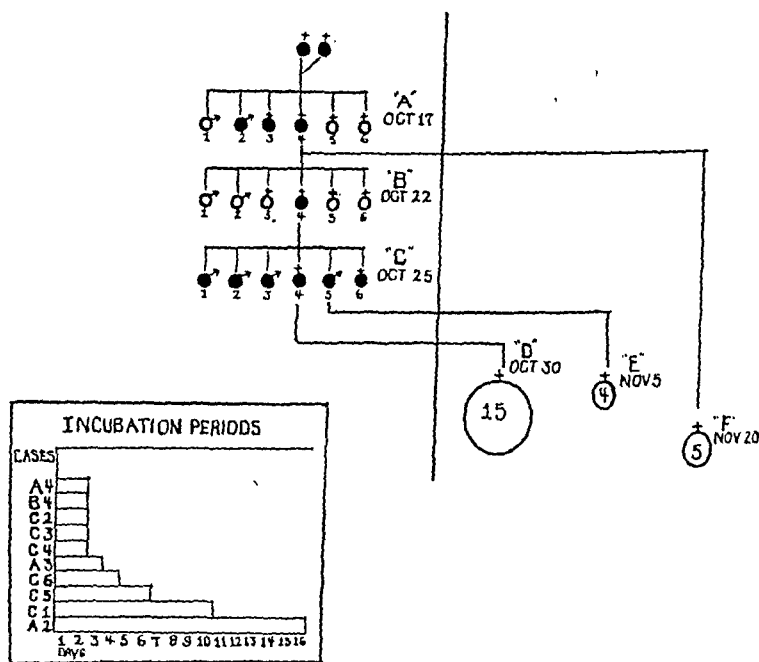


FIG. 3.—Cases of myalgia among volunteers.

sterile precautions. The blood used for the first group of volunteers—Group A—was obtained on October 17 from the two patients who had become ill with myalgia on the previous day. The blood was pooled, both donors belonging to blood group O, and was administered intravenously in amounts of 10 c.c. to each of six normal persons. Three developed myalgia. Patient A4, who showed typical “stiff neck” forty-eight hours after receiving the blood served as the donor for B Group. Each of the six received 5 c.c. of blood intravenously on October 22, and one of the six (B4) had a “stiff neck” forty-eight hours later. About the same time another member of B Group had a mild pain in the neck which lasted only a few hours. Because of the mildness and brief duration of the pain this person was not considered a definite case of myalgia. On October 25, the day after onset of symptoms, blood from patient B4 was administered to the six members of Group C; five received 1 to 4 c.c. intravenously and one received 3 c.c. intramuscularly. All of these developed myalgia within ten days.

Section of the History of Medicine

President—J. F. HALLS DALLY, M.D.

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The Development of Hospital Services with Particular Reference to the Municipal Hospital System of London

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BRIEF EARLY HISTORY OF HOSPITALS—TO 1600(a)

It is consoling to our humanitarian instincts that researches confirm what we would all like to believe, namely, that the care of the maimed and sick is deeply rooted in the early history of mankind. "Doctors" appear very soon upon the scene of life, for example: Solus of Assyria. Egypt, China, India, Greece and Rome all had hospitals. Egypt and Rome had State-paid doctors. The word "hospes" connotes both host and guest and the idea of a "hospital" therefore lies in hospitality, a characteristic of early civilizations. Hence the monastery "hospitium" and "hospice" and the present-day Chelsea Royal Hospital and Greenwich Hospital. At the beginning, the doctors were the priests—thus inaugurating that close connexion between religion and the churches and medicine and hospitals (care of soul and body) which in some forms—such as the Archbishop of Canterbury's power to confer the "M.D. Lambeth", and hospitals staffed by nuns—still continues. Rome had "specialists" (aurists, oculists, dentists) and some of its hospitals were put into the purer air of the country. Bishop Basil founded a hospital at Caesarea (A.D. 370) and Archbishop Chrysostom at Constantinople (A.D. 400).

In Early England—as on the Continent—the Church, especially the religious communities, provided the hospitals, the development of which was stimulated by the need for caring for lepers(b), by devastating periodical plagues (e.g. the Black Death in the fourteenth century), epidemics of cholera, and by the poverty and servitude of the working people, who could make no provision from their own resources. The incidence of leprosy, which already existed in England in the fifth century, was increased by returning Crusaders—and so necessitated many "isolation" hospitals, "houses of separation", leprosy-houses, "leprosaria". Liberton ("Leper-town") near Edinburgh was a leper community. The oldest hospital still functioning (St. Bartholomew's, Rochester) started in 1078 as a leper hospital. Amongst the Religious Orders doing much hospital work were the Knights of St. John, Knights of Malta, and particularly the Order of St. Vincent de Paul. A "Hospital" was an incorporated almshouse just like a university college, save that its aims were eleemosynary and not educational. The hospital or "House of Pity" was usually in the Bishop's House, from which it spread to separate *ad hoc* buildings, the clinic and the infirmary. Poverty (always allied to sickness) was the only common denominator of the patients, and it may be said that all these places were more for care than cure. The first general hospital in England, St. Peter's, York, was instituted by King Athelstan and the Cathedral Canons. St. John's, York, was founded by Lanfranc, Archbishop of Canterbury, in 1084.

St. Bartholomew's, London, was founded in 1123 by Rahere, Henry I's jester(c). St. Thomas's began its great work in 1200. These, with all other similar monastic foundations, were abolished by Henry VIII in 1538, and being without funds their work practically ceased. The King could not very well allow this to happen; he was therefore compelled (with the City Corporation) to afford financial help. For this reason these two, with three others (Christ's, Bridewell and Bethlehem (St. Mary's or Bedlam)) became known as "Royal" Hospitals(d). They were "rate-aided". This connexion with the parochial system of the City of London may well be regarded as the embryo of public hospitals, and provides an interesting mediæval example of a service comprising several "hospitals" connected together and dealing with both bodily and mental ailments. Incidentally the provision of "State-aid" to charitable organizations is no new thing. King's College, Cambridge, and Eton College (founded by Henry VI) owed much to

(a) Ref. (2), 1-38.

(b) (2) 38, 43 and (4).

(c) (1) (16) 2.

(d) (2) 59-64 (16) 3.

hosts. It is quite possible that a disease which produces so little systemic reaction in human beings would not be detectable in the experimental animals used.

DISCUSSION

The form of myalgia described here is not Bornholm disease, which unfortunately has been given the all-inclusive name "epidemic myalgia". Bornholm disease seldom involves the trapezius muscle, and in addition differs by reason of its characteristic pleural pain, fever and leucocytosis (Sylvest, 1934). Recently two other epidemic diseases have been described under the titles which suggested a possible identity with our cases. These are: "Epidemic Myositis, with Neuritis, Erythema and Meningeal Symptoms" (Williams, 1941), and "Brachial Neuritis Occurring in Epidemic Form" (Wyburn-Mason, 1941). The clinical descriptions given show very little resemblance to the disease considered here. An epidemic disease which closely resembles the one under discussion is "Persistent Myalgia Following Sore Throat" (Houghton and Jones, 1942). They describe an outbreak of seven cases among a group of hospital nurses characterized by severe headache and mental depression in addition to myalgic areas in various parts of the body. Their cases differ from ours, however, by the presence of persistent fever, epistaxes and sub-ungual hæmorrhages.

The data obtained in the surveys show that acute myalgia of the neck and shoulders can occur in epidemics. Outbreaks of the disease are not easily recognized because of the general mildness of the symptoms, the slight interference with ordinary activities, and the absence of fatal cases. Furthermore, the dispersion of cases in an outbreak shows little sharp grouping. In the epidemics recorded here it is of interest that the medical officers in charge were unaware of the large number of cases in the communities under their care. It is probable that epidemics frequently occur, but are seldom recognized.

The widespread opinion that exposure to cold or draught tends to induce this form of myalgia cannot be readily dismissed. Assuming that the disease is caused by a specific infectious agent, such physical factors as cold or draught may well influence its development; analogies might be drawn with the common cold or herpes simplex.

The evidence for experimental transmission of myalgia is incomplete. The disease could not be produced in the experimental animals used. The apparent transmission to volunteers is subject to the criticism that these were members of a community in which the disease was currently prevalent and that the condition was not transmitted in other communities. The points in favour of experimental transmission are: First, the incidence of myalgia in the volunteers was far higher than in the remainder of the community. Statistically, the probability of this difference being due to chance is less than 1:10,000. Second, the incubation period in seven out of the ten volunteers was two to four days after inoculation. Third, the proportion of severe illnesses in the group was greater than among those who acquired the disease spontaneously. Fourth, reasonable explanations have been offered for the failure to transmit the disease in other communities. Final proof of this matter must await further evidence.

SUMMARY AND CONCLUSIONS

Epidemiological studies on acute myalgia of the neck and shoulder regions have been made in small groups of people in England during the winter of 1941-42. The evidence obtained indicates that the prevalence of this condition fluctuates in a manner similar to that of a communicable disease.

The clinical characteristics of this type of myalgia have been studied by an analysis of 125 cases which were detected during the epidemiological investigations. Certain clinical types have been defined. Most affected persons have a mild, self-limited illness of fairly uniform course, but occasionally the disease passes into a chronic form, extends to other parts of the body, and eventually produces the clinical syndrome of generalized fibrositis. These chronic cases would not ordinarily be identified as having originated from an epidemic of benign myalgia of the neck.

Experiments were performed in an effort to determine the transmissibility of the disease to human beings and to experimental animals. Transmission to human beings appeared to be accomplished through the agency of whole blood from acute cases, although the evidence is not conclusive because of the prevalence of the disease in the community at the same time. Attempts to establish the disease in other hosts were unsuccessful.

The evidence from these investigations supports the concept that acute myalgia of the neck and shoulders should be regarded as an infectious disease.

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the "general" hospitals which the L.C.C. inherited also in that year from the Metropolitan Boards of Guardians.

The earliest separate infirmaries were St. Pancras, St. George-in-the-East and Wandsworth. By 1930 all the London Boards had established "separate" Poor Law hospitals. In the early days, the relieving officer's order was an essential preliminary to the admission of a patient but the medical superintendent always had power to admit "emergency and urgent" cases, and it was by an elastic use of these words—brought about largely by the pressure of public opinion—that more and more patients were admitted direct by the medical superintendent. More and more acute cases were admitted, in addition to the "chronics". Operating theatres, X-ray departments and maternity units were provided at all, and special clinics, such as for radium therapy, at some—so much so that thousands of patients quite outside the scope of the destitute Poor Law class were admitted, paying for their treatment as they could afford.

Since the 1914-18 war there has been a very great increase in "hospitalization". Many thousands now turn to hospitals, as in-, or out-patients, who, thirty years ago, would never have dreamt of doing so. Small houses, lack of servants, greater confidence in hospitals and the increasing complexity of treatment—all have played their part in this.

Another cause of the increasing use made of the Poor Law hospitals was the inability of the voluntary hospitals to deal with the growing demands of the sick poor. In 1909 throughout the country the voluntary hospitals had provided in all some 25,000 beds. In the same year there were 100,000 sick beds in the Poor Law institutions and infirmaries actually occupied by patients^(k). The voluntary hospitals also have (especially those with medical schools) always adopted a "selective" method for admission to their wards; they are unwilling to accept patients who are likely to occupy beds for prolonged periods and the "interest" of the case is at times an important factor bearing on admission or rejection, whereas of course the municipal hospital must accept all comers who are destitute in that they need hospital treatment and cannot otherwise obtain it. Thus, when the L.C.C. took over the 29 general Poor Law hospitals in 1930, although they were not a unified service such as the M.A.B. "special" hospitals had become, and although they differed very much in their general standards and popularity, nevertheless they constituted a series of efficient institutions and gave the London County Council a good start in the evolution of a municipal general hospital service for the metropolis.

On the question of municipal hospitals generally we should refer to what was known as the "Bradford" experiment. Section 131 of the Public Health Act, 1875^(l) gave local authorities power to provide hospitals for the inhabitants of their districts (and to recover the cost of treatment from the patients)—but only one municipality, Bradford, used these powers, and established in 1920 a municipal general hospital, the only one prior to 1930 except the small hospital provided by the Borough of Barry. St. Luke's, Bradford, had 900 beds, a large consulting staff and was governed by the Corporation Health Committee. The powers of Section 131 were conferred on County Councils by the Local Government Act, 1929 (Sec. 14).

THE METROPOLITAN ASYLUMS BOARD^(m)

The main object of the Metropolitan Poor Act, 1867, was to achieve better classification throughout London of Poor Law cases. There were too many Boards of Guardians for each to make provision for "special" cases such as fever, smallpox, mental defective, boys for sea-training (the T.S. "Exmouth"); and a metropolitan central authority was the best solution. Thereby the cost of dealing with these "special" classes was equalized (through the Metropolitan Common Poor Fund, established by the Act) over the whole of London. The work of dealing with sick children was given to the M.A.B. in 1897 and the responsibility for the casual poor was transferred to the Board in 1911. In 1928 the management of the Metropolitan Common Poor Fund was entrusted to the Board.

Prior to 1867 there was no organized provision in London for the isolation of fever cases. There were two private hospitals for paying patients and the workhouse infirmaries provided isolation units for the poor. The Eastern, South-Western and North-Western hospitals were soon built by the M.A.B. The smallpox epidemic of 1870-71 and another in 1876-8, put great strain on the accommodation. A hospital ship "Dreadnought", lent by the Government, was moored off Greenwich and a camp of tents for convalescents was erected at Dartford. The Board could only admit to its hospitals "pauper" patients and the general public clamoured for the removal of this limitation. There was no compulsory notification of infectious diseases and consequently the control of epidemics was impossible. The Royal Commission of 1881, appointed to consider these difficulties, recommended that (a) Fever hospitals should be entirely disconnected from the Poor Law.

^(k) ⁽⁹⁾ 1V ⁽¹⁾ 1930, 44 ⁽¹⁸⁾.

^(l) ⁽¹⁶⁾ 29, ⁽¹⁷⁾.

^(m) ⁽²⁾ 92, ⁽¹²⁾ ⁽¹³⁾ ⁽¹⁴⁾ ⁽¹⁶⁾ 14.

"forced benevolences", an early form of taxation, as did Cardinal Wolsey's Ipswich Schools and Christ Church, Oxford.

The Tudor period, with the final legislation of Queen Elizabeth, saw the divergence of the voluntary from the "State" hospital, which, under the Poor Law, then began its separate career.

THE VOLUNTARY HOSPITALS(e)

The voluntary hospital movement made great progress in the first half of the nineteenth century, when the industrial revolution created great fortunes for the few and much suffering for the many. The springs of benevolence flowed freely to mitigate the hardships of the poor. The Westminster (1719), Guy's (1724), St. George's (1733), London (1740), the Middlesex (1745), and others had been founded earlier, but between 1800-1850 there were added Charing Cross, the Seamen's, Royal Free, University College, King's College, St. Mary's, the Royal Northern. Manchester saw the first fever hospital. The London Fever Hospital, now no longer used for the reception of fever patients, was for many years prior to 1939 the only voluntary fever hospital in this country. It was founded in 1802. The Society of Friends started an Asylum for Lunatics in York in 1788. Cottage hospitals (peculiar to Great Britain and the U.S.A.) were started by Albert Napper, F.R.C.S.(f). The first out-patient department or dispensary was opened in 1700. Convalescent hospitals are a recent but growing development(g), as are wards or special blocks for "paying"(h) or "private" patients (although of course financial stringency has resulted in all patients being asked to pay what they can. "Supported by voluntary contributions only" probably does not now apply to any hospital). The fever and mental hospitals were the first hospitals, built as such and not originating in sick wards of work-houses, to be provided entirely from public funds. The National Insurance Act, 1911, was the first great step towards the State provision of medical services on the grand scale. The rate- or tax-supported personal health services now include the School Medical Service, Maternity and Child Welfare Services, Tuberculosis and Venereal Diseases Schemes and the medical work of the Ministry of Pensions. As part of many of these services the voluntary hospitals work for, and are paid by, the local authority or the State. All the above are apart from the provision of hospital services *per se* by local authorities.

THE POOR LAW HOSPITALS—TO 1930(i)

The great Poor Law Statute of 1601 (Eliz. 43, Cap. 2) initiated that vast system of Public Assistance to the destitute known as "The Poor Law". Previously the "voluntary" subscriptions of the public had to be subjected to pressure by bishop and parson to secure their sufficiency and continuity. A poor rate was levied by the Overseers of the Poor. Children were set to work, the lame, impotent, old, blind and others "being poor and unable to work" were catered for. The home of the homeless or the poor-house was at first in cottages, then in the old-fashioned workhouse (still to be seen in country districts). Improvements were effected (e.g. combinations of the units, the ecclesiastical parishes), and, following a Royal Commission, the Poor Law Act of 1834 brought about great reforms. New and larger areas of administration—Unions—were created, Guardians succeeded the Overseers, classification of inmates was commenced and Government control and oversight in the form of the Poor Law Commissioners (which became the Poor Law Board in 1847, the Local Government Board in 1871 and the Ministry of Health in 1919) were established. The sick were taken from their homes (in 1868 two-thirds of the sick poor had domiciliary treatment only) to the workhouse sick wards, which developed after 1867 into the "separate" infirmary or Poor Law hospital. This was a substantial advance. In it appeared good medical and nursing staffs. London pioneered the separate infirmaries, followed by Manchester, Birmingham, Leeds, &c. Training schools for nurses were established; but the honour of being the first Poor Law institution to do this goes to the Liverpool (Brownlow Hill) Infirmary in 1865, followed by the London Marylebone Infirmary (now St. Charles' L.C.C. Hospital) in 1884. Classification of patients became essential. The voluntary hospitals have always been averse from receiving mental cases, "chronics" (those with advanced heart or lung disease, incurable lesions of the nervous system, inoperable cancers, the senile, the epileptic, &c.), the tuberculous and infectious. All these went, or were referred, to the Poor Law infirmaries. The Metropolitan Poor Act (the Gathorne Hardy Act) of 1867 is a great landmark in the hospital history of London. It founded the Metropolitan Asylums Board and with it there appeared the first organized hospital system in this country. Before describing that system as it had developed in 1930, it is necessary to consider

(e) (1) (2) 55. cf seq. (16) 3-7. (f) (2) 851. (g) (2) 34, 849 (16) 11, 36.
(1) (2) 78, (3) 64 (5) (6) (7) (8) (9) (11) (12) (13) (14) (16) 28.

(h) (16) 152.

Council of the County . . . and as from that day all then existing Poor Law authorities shall cease to exist.

The great advantage of this Act was that it removed the handicap to health authorities which arose through their having, in practice, no control over the rate-supported general hospitals as they were administered by separate local authorities. The provincial health authorities had provided maternity hospitals, sanatoria, fever and sometimes orthopaedic hospitals whilst the Guardians had often provided accommodation for the same classes of patients. At long last this Act of 1929 integrated the public hospital and public health services of the major local government areas.

"*Appropriation*" (1).—The County Council could have continued to administer under the Poor Law Acts the hospitals transferred to it, but it adopted the alternative and enlightened policy of administering them under the Public Health Acts. This necessitated changes in accountancy, meant that costs of treatment are recovered under the Public Health instead of the Poor Law Acts, and removed the hospitals from the detailed oversight of the Ministry of Health which is exercised over Poor Law establishments.

(u) There were transferred to the Council seventy-six general and special hospitals, containing 37,202 beds. With the 22 mental hospitals they make a grand total of 98 hospitals and 71,771 beds administered as one complete hospital system. The "special" units (v) are of considerable interest, including as they do *inter alia* the Plastic Surgery Unit under Sir Harold Gillies at St. Charles' Hospital, the Thyroid Unit at New End Hospital which was initiated by Sir Thomas Dunhill, and the Units at Lambeth Hospital (cancer of the uterus under Sir Comyns Berkeley and the cardio-vascular Unit under Viscount Dawson of Penn, and the late Lawrence O'Shaughnessy).

A few summarized statistics of the hospitals show 52,000 more admissions in 1937 than in 1931—an increase of 26% and a total of over a quarter of a million in-patients treated (apart from the mental hospitals).

It should be noted that throughout this paper, save in one or two instances where the war is specifically mentioned, we deal with the state of things as existed in August 1939, though the statistics given are in most cases for 1937—the latest year in respect of which complete figures are readily available.

The war wrought tremendous changes in the London hospital services which cannot be described here. This much can, however, be said—that London would have been inadequately prepared for the strain aerial warfare placed upon its hospital resources had it not been for the development of the L.C.C. hospital services which took place between 1930 and 1939. The pity is that the steady development which had been going on was stopped by the tragic events of 1939.

Head office organization.—It may be of interest to describe how a great hospital service is organized centrally. To assist the Medical Officer of Health there are, in addition to a Deputy, two Principal Medical Officers (one in charge of the hospitals and the other of the public health work generally and the School Medical Service) and other medical staff. The Department is classified into four Divisions (Hospitals, General Public Health, Staff and Finance) plus the Ambulance Service—and the Mental Health Service. There is a Chief Clerk, an administrative officer in charge of the Mental Health Service, four Principal Clerks (one in charge of each Division) and a large clerical staff. There are also at County Hall a Chief Dental Surgeon, Ophthalmologist, Aurist and other specialist medical and surgical staff, a Chief Chemist, Inspectorate (male and female) and—in charge of the vast nursing staff—a Matron-in-Chief.

It is a comfort and a help to feel that these many thousands of people—of medical and other professions, lay staff, nurses, engineers, domestics, clerks, porters and others—all work so well together as one team. The objective of sound administration is to guide without too much direction, to foster local initiative, to collect good ideas and pass them on, to leave experienced doctors to get on with their doctoring and not to interfere between any doctor and his patient.

Departmental committees.—When the Council knew of the vast services coming to it in 1930, it realized that to effect smooth transference of authority changes of procedure should not be too hastily made. Much preliminary work had been done and many conferences held, including meetings of medical superintendents, matrons and stewards, and it had been understood that any difficulty which might arise should be dealt with in exactly the same way as under the previous régime. A series of departmental committees (a sort of internal Royal Commission) was set up to deal with such matters as Hospital Standards, Maternity, L.C.C. Pharmacopœia, Pathology, Forms and Records (this committee instituted the interesting system of statistical work by mechanical tabulation with "Powers"-cards), Ambulances, Supplies, Staff, Economy (w). This valuable depart-

(1) (9).

(u) (9).

(r) (9) espec. IV (1) for 1930.

(w) (9) IV (1), 1930, 2 et seq.

(b) Fevers should be notified to district medical officers of health. (c) Hospitals in London should be for "fever"; smallpox patients should go to Dartford. (d) Country convalescent fever hospitals should be established.

(a) was achieved by the Diseases Prevention (London) Act, 1883 (Poor Law Acts, 1889, and Public Health (London) Act, 1891). (b) was implemented by the Infectious Diseases Notification Act, 1889. (c) The hospital ships "Atlas" and "Endymion" and "Castalia" (superseding the "Dreadnought") were moored off Dartford. Permanent hospitals were built (Joyce Green, Orchard, Long Reach) and the ships given up in 1904. Patients were conveyed, owing to the time taken by horse ambulance, from wharves in London by river ambulance steamers; now all smallpox cases go to Dartford by motor ambulance. (d) The Board built two large convalescent fever hospitals in the country.

Like the general hospitals, the infectious hospitals soon became popular. The proportion of admissions (scarlet and enteric fever and diphtheria) to notifications in 1890 was 33·6%, and in 1929, 93·5% (in the case of all notifiable diseases admitted)(n). The mortality rates, on the contrary, fell. In 1890 they were in the case of diphtheria 30%, in 1929, 3%; in scarlet fever 6% and 0·6%, respectively. The Board's ambulance service, started in 1879, grew with its use and developed into a most efficient system with six large ambulance stations and over 150 motor vehicles(o). Measles and whooping-cough were admitted. Hospitals or units were provided for V.D., ophthalmia neonatorum, zymotic enteritis, encephalitis lethargica, juvenile rheumatism, mastoiditis, cancer of the uterus, &c. Classes for the instruction of medical students in fevers and smallpox were established in 1889. The immunity of the smallpox staff from the disease was remarkable. From 1884-1901, 17,900 smallpox cases were treated by 2,198 staff, not one of whom, where there had been successful revaccination, contracted the disease; later records show similar results. The Board made its own diphtheria antitoxin at the Belmont laboratories and stables, where there was a bacteriologist responsible also for the general bacteriological work of all the hospitals. Two group laboratories were provided, also hospital laboratories in each hospital; and a general director of research and pathological services appointed.

The M.A.B. also established asylums for imbeciles and the feeble-minded(p). Darenth, for improvable, was a most successful institution, a large amount of useful work being done there in the schools and workshops. From the latter goods to the value of over £70,000 per annum were turned out. An interesting experiment was conducted—most successfully—at Tooting Bec Asylum, where aged senile cases were, and are, received without any "certificate" under the Lunacy Acts (to avoid stigma on the patient or relatives). Its name was altered (anticipating later general titular amendment) to Tooting Bec Hospital.

The Board built new or adapted existing buildings as hospitals for (a) children suffering from ophthalmia, ringworm and other skin diseases; (b) epileptics; (c) the convalescent (seaside hospitals). Their Queen Mary's Hospital at Carshalton is the largest children's hospital in this country and probably in the world (1,284 beds). The history of the treatment of Poor Law children suffering from trachoma and of the investigations which led to the building of High Wood and White Oak Hospitals are of great interest and are dealt with fully in an article in the L.C.C. Annual Report, 1935(q).

Another important branch of the Board's work was the provision of hospitals and sanatoria for the tuberculous, initiated to give the "sanatorium benefit" created by the National Insurance Act, 1911, and continued under another guise by the Act of 1920(r). Almost from the inception of the scheme insured and non-insured were admitted alike. Some of these institutions were specially built and others adapted.

In 1930 the L.C.C. found it much easier to "take over" the M.A.B. special hospitals, which the Board had welded into a homogeneous, efficient, unified service, than it was to take over the general hospitals, which came to it from 25 different Boards of Guardians, each with its own system.

THE LOCAL GOVERNMENT ACT, 1929—THE LONDON COUNTY COUNCIL(s)

The Local Government Act, 1929, dealt with the transfer of functions, powers and duties from the Poor Law authorities to the County and County Borough Councils, the major local authorities, on whom the Minister of Health in a recent Parliamentary statement said he intended to place additional responsibilities in connexion with hospital work. The most important section of the Act of 1929 was the first, which says simply that on April 1, 1930, the functions of each Poor Law authority shall be transferred to the

(n) (9) (12) (13) 27. (o) (9) (12) (13) 77. (p) (12) (13) 41. (q) (15).
(r) (12) (13) 41. (s) (6) (9) (10) (12) (13) (16) 58.

Names of hospitals.—The Council has rechristened many of the general hospitals, giving them the names of Saints with local associations (e.g. Marylebone Infirmary became St. Charles' Hospital) or geographical names more appropriate to their position (e.g. Southwark Infirmary became Dulwich Hospital(c)).

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In 1932-3 a "Joint Survey of the London Voluntary and Municipal Hospitals" was prepared(e) giving detailed statistical information, with maps, of the use made of the London hospitals. The interesting fact emerged that whilst the municipal hospitals are almost entirely occupied by inhabitants of the administrative County of London (only comparatively few "out-county" cases come in under special arrangements), the London voluntary hospitals in 1931 admitted 40% from outside the county boundary.

Building works at the hospitals(f). *Planning and development, &c.*—It will be realized that—in addition to work dealing with equipment, staff, classification and co-ordination—the very much thought, time and money have had to be spent on the actual buildings of the hospitals, new structures, repairs, reconditioning, &c. Not only have urgent works been carried out as required but a plan was formulated spreading further necessary works over a period of years. The war has put an end to the carrying out of this plan. It is hoped that it will be implemented when peace returns.

There are three reasons why the volume and variety of works completed and contemplated were so great: (i) The backward condition of many of the hospitals in whole or in part in 1930. Their shortcomings, in one hospital or another, extended to every component part of a hospital and included deficiencies of all kinds, overcrowded wards, out-of-date or absent ward washhand basins, dreadful sluice rooms, poor ward kitchens, inadequate hot water supply, heating, lighting or telephone systems and lifts, insufficient medical science which has coincided with a like advance in the engineering, architectural and other sciences. There has, therefore, been need for a progressively greater and more complicated provision of medical equipment with its necessary accommodation and the demand for a better type of hospital building. (iii) Natural decay and the normal effects of wear and tear, particularly as long notice of their demise was given to the former authorities and some, naturally, did not proceed with developments which they otherwise might have done. The number and extent of the buildings are enormous and the repair and replacement required in any one year considerable. The main factor determining the need for expenditure was age, some parts of the buildings being more than a century old. It must, however, be pointed out that some of the hospitals transferred to the Council were in excellent condition.

Voluntary workers.—Apart from the voluntary services of members of the Council and of the hospital committees, much help is received in our hospitals from voluntary workers of all kinds. They include boy scouts, girl guides and, since the war, members of such bodies as the Women's Voluntary Services, the Order of St. John and the British Red Cross Society, which has always managed the hospital libraries.

Rheumatism.—The Council has a scheme for the treatment of juvenile rheumatism for which there are special units at Carshalton, Brentwood, Norwood, Sutton and Dartford. At St. Stephen's Hospital there is a special unit with beds for adult patients. It works in close association with the British Red Cross Clinic, Peto Place, and the St. John Clinic, Ranelagh Road. Special treatment which the Council cannot provide in its own institutions is given where necessary. For example, patients requiring spa treatment are sent to the Royal Mineral Water Hospital, Bath, and St. John's Brine Bath Hospital, Droitwich.

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(c) (9) *passim*.

(d) (9) II, 1930, 14, and IV (1).

(e) (10).

(f) (9) IV (1), (16) 334

mental committee system, which is only possible in a large service, still continues.

Admissions.—Admissions to the general hospitals are now mostly on the sole authority of the medical superintendents with or without a medical certificate, or on an order by the Medical Officer of Health. The admissions by relieving officers' orders (the old Poor Law method) decreased from 54,500 in 1932, to 7,000 in 1937, whereas patients admitted by the medical superintendents increased from 72,000 to 155,000(x). This sheds a great light on the rapidly changing nature of our hospitals—from Poor Law infirmaries to general municipal hospitals. Another point for notice is the doubling (20,000 as compared with 10,000) in the number of births—showing how more and more mothers of London are using our maternity wards. In 1938 the births were 21,147. In 1937 35% and in 1938 37% of all births to mothers resident in the metropolis took place in the Council's general hospitals. The fever hospitals admit all types of fevers; the general hospitals admit everything—in fact, the L.C.C. hospital service may claim that it presents “the ever open door”.

Classification of patients is of primary importance and a large hospital service gives ample opportunity for this. Not only are the hospitals themselves classified into—acute, chronic, convalescent, fever, tuberculosis, &c.—but there are many special units, e.g. in addition to those previously mentioned, for deep X-ray and radium therapy, fractures, rheumatism, diabetes, gastric cases, ophthalmic cases, chest surgery, &c.

The *chronic sick*, including the bronchitic, the cardiac, the patients with inoperable cancer, &c. and the enfeebled aged, are patients practically wholly confined to bed, requiring daily medical or nursing attention. Although 3,500 of our chronics have been evacuated we still have about the same number under our care—3,500 was the pre-war figure. From the point of view of training both doctors and nurses there is much to be said for a “chronic” department in an acute hospital, but unfortunately such patients are not regarded as of great interest. The Council has a few hospitals occupied solely by the chronic sick. Perhaps the compromise of a large “acute” hospital with one or more “chronic” blocks or sections is the best solution—and indeed that is what we do in many instances.

The care of the chronic sick(y) constitutes a serious problem for the municipalities—and one which the voluntary hospitals (except for one or two places like the Putney Home for Incurables and the Birmingham Jaffray Hospital) have never tackled.

The *infirm* are persons normally able to get up and use day rooms but who require help in dressing, &c. These are dealt with in the Social Welfare Committee's “institutions”.

Healthy orphaned and derelict children under 3.—These fall to us to be dealt with and are catered for in “nurseries”. At 3 they go to the Education Department's Residential Schools. (They are practically all now evacuated to the country.)

Mental.—There are “observation units” at four hospitals—one, St. Pancras, recently opened, with the latest improvements. These observation units are peculiar to municipal hospitals and are part of the machinery of the Lunacy Acts. We have also three psychiatric out-patient clinics, run by specialists from the mental hospitals.

Education of children in hospital(z).—The late M.A.B. always recognized the importance of the education of children in its hospitals. They were “long-stay” hospitals and the fact that a child is physically or mentally handicapped makes its education all the more important. There were commodious, specially built school buildings at some of the children's hospitals just like an ordinary elementary school. If the children could not get to them, the teachers went to the wards. Now the education department of the Council provides the teachers and the equipment, and a first-rate education adapted to the needs of the children is given. Scholarships are awarded to suitable pupils. The Board of Education has since “certified” these schools under Part V of the Education Act, 1921. and they qualified, in consequence, for educational grants.

Rehabilitation of adults is on a par with the education of children—both are intended to enable the patients to become self-supporting citizens. Unfortunately rehabilitation has not yet received the attention it deserves.

Medical research(a).—The L.C.C. hospitals, with their 72,000 beds, offer a vast clinical field for medical research and every advantage is taken of these unique opportunities. There are two Clinical Research Committees from our own staff and of course there is a great deal of individual research work done as well(b). Facilities are given for clinical trials of new therapeutic substances and for access to types of rare cases to those writing papers. The Rockefeller Foundation has given generous help to the Central Mental Pathological Laboratory of the Maudsley Hospital.

(x) (9) *passim*.(y) (2) 303, 306, (9) *passim* (11).

(z) (12) (13).

(a) (9) (12) (16) 66.

(b) (9), see Series IV (111).

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(e) (9) *passim*.

(d) (9) II, 1930, 14, and IV (1).

(e) (10).

(f) (9) IV (1), (15) 334

Patients with symptoms of neurasthenia or other mental disorders are transferred to the Lady Chichester Hospital, Hove, or to one of the many homes of the Mental After-Care Association.

Amenities for patients.—The Council provides in its hospitals: Chapels or devotional rooms (well equipped and decorated) and chaplains, ministers and religious instructors of all denominations—some of the chapels are very beautiful buildings, particularly that at Queen Mary's, Carshalton—libraries, newspapers and magazines, games, tobacco, sweets and toys (for children), flowers, materials for therapeutic occupational and recreational work, handicraft workshops and canteens. Clothing is also provided where necessary. "Samaritan" funds for financial help to patients, usually on discharge, exist at all hospitals. Their sources are donations and legacies and they are administered by the almoners. Gifts, sometimes substantial, are constantly being received from patients and their friends. Although our hospitals are rate-supported—and, therefore, need no extraneous assistance—these expressions of gratitude are welcome. Many are ear-marked for special purposes, e.g. "for the nurses" and these go into nurses' recreation funds.

Dietary.—The Medical Officer of Health has prescribed dietary scales for the various classes of patient. The doctor in charge can, of course, always order "extras" and special diets. The bringing in of food by patients' friends is discouraged. We employ trained and qualified dietitians and food supervisors and every care is taken to see that the meals are served hot.

Subsidiary services and activities(g).—The following are amongst the supplementary services provided:

Dental: Every hospital, including the mental hospitals, has a dental surgeon who attends in accordance with requirements. There are eight whole-time and some twenty-eight part-time. As an experiment to determine whether the service should be extended, whole-time dentists have been appointed to each of two hospitals. They are required to examine the teeth of every patient and provide treatment for all whose illness has been caused or aggravated by dental trouble. Ante-natal patients receive special attention. Dentures are made at our central dental laboratory. In 1937, 13,152 in-patients and 10,427 out-patients received dental treatment. This involved the giving of 10,000 anaesthetics, and the provision of 1,500 dentures.

Maternity: Great advances have been made—300 beds added (making 1,000)—ante- and post-natal clinics developed. In 1937, 21,150 women were seen at 23 ante-natal clinics (132,270 attendances). There is full co-operation with the Borough Councils' Maternity and Child Welfare work. Necessitous expectant mothers receive extra nourishment. The Council also provides a Domiciliary Midwifery Service, employing directly 75 midwives, and under agreements with outside hospitals and nursing associations, 50 full-time and 51 part-time midwives. In 1940, 14,138 domiciliary confinements were attended under these arrangements, but in 1941, owing to "blitzes" this number fell to 6,531. We maintain in the hospitals an emergency obstetric service, "flying squads"—doctors and nurses—who can be rushed to any difficult or urgent maternity case at any time.

Surgery: Existing operating theatres have been remodelled and new ones of the latest design built. The number of consulting surgeons engaged has been considerably increased and the theatre nursing staffs added to and improved. In the general acute hospitals (only) the number of major surgical operations under general anaesthetics was in 1931, 31,063, in 1937, 47,955. The Council's hospitals are being used increasingly for emergency surgery.

Tuberculosis: All patients are dealt with under the Tuberculosis Scheme, which secures continuity of service and treatment through its very great resources (dispensary, hospital, sanatoria). In the general hospitals there are some 1,000 beds used for non-ambulant patients unsuitable for sanatoria, and for emergency and observation cases. 2,186 beds in L.C.C. tuberculosis hospitals and sanatoria and 1,345 in voluntary institutions are used.

Venereal disease: The Council acts as Manager of the London and Home Counties Scheme. It maintains the Whitechapel Clinic, the largest in London, and the Endell Street Clinic; and subsidizes 16 clinics at voluntary hospitals. Children are sent to the Waddon Country Home. There is a special hospital for maternity cases infected with venereal disease and a special unit for children so infected. There are wards set aside for the treatment of V.D. patients in certain general hospitals. Everything is done with the co-operation of religious and social workers, for the assistance, rehabilitation and after-care of female patients. Trained instructresses attend to teach them handicrafts, needlework, &c.

X-ray and physiotherapy: These are needed more and more in a modern hospital and our hospitals have not fallen behind. All have X-ray and massage departments with

trained radiographic and massage staff. There were over a million attendances in these departments in 1937—more than twice the number in 1931. Deep X-ray and radium treatment of cancer is provided at Lambeth and Hammersmith hospitals.

Other items which may be of interest are:

Adoption of children: The work connected with the adoption in suitable cases of orphaned, &c., children by approved applicants under the Adoption of Children Act, 1926, is done in the Public Health Department.

Lady almoners: A large staff now employed on social and charitable work and the collection of maintenance charges, &c.

L.C.C. Ambulance Service: 20 stations, 400 staff, 200 ambulances (pre-war: vastly expanded now).

Charges to patients(h): Patients pay what they can, but fever, smallpox, T.B., V.D. and E.M.S. patients are treated free.

Consultants and specialists: The consultants' panel—pre-war—contained 250 names—the appropriate consultant being available at any time at any hospital.

District medical and nursing services: The council's continuation of the old "parish doctor" system—work now associated more or less closely with the general hospitals. A large subsidy is paid for district nursing.

Farms and gardens: Most hospitals, especially those outside the central area of London, have gardens—some very beautiful (Carshalton, Heatherwood, Dartford). At many, especially the mental hospitals, there are farms under bailiffs and trained staff. Farm work is a therapeutic measure at some. In view of the national effort in food production, a central Farms Committee has recently been set up. The total area of farm land is now 6,000 acres. There are 1,530 cows, 1,630 sheep and 3,600 pigs.

Hospital finance(i): A complicated subject, but we must say that *costings* are an essential part of the work of an efficient hospital system. Costings often show that apparently the same processes or procedures cost twice as much at one hospital as at another. Extravagance or economy (without loss of efficiency) are often habits of mind and the latter can replace the former once the facts are known. Running hospitals is "big business" and business methods must, to the extent appropriate, be applied.

Laundrywork is done in the hospital laundries with a few exceptions, where it is done by contract. Some 70,000,000 articles are washed yearly. An expert on laundrywork has been appointed.

Medical (under- and post-graduate) and nursing education: Facilities for London medical students in general and obstetric work, fevers and mental cases—various post-graduate facilities—the British Post-Graduate Medical School at Hammersmith. L.C.C. hospitals are training schools for all groups of nurses—general, children's, fever, T.B., mental, and male.

Out-patients: Departments at all general hospitals. In 1937 nearly one million attendances.

Pathology: A complete service—group and hospital laboratories and a serum institute.

Staff: General and special hospitals: total 21,000 including medical 760, nurses 11,000, clerical 500, domestic 9,000. Mental health services total staff is 12,000.

Supplies: Annual cost of supplies £4,000,000—Food, £1,500,000—Medical and surgical supplies, £300,000—all standardized.

A FEW COMPARISONS BETWEEN VOLUNTARY AND MUNICIPAL HOSPITALS

(k) An essential difference is that a Voluntary Hospital is an individual unit. It prides itself on its individuality and freedom from control. A hospital service, particularly a large one with a statutory duty to provide for the inhabitants of its district, fits individual hospitals into a co-ordinated scheme. A service has to provide the best treatment for everyone who applies. It cannot have any gaps. It cannot say "We have no bed for you". Moreover, it can specialize and thereby provide the highest skill and the best equipment for unusual and particularly complicated or expensive forms of treatment. In a service there is "free trade" in information—the good ideas of the individuals

(a) (6) 42, (9) IV (II), (16) 103.

(i) (2), (9) Series IV (II) and (16).

(k) (2) 85, (16) 27, 63.

forming the team are passed to all the other members. There are frequent conferences and discussions at which ideas are pooled. Experts in specialized forms of treatment and in the specification (often involving long research into the best design) and the purchase of supplies, in hospital engineering and architecture, can be provided for a service, on a scale which could not be justified for individual hospitals. The ideal is to retain individuality and freedom to try experiments and improvements in the separate institutions of a service but to give them also the benefits of team work and combined experience.

The King Edward Fund, the British Hospitals Association and the Nuffield Trust form links between individual voluntary hospitals, but they cannot weld the individual hospitals into a service.

The systems of administration are not so different as is sometimes thought. In both voluntary and municipal hospitals the management is in the hands of public-spirited people who are giving their services to the community free of charge. But there is this difference, the managers of municipal hospitals are responsible to the ratepayers who elect the members of a Council and the members can be turned out at the next election if the public are dissatisfied with the services rendered. As a consequence complaints are regarded very seriously. Voluntary Hospital Committees have to run on such gauntlet. Vacancies on the Committees are filled by co-option. They are responsible to the subscribers and to their consciences, but the patients for whom the services are provided have no votes and cannot elect a new Committee.

There are differences in the methods of staffing municipal and voluntary hospitals which need not be described in detail. Both systems have their advocates but there are indications that they will tend to approximate more closely to each other as time goes on.

Sir Henry Burdett in "Hospitals and Asylums of the World" (1893) pointed out the differences, at that time, between voluntary hospitals and Poor Law infirmaries (municipal hospitals); to-day these "differences" have been modified or removed entirely(1). The majority of patients in the infirmaries are no longer "chronics" as they were in Burdett's time. The L.C.C. hospitals now do an enormous amount of acute work—and their equipment is as good, if not in many instances, better than in any voluntary hospital. The infirmaries had no out-patient departments. All L.C.C. general hospitals have such departments—many doing very heavy work. The infirmaries could not get rid of an unwanted patient. This is still true if the patient is medically destitute. Burdett also mentions there are "no Medical Schools", which is, again, still true, though the Medical Schools are relying increasingly on the Council hospitals for clinical material, especially in obstetrics. He says "Classification is bad". It is now good. "Guardians are loth to spend money." This is a fair accusation but cannot hold in the case of the L.C.C. "The County Councils should 'take over'." This was done in 1930. "The infirmaries have no honorary medical and surgical staff"—but the Council has a very complete staff of paid consultants. It has often been pointed out that whilst voluntary hospitals have to solicit the public for funds, municipal hospitals are relieved of anxiety by their power to levy a rate(m).

SOME OF THE IMPROVEMENTS EFFECTED BY THE LONDON COUNTY COUNCIL(n)

The Poor Law infirmaries of fifty years ago provided food and shelter for their patients; but their skilled staff and equipment were so meagre that they could only provide in very small measure the services of medicine and surgery which were available at that time. The medical staff of even the largest institutions consisted only of a medical superintendent and—in some cases—a deputy. Paid nurses were hardly employed at all and only in very small numbers. The training of probationers had been initiated in a few, but much of the nursing was still done by pauper inmates. There was but scanty provision of medical and surgical equipment, none for any special form of treatment, and no operating theatres. There had been great developments before 1930 but from the outset the L.C.C. has pursued a policy of progressive improvement in every direction and in every hospital coming under its charge. Its aim was to raise them all to the highest modern standard, and although this was a formidable task and all its plans have not yet been completed, a great deal has been achieved, and the result is seen in the hospitals as they exist to-day. The depressing conditions of the past have been replaced by cheerfulness and comfort. Everywhere the ward walls

(l) (2) 85.

(m) (2) 115, (16) 315.

(n) (8) (9) *passim*.

are now decorated in attractive colours; central heating and modern lighting have been provided, and many open-air balconies have been erected. Under other headings we have referred to the great increase in the quantity and quality of the work carried out in the Council's hospitals, and reference to the statistical tables contained in the annual reports (those dealing, *inter alia*, with operations, out-patients, physiotherapy, massage, maternity, X-ray work, convalescence, dental work, number of staff, and so on) shows how the work carried out has, since the Council took it over, multiplied in some cases fourfold.

The Council has issued since 1930 a series of annual reports dealing with its hospitals, which to anyone interested in the subject, are mines of information.

Views of the Ministry of Health.—In its Annual Report, 1934-35(o), the Ministry says: "The effects of the Local Government Act on organization have nowhere been more strikingly illustrated or on such a great scale as in London. To achieve the great advances made, unified control from the beginning and a single head (the County Medical Officer of Health) were essential." Sir Kingsley Wood, the Minister, added: "London (whose area, population and resources exceed those of not a few sovereign states) provides an example and an encouragement as to what may be achieved in the development of public, medical, institutional and welfare services."

The L.C.C. Mental Hospitals.—These fall into two classes (i) those (originally "lunatic asylums") belonging to the Council on March 31, 1930, and (ii) those mostly for imbeciles and feeble-minded transferred from the M.A.B. on April 1, 1930. (This transfer permitted similar classification and unification as in the case of the other hospitals.) There are 22 mental hospitals (temporarily 24) with over 34,000 beds. Until County Councils were formed (under the Local Government Act, 1888) the provision and administration of county lunatic asylums was the duty of the Justices. Hanwell (now St. Bernard's Hospital) was the first, in 1831. It was here that "The Retreat (York)" humane methods superseded the "restraint" (handcuffs, muffs, leg-irons, &c.) formerly in vogue. Colney Hatch (Friern Hospital) followed in 1851, and then the others. The Mental Deficiency Act, 1913, separated and specialized the care of the mental defective and there are four hospitals exclusively for this class: Manor, Farmfield, Brunswick House and Southside.

Maudsley Hospital (largely the gift of Dr. Henry Maudsley, a distinguished alienist) is for patients with a good chance of recovery and is an important research centre in psychiatry. It received "voluntary" and "temporary" patients fifteen years before the Mental Treatment Act of 1930. It has two University of London Chairs—in clinical psychiatry and mental pathology.

St. Ebba's: A "villa" hospital for recoverable patients.

Horton: Here is concentrated the treatment of general paralysis, &c., by malaria therapy.

The mental hospitals have surrendered (by overcrowding mental patients) 7,800 beds to the Emergency Medical Service for air-raid casualties, &c.

The Council has recently recognized the importance of treatment as distinct from detention in mental hospitals by transferring their management to the Public Health Department.

THE WAR

As a result of war-time restrictions building developments have been suspended. Unprecedented demands on our beds have been made and met. Staff, both at the County Hall and in the hospitals themselves, have left for, or been called to, H.M. Forces. Key staff have been seconded to Government Departments and expansion and progress in practically every direction have been either stopped or considerably curtailed.

Air-raid damage to hospitals.—From the commencement of the air raids on London in September 1940, to their cessation in May 1941, air-raid "incidents" occurred at our hospitals. These hospitals included all those within the administrative County of London and the damage ranged from that of a slight nature (windows and roofs damaged by blast) to total destruction of large parts of hospitals which necessitated their complete closing while repairs to vital equipment such as operating theatres, kitchens, laundries and boiler houses were carried out. Repairs were put in hand immediately wherever practicable and by March 1942 much accommodation had been brought back into use. Compared with the extent of the damage and the number of incidents the casualties among patients and staff were fortunately small. We would pay a sincere tribute to the bravery and devotion to duty displayed by our hospital staffs during these "blitzes".

Doctors and nurses and all grades "carried on" in a magnificent manner. Many have been decorated for their gallantry, but many more performed deeds of quiet and unnoticed heroism, and a sense of vital duty well and nobly done without ostentation is their only reward.

The War Emergency Hospital Scheme of the Ministry of Health is a hospital service in which the L.C.C. has played its part—the details of which cannot be considered here.

We have endeavoured to make this paper factual only and have repressed any inclination to express opinions. If any of our statements can be regarded as opinions they are our own and not (necessarily) those of the County Council. It is tempting to forecast the future but time will not permit(p). Suffice it to say that the L.C.C. Service shows what an organized hospital service under public control can do.

It has been stated that the four main functions of a hospital are: (1) To cure the sick; (2) medical and nursing education; (3) investigation and research; and (4) disease prevention. We are trying to do all we can in all four directions.

We would like here to pay a warm tribute to the work of Sir Frederick Menzies whose vision and wise leadership made possible the progress which we have endeavoured to outline.

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JOINT DISCUSSION No. 5

Sections of Ophthalmology and Neurology

Chairman—A. J. BALLANTYNE, M.D.

(President of the Section of Ophthalmology)

[February 13, 1942]

DISCUSSION ON WAR INJURIES IN RELATION TO OPHTHALMOLOGY AND NEURO-SURGERY

Mr. Frederick Ridley: In 1915 the Ophthalmological Society of the United Kingdom held a discussion on war injuries. Hysterical blindness and blepharospasm, functional night-blindness, the fact that hypermetropia often becomes wholly manifest under the stress of active service, and papilloedema, were discussed and the observations are of importance to-day. Lister drew attention to the fact that increasing papilloedema is of more importance than the observation of papilloedema *per se*. Even now this is not sufficiently recognized and our current clinical notes do not, as a rule, indicate clearly the degree of papilloedema. It would be helpful to the neurologist and to the ophthalmologist if every case were described as "doubtful", "definite", or "gross". Too often, under war conditions, the full notes of disc appearances and measurements of swelling do not travel with the patient but, if the appropriate adjective were always coupled with the diagnosis, change in degree of papilloedema, which is all important, would be apparent.

The following groups of cases have been selected as being either unusual in civilian practice or as having proved of common interest to my neurological colleagues and to myself. All were observed in the course of work under the Emergency Medical Service.

GROUP 1

Diplopia due to displacement of the eye.—Displacement of the eye as a result of fractures of the orbit associated with great violence is not uncommon, especially when the fracture involves the middle third of the face. The eye is often protruded for several days owing to orbital hæmorrhage and œdema. Later the eye is displaced, usually downwards, and enophthalmos may be observed. The patient complains of diplopia, this may be confusing unless the displacement of the eye is recognized. It rarely simulates a typical oculomotor nerve palsy. In many cases there is only a small field of single vision. Treatment consists of orthoptic training assisted in many cases by operation.

Diplopia due to local injury to muscles.—Local injury, especially that due to lateral blows on the head, often gives rise to direct bruising and paresis of the external rectus muscle, with diplopia. Later on limitation of movement of such an injured muscle may be permanent. In either case the condition may be confused with a 6th nerve paresis.

GROUP 2

Direct injury to the eye.—*Comotio retinæ* is often seen and may complicate the diagnosis if papilloedema is suspected. Traumatic mydriasis has been observed many times. It may be confusing in a patient admitted unconscious following a severe head injury. There may be local injury, the pupil is not fully dilated and its size does not alter. Traumatic mydriasis may result in a permanently non-reacting semi-dilated pupil, which may complicate subsequent neurological diagnosis.

Indirect retinal changes.—Two types of traumatic retinal angiopathy are described. Purtscher (1910) described multiple glistening white areas, $\frac{1}{2}$ to $\frac{1}{4}$ disc diam. scattered over the posterior pole of the eye and situated in the inner layers of the retina, following

compression injuries of the head or thorax. Small preretinal hæmorrhages and a mild œdema of the disc may also occur. Recovery without residual scarring is to be expected within one week. Wagenmann described a second group in which small hæmorrhages in the inner retinal layers about the posterior pole occur without the patches of œdema. This is the characteristic appearance in retinal hæmorrhage of the newborn. Two cases of this type were seen. One was a young man who was involved in a bomb explosion. No history was available but the retinal appearance led to the discovery of a deep bruise about four inches long above and parallel to the clavicle on the affected side. It is possible that he had lain with his neck over a bar of some kind or had fallen across such an obstruction. The case is of interest as an unusual type of retinal hæmorrhage and is perhaps unique in being due to unilateral obstruction of the great veins at the base of the neck. A second similar case was seen in a patient who received a gunshot wound in the occipital region and developed extensive hæmorrhages involving the inner retinal layers in both eyes. These hæmorrhages cleared up within seven days. This case may belong to a third group or may be classed with retinal angiopathies due to compression of the head, in which the pathology of the retinal hæmorrhages is obscure.

GROUP 3

Direct injuries to the optic nerve.—One case in this group, a penetrating wound of the left orbit dividing the optic nerve, was of interest in that optic atrophy and narrowing of the retinal arteries was seen only twelve days after the injury. This is the earliest onset of optic atrophy I have seen; one other showed atrophy at seventeen days. The majority have been observed at the recognized interval of three weeks after injury.

Indirect injuries to the optic nerve.—This note is an abstract of a series of such cases reported by Dr. J. W. Aldren Turner.

Twelve cases of this type were observed in a series of 450 head injuries admitted to an E.M.S. Centre. In 11 cases the injury was frontal, often somewhat lateral, and in one parietal. In two patients the injury was a minor one: in four there was loss of P.L. the others ranging from $\frac{6}{9}$ to hand movements. Field loss was variable, always involving constriction of the peripheral field, three showed central defects with steep edges, three showed large sector defects, two involving the fixation point. Recovery may be seen up to four months but is rare subsequently. Progressive deterioration for several months is often recorded. Pallor of the disc within seventeen days was observed but three weeks is the rule. The pupillary response is sluggish but the normal consensual response distinguishes it from the Hutchinsonian pupil. The pupil is not dilated save in complicated cases. Complications included three cases of cerebrospinal rhinorrhœa, three of anosmia and one 3rd and one 6th nerve palsy.

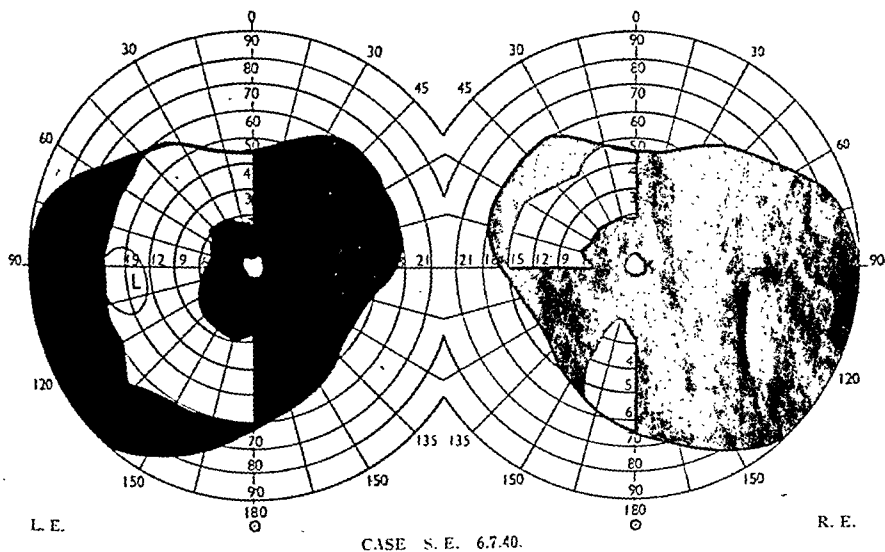
In all twelve cases stereoscopic X-rays showed no fracture involving the optic foramen or canal. The lateral character of many of these injuries and the fact that several were of minor severity does not support the theory of antero-posterior deformation of the skull as the cause of these injuries. Damage to the optic nerve is probably due to a vascular lesion in the sheath or in the nerve substance as has been suggested in lesions of the chiasm. Mr. Ridley has suggested that the blow is transmitted by the skull directly to the optic nerve which is firmly fixed in its canal, and which is thus displaced sharply backwards while the loosely supported globe is "left behind". As the eyes will often be looking towards the direction of the blow the optic nerve will be relatively on the stretch at the moment of impact and the optic nerve at the foramen will thus be sharply snatched and the vessels in the sheath or nerve substance may be torn. Occlusion of the central artery of the retina occurs in similar injuries and may be due to the same mechanism.

GROUP 4

Visual field changes in penetrating wounds of the vault.—This group has provided many cases of interest but few that might add to our knowledge. Aphasia, alexia and hemiplegia have been observed in association with homonymous hemianopia but the order in which recovery has taken place and the site of injury have rarely presented a clear-cut clinical picture.

One case (S. E.) who suffered a gunshot wound, the fragment entering in the left occipital region and lodging in the right posterior parieto-temporal region, is of particular interest. The fragment passed through the left visual cortex just in front of the pole, the macula and about five degrees around it being spared, and passed upwards and forwards with low velocity, passing through the right optic radiation. The right field defect is a typical hemianopia with sparing of the macula (fig. p. 47). The left field defect shows peripheral loss and is unusual in that the nasal field of the right

eye is not congruous, being divided by a large blind area which has a clear-cut horizontal axial border. This may be regarded as an incomplete quadrantic defect. The left field is characteristic of injury to the right optic radiation, or tract.



CASE S. E. 6.7.40.

In this case the fragment could not have involved the tract and, in spite of the incongruity, the lesion must be situated in the right optic radiation. The incongruity was confirmed by repeated examinations and lends support to the possibility, hitherto lacking satisfactory clinical evidence, that the fibres from the two eyes may not be completely mixed at this level.

Dr. W. Russell Brain: Out of 528 consecutive cases of head injury, almost all closed injuries, seen on an average ten months after the injury, only 53 patients, or 10%, showed any ophthalmological symptoms, as follows:

| | | | | | | |
|-------------------------------------|-----|-----|-----|-----|----------|---------------|
| Ophthalmoplegia | ... | ... | ... | ... | 26 cases | 5.0% of total |
| Pupillary abnormalities | ... | ... | ... | ... | 15 " | 2.8% " " |
| Nystagmus | ... | ... | ... | ... | 10 " | 1.8% " " |
| Ptosis | ... | ... | ... | ... | 6 " | 1.0% " " |
| Injury to optic nerve | ... | ... | ... | ... | 5 " | 0.9% " " |
| Homonymous visual field defect | ... | ... | ... | ... | 5 " | 0.9% " " |
| Disorder of higher visual functions | ... | ... | ... | ... | 6 " | 1.0% " " |

Ophthalmoplegia may be caused by an injury either (i) in the orbit, (ii) between the orbit and the brain-stem, or (iii) in the brain-stem. (i) In at least 9 of the 26 cases of ophthalmoplegia the injury was in the orbit. Orbital injuries may involve only a single muscle, especially one of the obliques, and the ocular palsy is often associated with evidence of bony injury to the roof or floor of the orbit and coincident damage to the supra-orbital or infra-orbital nerve. (ii) In 13 cases the lesion appeared to be between the orbit and the brain-stem. This group included 9 out of the 10 cases of unilateral external rectus palsy. There were three cases of complete or partial 3rd nerve palsy, one due to traumatic aneurysm of the carotid and cavernous sinus. (iii) There were only four cases in which persistent ophthalmoplegia was due to a lesion of the brain-stem. In 3 of the 4 cases the ocular movement lost was conjugate convergence, which in one was associated with weakness of conjugate elevation.

Pupillary abnormalities were present in 15 cases. Apart from the 3 cases of 3rd nerve palsy and one case of subdural haematoma, the lesion appeared to be in the mid-brain. Inequality of the pupils is the commonest abnormality, but the difference in size is not usually great: the larger is usually the abnormal one. Either the reaction to light or that to accommodation may be sluggish, and on one or both sides, but in my experience it is rare for both to be affected together. Iridoplegia is thus a commoner persistent symptom of mid-brain damage than ophthalmoplegia.

Ptosis is more unusual, and, apart from 3rd nerve lesions, was found in only 4 cases and then was only slight.

Nystagmus was present in 10 cases: in 8 cases it was interpreted as a symptom of mid-brain damage: in the other two a rotary nystagmus to one or both sides was of vestibular origin, and was associated with deafness resulting from injury to the internal ear.

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Mr. Wylie McKissock: The necessity of closer collaboration between ophthalmologist and neuro-surgeon has become more evident in time of war, for the orbit and its contents, together with the intracranial visual pathways, form a common territory for the specialists in these two branches of surgery.

Many cases of war injury involve both orbital and intracranial contents and require the services of both specialists for their adequate treatment. Believing, as we do, that there is no great urgency in the excision of wounds involving the skull and its contents there is always ample time to achieve the necessary combination of specialists who should, in many cases, operate together.

An attempt to produce a clear-cut classification on anatomical grounds of the possible varieties of injury to the visual mechanism has proved unsatisfactory; here is a simpler form of subdivision based largely upon the types of injury we have met: (I) Injuries involving the orbital contents alone. (II) Injuries involving the orbit and accessory nasal sinuses. (III) Injuries involving the orbit and the intracranial membranes. (IV) Injuries involving the orbit with penetration of the brain. (V) Injuries involving the visual pathway from the optic foramen to the occipital cortex.

I.—INJURIES INVOLVING THE ORBITAL CONTENTS ALONE

The entire diagnosis and treatment of this type of injury naturally devolve upon the ophthalmic surgeon and it would be presumptuous of me to attempt to discuss them.

It should, however, be remembered that there may be an associated cranio-cerebral injury of the closed type. Many patients, therefore, with a wound of the orbital contents alone, especially if produced by blunt injury, where there has been a history of unconsciousness at the time of wounding should be referred to the neurologist or neurological surgeon for full neurological examination, radiological investigation of the skull and lumbar puncture to determine the presence, degree or absence of associated closed injury to the brain or to its covering membranes. In the majority of cases showing evidence of intracranial damage the routine dehydration treatment of restricted fluid intake, rectal injection of magnesium sulphate and repeated lumbar puncture, designed to keep the intracranial pressure within normal limits, may well be carried out in the ophthalmic department with but occasional visits from the neurologist or neurological surgeon. The important point to remember is that such associated closed head injury may well be present especially in casualties from bombing.

II.—INJURIES INVOLVING THE ORBIT AND ACCESSORY NASAL SINUSES

Injuries to the orbit associated with an opening into one of the accessory nasal sinuses may be produced by blunt injury to the surface of the head or eye, by a penetrating wound of the orbit entering an accessory sinus, or by a missile passing through the skull and brain and thence invading an accessory sinus as well as the orbit.

Of the first variety we have had one example (N.) in which a young man was struck by a fairly large piece of metal which flew off a Diesel pump. The metallic object struck the head violently over the anterior surface of the left orbit but did not render the patient unconscious. He suffered almost immediately from severe swelling of both eyelids on the left side and extreme pain in the eye associated with vomiting. When examined he showed true emphysema of the orbit, proptosis of the left eye with marked chemosis, and some subconjunctival hæmorrhage. He had, in addition, a partial left anosmia and it was felt that he must have sustained a fracture into the ethmoid air cells which had permitted the extravasation of air into the orbital cavity. All movements of the eye were limited and extremely painful and there was diplopia but no external wound over any part of the head. After careful investigation there proved to be no evidence of associated intracranial injury and at the end of four weeks the patient was discharged well and symptomless but still with slight residual weakness of the left superior rectus muscle.

Of the second variety the following is a good example (E. G.). A very obese woman of 54, already blind in the right eye for many years, received an injury from flying pieces of cement entering the left eye, left inner canthus, and the root of the nose. The left eye was disorganized and a deep cavity existed over the bridge of the nose at the level of the glabella. It proved unfortunate for us that the patient had severe varicosities of the veins of each leg, a large brawny induration on the left leg from an old varicose ulcer and a dirty ragged wound on the left ankle. Twenty-four hours after the injury Mr. Goldsmith excised the left eye, after which, through a small scalp flap, we excised the whole wound. This involved opening and partially excising both frontal sinuses, the anterior and middle ethmoid air cells, and removing the nasal process of the frontal bone on each side. Many foreign bodies lay deeply in the cavity but the cribiform plate was intact and nowhere was the dura mater exposed. After dusting with sulphamidamide powder the wound was lined with gutta-percha tissue and loosely packed. At the same time the wound of the left ankle was completely excised and sutured by Professor Pilcher. On the fourth post-operative day the wound looked very healthy and there was no discharge from it. A swab taken at this time grew only a few colonies of *Staphylococcus aureus*. On the sixth post-operative day the patient suddenly died of a large pulmonary embolus derived, presumably, from the left femoral vein.

The third variety of this type of injury to the orbit and to an accessory nasal sinus, by a missile entering the cranium from above, has not occurred in our series.

III.—INJURIES INVOLVING THE ORBIT AND THE INTRACRANIAL MEMBRANES

Foreign bodies or penetrating wounds of the orbit may impinge upon the deep boundaries of this cavity and the former may become impacted in the bony orbital wall, thus protruding into the subarachnoid space, or pass completely into the cranial cavity. Where the foreign body is comparatively small we have adopted an expectant policy and all cases where this course has been followed have, so far, given comparatively good results.

One case (M.) had a small bomb fragment which had entered the inner end of the left lower lid and become impacted in the wing of the sphenoid at the back of the orbit. The cerebrospinal fluid contained a moderate amount of blood and it seemed reasonable to suppose that this was due to damage to the covering membranes of the brain at the

As in the case of ophthalmoplegia of mid-brain origin, iridoplegia; ptosis and nystagmus all tend to disappear within a few months of a head injury. The prognosis of ophthalmoplegia due to lesions between the brain-stem and the orbit and in the orbit itself is more uncertain. Certainly recovery may occur with either, but is unlikely to be complete when the initial paralysis is severe, when there is much deformity of the orbital wall, or when ophthalmoplegia is still present six months after the injury.

INJURIES INVOLVING THE VISUAL PATHWAYS

Papilloedema is not uncommon in the acute stage of head injuries and is probably a symptom of increased pressure of the cerebrospinal fluid. It appears almost always to subside completely without any impairment of vision and it is very rare to see even slight secondary optic atrophy in the victims of a head injury.

Direct injuries to the visual pathways occurred in 10 cases in this series: just under 2% of the total. In 5 cases the optic nerve was injured. The cause, as Traquair (1931) suggests, is probably the rupture of vessels passing from the sheath into the nerve substance.

Traumatic lesions of the optic chiasma appear to be uncommon or at least not commonly recognized. Traquair, Dott and Russell (1935) discovered 27 cases in the literature and added three of their own, two in 450 cases of head injury. The visual fields resemble those found in cases of compression of the chiasma by a tumour, and these authors believe that the lesion is not a direct tear of the chiasma but a vascular injury.

In 5 of my cases there was a visual field defect of homonymous distribution, but in none was the hemianopia complete. In 3 the lower quadrants suffered more than the upper, and in one the defect was limited to the lower quadrants. Homonymous field defects are thus uncommon after closed head injuries. Penetrating injuries of the optic radiation usually produce a complete hemianopia or large irregular areas of blindness, but quadrantic or other regular defects may occur.

THE PARIETO-OCCIPITAL CORTEX AND THE HIGHER VISUAL FUNCTIONS

The importance of vision in every sphere of human activity has led to an extremely complex organization of higher visual functions which depend upon the integrity of the parieto-occipital region or visuo-psychic area of the brain. The following is a brief classification of the principal disorders of the higher visual functions, from some or other of which 6 patients in this series suffered.

(1) *Alexia*: This was present in 4 out of the 6 cases, in varying degrees of severity.

(2) *Visual object-agnosia*: This was present in the early months after the injury in one case.

(3) *Loss of topographical memory*: This was present in two patients who in consequence were unable to describe their houses, or find their way about.

(4) *Loss of visual imagery*: In one case this was the sole sequel of a severe head injury. The patient five years later is completely unable to form any visual picture. He cannot visualize his wife or his house or any familiar object, and though he still dreams he no longer dreams in visual images.

It is probable that all the foregoing disorders are related to cerebral dominance and only occur as a result of left-sided lesions in right-handed persons.

(5) *Defective visual localization in space*, on the other hand, may be limited to either half-field as a result of a lesion of the opposite parietal lobe (Brain, 1941). It is characterized by an inability to appreciate the absolute and relative position of objects seen, especially in relation to the patient's sagittal plane. This was present in 2 cases. Bilateral lesions cause a much more severe disturbance than unilateral ones. Defective visual localization leads to misdirected grasping and pointing and disordered ocular movements.

(6) *Visual inattention*, present in one case, may similarly be limited to one half-field.

(7) Finally, there is one disorder which I mention for completeness, although I have seen no traumatic example of it: *inattention to, or agnosia for one-half of space*. This has been observed as a result of lesions of the right parieto-occipital region only and is usually, but not invariably, associated with a left homonymous hemianopia. Imperception of the left half of space, which is a more profound disturbance than merely a hemianopia, leads to one form of visual disorientation (Brain, 1941).

Disorders of the higher visual functions are likely to occur only with severe head injuries. Though gross confusion may last for weeks, improvement continues for many months, but recovery is never complete.

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Section of Orthopædics

President—C. LAMBRINUDI, F.R.C.S.

[June 13, 1942]

Meeting at R.A.F. General Hospital

REHABILITATION IN ORTHOPÆDIC CASES

In the course of a ward visit, the work of clinical secretaries, clinical photographers, radiographic printers, and rehabilitation orderlies was demonstrated. Patients with limbs immobilized in splints, in traction and in plaster showed the special exercises they perform for five minutes every hour of the day. Sandbags of graduated weight, made by the patients themselves were at the foot of nearly every bed. Simple gymnastic apparatus, billiard tables, dart boards, and tenniquits were in the solarium. The orderlies taught the patients exercises, and supervised occupational therapy, Morse communication across the ward, and simple games in the grounds. A film showing all stages of treatment from early ward exercises, to treatment in special rehabilitation centres, and finally in flying units was shown by Flight Lieutenant A. Zinovoff.

Dislocation of the Knee-joint with Capsular Interposition

By Group Captain H. OSMOND CLARKE

COMPLETE dislocations of the knee are rare; they are usually easy to reduce, and if reduction is efficiently maintained a stable, mobile and, sometimes, normal joint can be expected. Certain complications may occur; for example, external popliteal nerve lesions produced by over-stretching in forcible adduction injuries, or by inclusion of the nerve in the joint; more rarely gangrene has followed rupture or occlusion of the popliteal artery. The following case is an example of difficulty in reduction and the cause for that difficulty is discussed.

On May 25, 1941, a Czech air-observer aged 26, a slender youth of magnificent spirit, sustained a dislocation of the knee in which the tibia was displaced postero-laterally on the femur. All manipulative efforts failed to effect complete reduction. The backward displacement was easily overcome but the lateral shift persisted stubbornly. On attempting to force reduction, the skin furrowed over the medial joint line as if it were being drawn into the joint. Unfortunately the initial violence had forced the medial femoral condyle so hard against the overlying skin that an area was devascularized and subsequently formed an ulcer. While awaiting healing of the ulcer, correction of the backward displacement was maintained in a plaster of Paris splint. This undoubtedly made the subsequent operation easier and is well worth while if, for one reason or another, a necessary open correction must be postponed. At operation the morbid anatomy of the injury was easily demonstrated. The capsule and quadriceps expansion was torn from the vastus internus, the internal ligament was avulsed from its femoral attachment, and the capsular flap lay interposed between femoral condyle and tibial tuberosity.

The condition may be compared with forcing a large button (the medial femoral condyle) through an irregular buttonhole (the tear in the capsule) which becomes so entangled around the button that manipulative extrication is impossible. A torn internal cartilage was removed, the capsule and internal lateral ligament dissected off the cruciate ligaments and the fat pad, and reinserted to the femoral condyle. A completely congruous reduction was achieved and held in plaster for twelve weeks. To-day this officer, following intensive rehabilitation and two manipulations, walks without a trace of limp, has a stable joint with 80 degrees of flexion from the straight position under powerful muscular control. He has been on ground duties for the past four months. It was a great pleasure to him, and most gratifying to me, when a few days ago I recommended his return to flying duties.

A Method of Bone Grafting the Tibia and Fibula

By Wing Commander J. R. ARMSTRONG

In bone grafting a fracture of the tibia and fibula the surgeon has three objects: (1) To reduce the fracture, placing the fractured surfaces in intimate contact and restoring normal alignment; (2) to secure the reduction by internal fixation which is mechanically stable; (3) to establish favourable conditions for bony union. Bone union is facilitated when the operation restores normal architectural structure, when bone absorption and new bone formation is minimal, and when close and stable contact is secured between large areas of raw bone on the graft and host, so that revascularization is rapid and easy.

site of the foreign body. The left eye was completely disorganized and was removed by Mr. Goldsmith but the metallic foreign body was left untouched. The intracranial bleeding soon ceased and simple dehydration therapy proved adequate to control the intracranial condition. The eye socket healed well and rapidly. Seventeen months later the patient was alive, symptomless and working.

The second case (C.) was essentially similar but here the foreign body, again a bomb fragment, passed through the back of the orbit and lay on the tip of the left petrous bone. This piece of metal had entered through a very small opening in the left upper lid, disorganized the eye, and passed on. The patient did not come to us until some weeks after injury, when he was admitted for persistent headache and numbness of the left side of the face, the left eye having been excised elsewhere. He proved to have complete paralysis of the left 5th nerve and a partial left facial palsy. He was a poor specimen of the complaining, compensation-hunting type and it was felt that excision of the foreign body, whilst proving extremely difficult technically, would do little towards cure of the headaches. No operation, therefore, was undertaken. Nine months later the patient was still under treatment for contraction of the left eye socket but had developed no serious intracranial complication from the presence of the bomb fragment.

IV.—INJURIES INVOLVING THE ORBIT WITH PENETRATION OF THE BRAIN

In this class of case the treatment depends to a great extent upon the nature of the wound and its mode of production. If produced by a small, high velocity, metallic fragment entering through the orbit and eventually becoming embedded in the brain there seems reason to believe that little need be done except for the local condition in the eye, if this has been damaged. Here is an example.

A young soldier suffered a wound from a small metallic bomb fragment which entered the outer end of the left lower lid, passed obliquely through the lateral half of the globe and then upwards through the roof of the orbit into the postero-inferior part of the left frontal lobe. The only surgical treatment carried out was excision of the eye by Mr. Goldsmith, the wound track being left severely alone. Dehydration therapy and repeated lumbar puncture were instituted for relief of the crano-cerebral wound. During the three weeks immediately following the injury there was an extensive cellular reaction in the cerebrospinal fluid and three pyrexial attacks at five-day intervals were associated with headache, photophobia and neck rigidity. After a course of A. & B. 693 the patient settled down completely and made a perfect recovery. Fourteen months later, when last heard of, the patient was working and in excellent general health. No complication would appear to have arisen as a result of the metallic foreign body lying in the frontal lobe.

At the other end of the scale is the patient in whom there has been gross damage to external tissues and gross penetration of bone and brain.

A woman of 35 (C. W.) illustrates this type of lesion very well. Part of the lath and plaster ceiling of her house was driven through the upper and inner part of the orbit and the inner end of the right supra-orbital ridge, destroying the right eye in the process. Some of the foreign bodies passed through the roof of the orbit from below upwards, coming finally to rest in the substance of the right frontal lobe. The right frontal sinus was fractured posteriorly and there was air in the subarachnoid space. Here was a very definite case for combined operation. Twenty-four hours after the time of injury Mr. Goldsmith excised the eye under local anaesthesia and cleaned and repaired the inner canthus, after which we carried out a complete excision of all loose bone fragments, damaged tissue and foreign bodies including many pieces of wood from the brain itself. The dural openings were closed with sheets of tissue from the scalp and the wound was left widely open, being lined with gutta-percha tissue and then gently packed. This patient was discharged from hospital three months from the day on which she was injured and was then symptomless and without abnormal physical signs in the central nervous system. She was subsequently operated upon by Mr. Aowlm who reconstituted her supra-orbital ridge, filled in the frontal bone defect and produced correct alignment of the eyebrow. At the present time, fourteen months after the injury, she is working, has no symptoms, and no obvious facial deformity.

Between the two examples in this class which I have endeavoured to illustrate lie many intervening variations, each one of which must, of necessity, be treated on its own merits, but would appear to me to require continuous supervision by both specialists throughout the period of treatment.

V.—INJURIES INVOLVING THE VISUAL PATHWAY FROM THE OPTIC FORAMEN TO THE OCCIPITAL CORTEX

Of the many possible injuries to the visual pathway from the optic foramen to the occipital cortex, with their attendant multiplicity of signs from total blindness through field defect to disturbance of visual memory and interpretation, we have had a few scattered examples.

The many complications which may arise in the course of treatment of the type of injury which I have been discussing are all too obvious. The threats of sepsis from the initial injury and from hospital infection of the operation wound, hang over us perpetually. The advent of the sulphonamides has done much towards prevention, as well as cure, of sepsis in its many forms but nevertheless the risks of meningitis, of osteomyelitis of the skull and of local abscess formation still exist. I believe that dusting of the post-operation wounds with sulphonamide powder may do much to prevent the development of such infection in its early stages and I have now adopted it as a routine measure.

One of the many points upon which I would like advice is that of the ultimate fate of these small metallic foreign bodies which pass through the orbit into the brain substance, which would prove extremely difficult to remove surgically and, on removal, would inevitably produce more brain damage than was already present. So far not one of our cases has fallen a victim to a brain abscess developing at the site of such a foreign body but the possibility of this development is a very real one.

The scarring around such foreign bodies and along the track of their entry may later give rise to a pull on the cerebral cortex, to traction diverticulum from the ventricular system and the subsequent development of traumatic epilepsy. Here again the investigation of such late sequelæ as brain abscess and traumatic epilepsy provides further opportunities for our ophthalmic associates, to aid us in the accurate localization of these lesions through the medium of the visual field defects which may occur.

Section of Orthopædics

President—C. LAMBRINUDI, F.R.C.S.

[June 13, 1942]

Meeting at R.A.F. General Hospital

REHABILITATION IN ORTHOPÆDIC CASES

In the course of a ward visit, the work of clinical secretaries, clinical photographers, radiographic printers, and rehabilitation orderlies was demonstrated. Patients with limbs immobilized in splints, in traction and in plaster showed the special exercises they perform for five minutes every hour of the day. Sandbags of graduated weight, made by the patients themselves were at the foot of nearly every bed. Simple gymnastic apparatus, billiard tables, dart boards, and tennisquits were in the solarium. The orderlies taught the patients exercises, and supervised occupational therapy. Morse communication across the ward, and simple games in the grounds. A film showing all stages of treatment from early ward exercises, to treatment in special rehabilitation centres, and finally in flying units was shown by Flight Lieutenant A. Zinovieff.

Dislocation of the Knee-joint with Capsular Interposition

By Group Captain H. OSMOND CLARKE

COMPLETE dislocations of the knee are rare; they are usually easy to reduce, and if reduction is efficiently maintained a stable, mobile and, sometimes, normal joint can be expected. Certain complications may occur; for example, external popliteal nerve lesions produced by over-stretching in forcible adduction injuries, or by inclusion of the nerve in the joint; more rarely gangrene has followed rupture or occlusion of the popliteal artery. The following case is an example of difficulty in reduction and the cause for that difficulty is discussed.

On May 25, 1941, a Czech air-observer aged 26, a slender youth of magnificent spirit, sustained a dislocation of the knee in which the tibia was displaced postero-laterally on the femur. All manipulative efforts failed to effect complete reduction. The backward displacement was easily overcome but the lateral shift persisted stubbornly. On attempting to force reduction, the skin furrowed over the medial joint line as if it were being drawn into the joint. Unfortunately the initial violence had forced the medial femoral condyle so hard against the overlying skin that an area was devascularized and subsequently formed an ulcer. While awaiting healing of the ulcer, correction of the backward displacement was maintained in a plaster of Paris splint. This undoubtedly made the subsequent operation easier and is well worth while if, for one reason or another, a necessary open correction must be postponed. At operation the morbid anatomy of the injury was easily demonstrated. The capsule and quadriceps expansion was torn from the vastus internus, the internal ligament was avulsed from its femoral attachment, and the capsular flap lay interposed between femoral condyle and tibial tuberosity.

The condition may be compared with forcing a large button (the medial femoral condyle) through an irregular buttonhole (the tear in the capsule) which becomes so entangled around the button that manipulative extrication is impossible. A torn internal cartilage was removed, the capsule and internal lateral ligament dissected off the cruciate ligaments and the fat pad, and reinserted to the femoral condyle. A completely congruous reduction was achieved and held in plaster for twelve weeks. To-day this officer, following intensive rehabilitation and two manipulations, walks without a trace of limp, has a stable joint with 80 degrees of flexion from the straight position under powerful muscular control. He has been on ground duties for the past four months. It was a great pleasure to him, and most gratifying to me, when a few days ago I recommended his return to flying duties.

A Method of Bone Grafting the Tibia and Fibula

By Wing Commander J. R. ARMSTRONG

In bone grafting a fracture of the tibia and fibula the surgeon has three objects: (1) To reduce the fracture, placing the fractured surfaces in intimate contact and restoring normal alignment; (2) to secure the reduction by internal fixation which is mechanically stable; (3) to establish favourable conditions for bony union. Bone union is facilitated when the operation restores normal architectural structure, when bone absorption and new bone formation is minimal, and when close and stable contact is secured between large areas of raw bone on the graft and host, so that revascularization is rapid and easy.

The usual inlay or onlay bone grafting technique does not always give sufficiently stable fixation, and it is not easy to secure accurate alignment of the fragments. I have, therefore, used a modification of the so-called "split-bone" technique described by Gill in fractures of the radius and ulna.

The tibia is exposed subperiosteally along its whole length. The fracture is reduced and held in position by clamps of the Lowman type applied to the posterior surface of the bone, so that the anterior two-thirds are left free. Care is taken to secure correct alignment of the fragments and it is often necessary to refracture or divide the fibula. Using a large diameter single saw the bone is split along half its length, in such a way that two-thirds of the divided area lie on the longer fragment, and one-third on the shorter fragment. The large graft thus produced is laid across the fracture and fixed with four vitallium screws; the gap is filled with the shorter fragment. Radiograms are taken to confirm the accuracy of alignment of the fragments. The normal bowing of the tibia varies so much from individual to individual that it is not always easy to ensure correct alignment without radiograms. If slight medial or lateral bowing is shown, this is easily corrected by removing the lowest screw, angling the lower fragment as much as may be necessary, and reinserting the screw. When the alignment is satisfactory the periosteum and skin are closed, and the fracture is treated in plaster in the usual way.

This operation is mechanically sound; the technique is simple; alignment can be controlled perfectly; the operation involves only the injured limb, no graft being taken from the normal limb; it is followed by rapid and certain union.

Impacted Fracture-dislocation of the Tarsal Navicular

By Wing Commander I. L. DICK

ON 6.11.41 P. E. C. was pulled up by a balloon cable in the dark and fell about 10 feet sustaining a fracture-dislocation of the talo-navicular joint. The fracture extended transversely across the lower third of the navicular and the large upper fragment was displaced dorsally on to the upper surface of the head of the talus. Two attempts at manipulative reduction failed. A third attempt at manipulative reduction aided by strong skeletal traction by means of a pin through the os calcis and a Kirschner wire through the bases of the metatarsals also failed.

While skeletal traction was maintained the displaced bone was exposed through a short dorsal incision. As the soft tissue was cleared from its proximal surface the navicular sprang back into position. The reason for the failure of manipulative reduction had been the impingement and wedging of the sharp edge of the navicular into a small depressed fracture on the upper surface of the head of the talus. Reduction by open operation was effected with ease and with minimal disturbance of the soft parts. Radiograms showed that reduction was anatomically accurate and that the major portions of the articular surfaces of the navicular were undamaged. It was therefore hoped that avascular necrosis of the navicular and osteo-arthritis of the involved joints might not occur and primary fusion of these joints was not done. The foot was immobilized in plaster for ten weeks. Four weeks later he returned to duty able to walk 4 miles in complete comfort without swelling of the foot. Mid-tarsal movement was somewhat limited but it was painless throughout its range.

Two months later he reported pain in the foot as he walked and examination showed that mid-tarsal movement was more limited and painful. X-rays showed that, though the texture of the navicular was normal and there was no sign of avascularity, osteo-arthritis was already established in the talo-navicular and naviculo-cuneiform joints. These joints were accordingly fused on 10.6.42.

This case confirms the view that if open reduction is required for a fracture dislocation of the tarsal navicular, primary fusion of the involved joints is indicated. Even in closed fractures of the tarsal scaphoid in which manipulative reduction succeeds, consideration should be given to the desirability of primary fusion of the involved joints when there is distortion of the joint surfaces.

Bone Pegging the Carpal Scaphoid

By Wing Commander A. A. BUTLER

FRacture of the carpal scaphoid is a frequent Service injury, and in a large series of fractures seen in the Weeton Clinic bone grafting was considered advisable in 50 patients. Originally the technique of Burnett (1937) and later the technique of Armstrong (1941) was employed, but some difficulty was experienced in accurately placing a large drill in this small bone which lies in an oblique position. Armstrong (1941) has pointed out that the long axis of the scaphoid lies at an angle of 45 degrees to the transverse plane of the wrist, and 40 degrees to the long axis of the limb. A special arm rest was

therefore devised which holds the limb in this position, so that the long axis of the scaphoid is exactly vertical. In this way it is unnecessary to estimate difficult angles; the surgeon need only concentrate on keeping the drill strictly vertical. The rest holds the arm more securely than is possible by an assistant, and since the position is never changed, all radiograms are comparable and easily interpreted. The rest, which is made of aluminium and can be sterilized, is fitted with a shelf to hold an X-ray plate in the correct position so that radiograms can be taken at various stages throughout the operation.

Brachial plexus block anaesthesia is used. The incision is placed over the tubercle of the scaphoid. A $\frac{1}{16}$ in. motor drill is inserted vertically to a depth of $\frac{3}{4}$ in. entering the lateral side of the scaphoid tubercle which is the centre of the vertically held bone. Radiograms usually show that the drill is correctly placed on its first insertion; if this is not so, the necessary correction is easily made. When the position is perfect the drill hole is enlarged to $\frac{7}{32}$ in. diameter by means of a cannulated trephine passed over the drill guide. A graft of the same thickness is taken with a dowel cutter from the olecranon. The tip of the graft is bevelled, and driven into the scaphoid drill hole. The position is checked by X-ray, the fracture impacted, the wound closed and a full-arm padded plaster applied. On the twenty-first day a forearm skin-tight plaster is applied, firmly moulded round the wrist-joint and palm, and extending almost to the interphalangeal joint of the thumb. The patient then resumes light duty and returns monthly for supervision.

Using this technique the bone graft has been well placed every time. The danger of damage to the articular surface due to a wrongly placed drill has been avoided, even in fractures near the proximal pole which present the greatest difficulty. The operation is usually completed within thirty minutes. It has been used in cases of established non-union, of delayed union, and in recent fractures in which delayed union is very probable such as fractures with mid-carpal dislocation or with wide displacement of the fragments. It is true that fractures with delayed union unite even without bone grafting if plaster immobilization is continued long enough, but in these cases it is believed that grafting is justified because of the considerable saving of time. In fourteen cases union was obtained in an average time of three months. In fractures with established non-union, grafting is considered advisable in order to prevent the onset of arthritis. In eighteen cases, union occurred in an average of five months. Of the remaining cases in the series eight are still in plaster, and ten having returned to duty have been posted elsewhere so that late follow-up has not been possible.

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Fracture Internal Malleolus and Diastasis Inferior Tibio-fibular Joint

By Wing Commander D. M. MEEKISON

For some years the writer has employed the method of open reduction and internal fixation of isolated fractures of the internal malleolus using a metal screw; since the introduction of vitallium this has been the metal of choice. Screw fixation has also been used for cases of diastasis of the distal tibio-fibular joint. The types of injury may be analysed thus:

[illegible]

Of the 34 patients, 22 were air crew, whose early return to duty is of great importance. The average age of 22½ years (excluding 1 patient of 57) was very favourable, only two other patients being over 30.

The operative procedure used is dictated by the type of injury. In all cases the pre-operative preparation is the same, i.e. thorough cleansing with soap and water, spirit and acriflavine twice or three times daily for a minimum of two days, preferably three. The limb is kept wrapped in sterile towels. In all cases a direct approach is made, because in the author's experience a scar over a bony prominence never causes disfigurement or inconvenience. All operations are done under general anaesthesia and with a tourniquet.

Fracture internal malleolus.—Sufficient exposure is made to visualize both anterior and posterior margins of the tibia. All fractures in this series showed interposition of tissue between the malleolus and shaft; occasionally a small loose fragment is found in the joint. The fracture line is thoroughly cleaned out and the fragment replaced in accurate position. The two criteria of accurate reduction are fitting of the "pattern" of the fracture line, and congruity of the anterior and posterior margins of the tibia. While

the malleolus is held in position by the assistant with a sharp hook, a vitallium screw is passed obliquely upwards through the fragment into the shaft, after a suitable drill hole has been made. The head of the screw is buried under the internal collateral ligament. The wound is closed and a padded cast applied. This is changed after ten to fourteen days to a new non-padded cast with a walking heel of sorbo rubber, in which weight bearing is permitted. Walking irons are not used in the plaster; they are to be condemned. Fixation is discontinued in six or seven weeks, and this is followed by viscopaste for a week or more. Rehabilitation may or may not be necessary.

Fibular fractures with diastasis.—A direct approach is made and when the fracture is accurately reduced it is held with a 4 hole vitallium plate, 3 short screws holding the fibula and one long screw in the second from top hole, transfixing the fibula to the tibia. The plate and screws may have to be removed later owing to their prominence.

Simple diastasis.—It should be noted that diastasis of the distal tibio-fibular joint may easily be missed. All injured ankles should be X-rayed in inversion and eversion when the original radiogram reveals no fracture. Unless the diastasis is diagnosed the patient may be left with a "sloppy" ankle. If radiograms show widening of the tibio-fibular joint and lateral movement of the astragalus, a short incision is made 1 in. above the ankle-joint and the fibula fixed to the tibia with a simple, long vitallium screw while the distal tibio-fibular joint is held snugly in position.

There was only one complication—sepsis in one case. This was the 57 years old patient with a severe fracture of the ankle, who was operated upon in the presence of an abrasion, not thoroughly healed, adjacent to the line of incision. At his cost, the lesson of the danger of abrasions has been driven home. Of the 34 cases, 14 have normal ankles, 17 are still under treatment without complications, 2 have good results (arthritic changes in 1, and flat foot in 1) and one has a bad result (sepsis). One sergeant with a simple diastasis made a trip to the Ruhr thirty-seven days after operation, and has what seems to be a normal symptom-free ankle.

Co-operation Between Plastic and Orthopædic Surgery

By Wing Commander GEORGE H. MORLEY, R.A.F.

FIVE cases are presented to indicate the field of co-operation between plastic and orthopædic surgery.

CASE I.—*Free split-skin graft, first day after accident.* A pilot sustained a comminuted fracture of both bones of the forearm with serious fragmentation of the bones and dislocation of both superior and inferior radio-ulnar joints. There was "degloving" of skin from the elbow, all except a narrow strip 4 in. by 1 in. being torn downwards from 2 in. above the elbow to 2 in. below the joint.

The day after injury, the skin flaps were united across the anterior aspect of the joint, and the remaining denuded area covered with a Thiersch graft from the thigh. The "take" was nearly 100%, but the upper 2 in. of the skin flap, being stripped from its blood supply, underwent necrosis. One month later pinch grafts were applied to this area. The skin was completely healed two months after injury, thus making it possible to perform a bone graft to the ulna at the third month, a bone graft to the radius at the fourth month and an excision of the head of the radius with inferior radio-ulnar arthroplasty at seven months. He is now on limited flying duties ten months after injury, with full movement of the elbow and wrist.

CASE II.—*Delayed primary split-skin graft, to heal large granulating wound.* Sergeant Pilot E. received a bullet wound of the right hand, most of the index finger being shot away, and the index metacarpal subsequently removed. He was first seen three weeks after injury, with a large granulating wound and stiffness of fingers and thumb. At the first change of plaster a split-skin graft was applied as a dressing. There was almost 100% "take", and within ten days it was possible to begin rehabilitation of the hand and regain mobility of the finger-joints. The transplanted skin lay directly upon the irregular carpal bones, and the cosmetic result was poor. A direct delayed flap of abdominal skin was therefore applied after removing all scar tissue, thus improving both function and appearance. Nine months after injury, owing to restriction at the web of the thumb, a local transposition of skinflaps was performed. He will shortly resume flying duties.

CASE III.—*Cross leg flap for compound fracture of tibia.* L.A.C. H. sustained a compound infected fracture of the left tibia, which was not united eleven months later. Bone grafting was impracticable owing to an unsound scar, involving two-thirds of the circumference of the limb at the level of fracture. There was considerable oedema of foot and ankle. A cross leg flap was transferred from the posterior aspect of the right calf. Five weeks later the wound was soundly healed, and shortly afterwards the fracture united firmly without bone grafting. He has now been on duty for four months, the fracture being firmly united, the oedema of foot and ankle completely relieved and the transferred flap settled and stable. This case not only illustrates the use of a cross-leg flap to afford a safe area of skin for surgical approach to the tibia, but also the relief of such a flap to tissues which, being distal to areas of dense scar tissue, are subject to stasis of blood and lymph, with resulting congestion and lymphoedema.

CASE IV.—*Excision and grafting of unsatisfactory burn scar.* Corporal C., as a result of an aircraft crash, sustained compression fractures of the 2nd and 3rd lumbar vertebrae, and third degree burn of the left hand. After tanning elsewhere, the burn was treated by the saline-sulphanilamide method. The scar was heavy, contracted and unstable, frequently cracking with painful fissures. Excision of the scar involved removal of almost the whole dorsum of the hand and wrist. A sheet of split-skin graft, fixed with sutures and immobilized with marine sponge, was applied. The result is a hand with 100% of movement in all joints, and with a supple and fully stable skin on the dorsum.

CASE V.—*Use of pinch grafts.* A pilot officer crashed and sustained a comminuted fracture of the right femur, facial lacerations, burns of the face, left thigh, leg and both ankles. In consequence of the bone injury, saline bath treatment was impracticable and the burns were congealed with triple dye. Later, the burn of the left thigh and leg required skin grafting, and pinch grafts were applied, with successful healing within three weeks. Full movement of the knee was regained. This is an example of the use of pinch grafts in a case where three factors indicate their use: (a) The cosmetic appearance was unimportant; (b) the donor area was limited as compared with the defect to be epithelialized; (c) the granulations were slightly infected.

The wide field of plastic-orthopædic co-operation may be summarized:

(1) *Early healing of wounds, especially wounds with skin loss.*—Early epithelialization limits the scar tissue laid down in the process of healing and lessens soft tissue contrac-

ture. Free skin grafts should therefore be applied either at the time of the primary operation or at the first change of plaster. These grafts should be regarded as dressings, and they frequently heal such wounds within a few days. They will take on any normal tissue except bare bone or cartilage, particularly well on muscle. Sulphonamide powder may with advantage be applied beneath such grafts.

(2) *The late healing of wounds.*—It is usually advisable to excise all scar until normal tissues are exposed. It is futile to apply a skin graft to an indolent area of granulations which is unable to support the natural growth of epithelium from the periphery because of its dense fibrous tissue base.

(3) *The provision of a safe area of skin through which a bone can be approached surgically.*—A flap of skin and subcutaneous tissue may be transferred directly from a conveniently situated area or transported from a more distant area in the form of a tubed pedicle graft. It is sometimes found that healing of the wound and relief from recurrent ulceration have removed the factors which were inhibiting union of the fracture. Bone grafting may thereby be avoided.

(4) *Providing an amputation stump with sturdy skin.*—Cross-leg flaps may be used to replace unstable scars on stumps where there is a shortage of skin, provided that there is no necrosis of bone. These flaps are particularly indicated when reamputation is inadvisable, e.g. because the stump is already short. In the case of the arm, direct flaps from the chest wall may be used.

(5) *Relief of soft tissue contracture.*—After excision of all scar tissue the resulting skin defect may be covered with a flap or with free grafts. Flaps are indicated where there is probability of recurrent contracture.

Fixation of Oblique and Spiral Fractures of the Tibia by a Single Vitallium Screw

By Wing Commander A. RONALD

A SHORT series of fractures of the tibia and fibula is presented to demonstrate the advantages of operative reduction and fixation by means of a single vitallium screw. The operation is performed seven to ten days after injury through a 4 in. incision. The fracture is reduced and the bone ends held in a Hey Grove's clamp. A vitallium screw of the correct length is then placed as nearly as possible at right angles to the line of fracture, so that it transfixes an equal thickness of bone in each fragment, and engages the cortex of each fragment. Correct placing of the screw is relatively easy in oblique and spiral fractures but more difficult when the fracture is transverse. In order to avoid subcutaneous projection of the head of the screw, a V-notch is cut $\frac{1}{4}$ in. deep. The hole is drilled from the apex of the notch, which is then deepened sufficiently to accommodate the head of the screw. The screw is driven home, the stability of reduction tested, periosteum closed, skin sutured and a padded plaster applied. Two weeks later, an unpadded plaster is applied. In spiral fractures, plaster fixation can usually be discarded in seven to nine weeks, and weight bearing in plaster may be safe even earlier than this. Transverse and comminuted fractures are immobilized in plaster for an average period of eleven to twelve weeks.

Treatment by this technique has been completed in 12 fractures of which 9 were spiral, 1 transverse and 2 comminuted. In every case union is sound by clinical and X-ray tests. The average time of union was several weeks less than the general average in tibial fractures treated by all methods, and many weeks less than in fractures treated by heavy skeletal traction. The instability of oblique and spiral fractures makes it necessary to prevent redisplacement either by internal fixation or by continuous traction. The advantage of this method of internal fixation is that perfect apposition and excellent fixation is secured with a minimum of foreign material, and rapid union is promoted. Unlike methods of continuous traction, the patient is ambulatory at an early date, his stay in hospital is minimized, and the period of immobilization in plaster is reduced. There is, therefore, minimal stiffness of knee and ankle joints and in all cases movement of these joints returned rapidly. The only disadvantage of the technique is the risk of sepsis; this is under the control of the surgeon and in normal conditions should not arise.

Traumatic Asphyxia and Disruption of the Pelvis

By Squadron Leader H. M. COLEMAN

A 20-year-old flight mechanic was returning to the dispersal point in a 30 cwt. lorry, when it overturned on a corner. He was pinned under the frame-work of the lorry, but was released in about two minutes. The weight was taken across his upper abdomen and lower chest, where he had minor bruising. He remembers a feeling of pressure across the abdomen spreading upwards as if the blood was being forced to the top of his body and head. He then lost consciousness, and next remembers being put on a stretcher with his legs tied together. His

vision was blurred as though looking through a mist. This gradually cleared, and within about three hours he was able to see normally.

His friend states that by the time he was in the ambulance, approximately ten minutes after the accident, he was conscious, breathing heavily and slowly, his chest red and his face turning purple. On admission to Station Sick Quarters a few minutes later he was fully conscious, face and chest a deep purple, bleeding from both ears and nostrils, subconjunctival haemorrhage in both eyes, pupils equal and reacted to light, pulse 84, respirations not increased. A few hours later he was seen by Air Commodore Stanford Cade, who reported that the face, forehead, chest and upper arms were violet in colour with oedema of the eyelids and conjunctivae and haemorrhage on both drums. B.P. 100/60. Patient shocked. Blood-stained urine was passed and a fractured pelvis and possible fracture of the base of the skull were diagnosed.

The next day, when transferred to P.M., R.A.F. Hospital, he presented a startling picture. The conjunctivae were blood red, oedematous, and gave the appearance of exophthalmos. The forehead, face, neck, chest and upper arms showed a deep violet red pigmentation. The absence of blanching under the pressure of a glass spatula showed that the blood was extravasated, and differentiated the condition from simple hyperæmia or congestion. The mark of his braces and collar were clearly outlined, by the absence of ecchymotic spots due to their supporting pressure. Even the outline of his collar stud could be seen.

The patient had also sustained fracture through both right pubic rami, a dislocation of the symphysis pubis, and a fracture through the sacral ala with upward dislocation of the left half of the pelvis. This was reduced and progress is satisfactory. There were also fractures of the transverse processes of the 3rd, 4th, and 5th lumbar vertebrae. Two ribs were fractured. Pulmonary oedema and congestion developed with râles in both lungs, rise in temperature, rapid pulse and increased respiration rate, but the radiographic appearances resembling military tuberculosis, noted in some cases of traumatic asphyxia, were not observed in this patient.

This is a classical example of the rare condition of traumatic asphyxia first described in 1837 by Ollivier, with typical "masque ecchymotique". The distribution of ecchymosis and the absence of pigmentation where light external pressure was maintained by braces, collar and collar stud, support the view that blood was forced backwards by pressure on the chest into the innominate, internal and external jugular veins which are unprotected by functioning valves, and therefore into the skin of the head and neck. The skin pigmentation gradually faded but did not go through the colour changes one might expect after blood extravasation. Within three weeks the skin appeared normal. There was, however, still some staining of conjunctivae two months after injury. Vision was normal $\frac{6}{6}$, there was no optic atrophy and no impairment of hearing.

Early Excision of Avascular Fragment of Fractured Carpal Navicular Bone

By Squadron Leader N. VERE-HODGE

THE accepted modern treatment of fractures of the waist of the carpal navicular bone with avascularity of the proximal fragment is prolonged immobilization in plaster with or without a bone grafting operation. The results are sometimes disappointing because loss of blood supply of the bone and degenerative chondritis lead to the rapid development of arthritis of the wrist-joint. No advantage is to be gained by late excision after arthritis has developed, but it has been suggested that early excision of the proximal fragment performed as soon as avascularity is recognized, may prevent the complication.

A flight mechanic, aged 21, injured his wrist in April 1941. He had continual pain, there was only a shiver of active movement, and the grip was weak. Two months later, when he reported sick, radiological examination showed a fracture of the waist of the carpal navicular. Plaster was applied, but after six weeks, radiograms showed that the proximal fragment was markedly avascular. Operation was performed without delay and the proximal fragment was excised. Post-operative treatment consisted of three weeks' immobilization in plaster followed by gradual mobilization and strengthening exercises.

He returned to his trade as a flight mechanic three months after operation. The wrist was comfortable; the grip was good; he had 50 degrees active dorsiflexion and 30 degrees palmar flexion. Six months later he states that he can lift weights up to 100 lb., and that even after a heavy day's work he has no symptoms. The wrist is not quite as good as before the accident, but it is infinitely better than before operation. There is now 55 degrees active dorsiflexion (60 degrees on the normal side) and 55 degrees palmar flexion (75 degrees on the normal side). Radiograms show no evidence of arthritis; the fractured surface of the remaining fragment of the navicular is rounded and smooth.

No general conclusion is to be drawn from a single case, but in view of the prolonged immobilization which would have been needed to secure union of this fracture, the probable necessity for a bone grafting operation, and the great probability of arthritis, the rapid return of good function in this patient after early excision of the avascular and necrotic fragment, is worthy of record.

Fractures of the Head of the Radius

By Flight Lieutenant A. E. BURTON

THE results of investigation in a consecutive series of 50 fractures of the head of the radius have been analysed. The age-incidence ranged from 18 to 41 years (a typical R.A.F. service age-group), and 70% were air crew members or skilled technicians. Three groups of fractures were identified, each with a distinctive mechanism of injury. Type 1: The impaled fracture from a fall on the outstretched pronated hand. The head of the radius was impaled on the capitellum which was injured at the same time that the head of the radius is fractured. The damage to the capitellum was seldom shown radiographically because only articular cartilage was involved, but the injury may be disclosed by a second X-ray fourteen days later. Type 2: The marginal fracture. This occurred as the

result of an abduction strain on the elbow with the forearm pronated; a segment of the articular surface was displaced. Injury to the capitellum sometimes occurred. Type 3: Undisplaced fissure fracture. This occurred from a direct injury to the elbow causing a transverse fissure fracture without displacement. In nearly all cases of impaled fracture, excision of the head of the radius was advisable, particularly in view of the frequency with which articular cartilage damage was disclosed at operation although not seen in X-rays. In marginal fractures excision was performed when there was displacement of the segment, or disturbance in the integrity of the articular plateau. Marginal fractures without displacement and fissure fractures were treated conservatively.

After three days' pre-operative skin preparation, the bone was excised through a short 2 in. incision directly over the head of the radius, deepened through the capsule with minimal dissection. Care was taken to avoid damage to the attachments of the orbicular ligament round the neck. The margin of the bone was smoothed off and a flap of periosteum placed over it. Active movements were begun twenty-four hours after operation but no massage or passive movement was used at any stage.

In no case in this series of 50 fractures was there instability of the superior or inferior radio-ulnar joints, cubitus valgus or new bone formation at the site of excision. The results are summarized below, the following criteria being used: *Excellent*—powerful arm, symptom free, full range of movement. *Good*—powerful arm, symptom free, less than 15 degrees limitation of extension movement. *Fair*—powerful arm, symptom free, more than 20 degrees limitation of extension movement. *Unsatisfactory*—residual symptoms and restricted movement.

(1) *Impaled fracture of radial head*—29 cases.

(a) *Excision*—20 cases. 17 good or excellent—average disability period 6 weeks. 3 fair or unsatisfactory.

(b) *No operation*—9 cases. 2 good—average disability period 31 weeks. 7 fair or unsatisfactory.

(2) *Marginal fracture of radial head with displacement*—5 cases.

Excision—5 cases. 5 good or excellent—disability period 6 weeks.

(3) *Marginal and fissure fractures without displacement*—16 cases.

No operation—16 cases. 12 good or excellent—disability period 6.5 weeks. 4 fair or unsatisfactory.

Sprains and Subluxations of the Ankle-joint

By Flying Officer J. ROWLAND HUGHES

SO-CALLED sprains of the ankle-joint are frequently complicated by tilting of the astragalus in the tibio-fibular mortice. Fifty-seven consecutive cases of ankle injury showing no bony damage have been studied with routine radiograms of both ankles in full inversion. Twenty-eight cases were simple sprains, with no tilting of the astragalus (49%). Twenty-nine cases showed degrees of tilting varying from 3 to 35 degrees (50%). Three cases showed diastasis of the inferior tibio-fibular joint. Those showing tilting of the astragalus were divided into three groups: Group I, 5 degrees tilt and less: 10 cases. Group II, 5-10 degrees tilt: 8 cases. Group III, over 10 degrees tilt: 8 cases.

The physical signs were variable, and the degree of local trauma was not necessarily related to the degree of tilting, but the tenderness in simple sprains and minor tilts was more often over the anterior expansion of the external lateral ligament, whereas with major tilting tenderness was below and behind the external malleolus, i.e. over the middle fasciculus. It must be stressed that in recent subluxations there is often considerable swelling and the sulcus below the external malleolus on inverting the foot, which characterizes recurrent subluxation, may be obscured.

The pathology in severe Group III types of injury is probably a complete rupture of the middle fasciculus of the external lateral ligament which is much the strongest of the three components (fig. 1 (a)). It is suggested that this occurs when the foot is forcibly inverted with the ankle at right angles. In this position the fibres of the middle fasciculus are almost vertical, whereas those of the anterior fasciculus and posterior fasciculus are horizontal. Forcible inversion with the foot in plantar flexion is more likely to tear the anterior fasciculus whose fibres are tending to become more vertical whilst those of the middle fasciculus are becoming more horizontal (fig. 1). It is probable that this is the type of lesion which occurs in most simple sprains and does not give rise to appreciable degrees of tilting. Rupture of the middle fasciculus is depicted diagrammatically in fig. 1 (b), which also illustrates how the lesion is only revealed on inversion of the ankle.

The importance of recognizing recent tears of the middle fasciculus is illustrated by the frequency with which recurrent subluxation occurs. This was present in almost 25% of the above cases and often required operative reconstruction of the ligament. The diagnosis is fraught with many pitfalls: (1) While the ankle is being radiographed, the

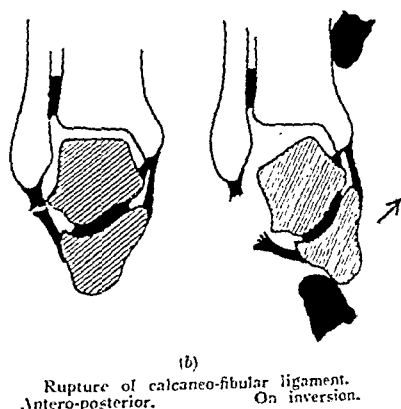
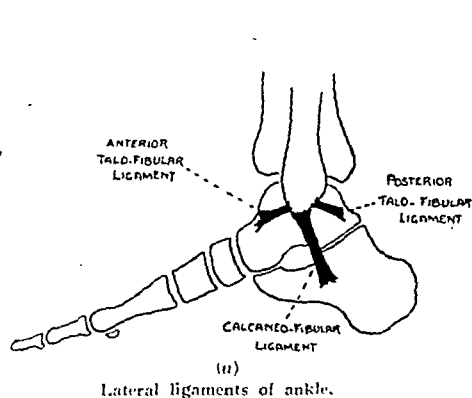


FIG. 1



FIG. 2



FIG. 3

FIGS. 2 and 3.—Subluxation of astragalus due to recent rupture of lateral ligament, with 19° tilt on inversion (fig. 3). Normal appearance of ankle in the antero-posterior view (fig. 2).

foot must be held in full inversion by a competent person holding the os calcis and the forefoot; it is not sufficient to hold the forefoot alone when X-raying (figs. 2 and 3). (2) If a definite tilt of the astragalus is noted (fig. 3), inquiry must be made into the history of previous trauma or symptoms—it may be a case of recurrent subluxation which will not benefit by eight weeks' immobilization in plaster. (3) The other ankle should always be X-rayed in full inversion for comparison. 25% of cases in the above series showed some degree of tilting in the contralateral ankle. (4) The injection locally of 10 c.c. of 2% novocain will often reveal a hitherto undisclosed tilt; pain having been relieved, peroneal spasm disappears. This emphasizes the necessity for X-raying in full inversion all ankles treated by local anæsthetic injection before walking is allowed.

Minor degrees of tilting of the astragalus demand no rigid immobilization; tilts of 3, 4 and 5 degrees are quite often seen in the contralateral ankle without symptoms or history of injury. Novocain injection or viscopaste for ten to fourteen days, or in cases with severe swelling, a walking cast for the same period, is sufficient. Group III and the severer Group II cases require rigid immobilization in a walking plaster for not less than eight to ten weeks. In cases of recurrent subluxation, operation for the reconstruction of the external lateral ligament of the ankle must have, as its primary object, the reconstruction of the middle fasciculus.

Section of Proctology

President—A. LAWRENCE ABEL, M.S.

[June 10, 1942]

DISCUSSION ON THE TREATMENT OF INOPERABLE CARCINOMA OF THE RECTUM

Mr. W. B. Gabriel: In defining what we mean by "inoperable" carcinoma of the rectum, several different groups of cases would appear to be involved: in some the fact of inoperability is obvious either clinically or on abdominal exploration, and in others the estimate of inoperability is a much more individual and personal matter in which the surgeon is chiefly concerned. The surgeon in charge of a borderline case will naturally be influenced by his experience and surgical facilities, and if a radical operation is contemplated his decision may in some cases be considerably affected by pathological and medical findings. Four groups of cases can be distinguished.

(1) *Locally operable and suitable for radical operation* which, however, cannot be proceeded with by reason of some general condition such as senility, mental instability, accompanying disease such as active pulmonary tuberculosis, severe degrees of renal or cardiac failure, or severe diabetes with complications. Refusal by the patient to have a colostomy or even a cutting operation of any sort is sometimes the reason for relegating an early carcinoma to the inoperable group. In these cases with a small malignant growth the natural expectation of life is fairly lengthy, and it is in these cases particularly that the choice of the best line of treatment is so important.

(2) *Locally operable but with metastases in the liver.*—Early venous spread to the liver is usually only discovered on abdominal exploration and, since the growth is probably non-obstructive and the expectation of life short, expectant treatment without colostomy is indicated.

The question of a "palliative" radical operation comes under this heading. Patients should not be subjected to the dangers and pain of a radical operation when there is no hope of cure by surgery. If there is any doubt as to the nature of a small nodule in the liver one must endeavour to inspect it by extending the paramedian incision upwards to see if the nodule is a white secondary deposit or a bluish-coloured cyst. If, however, it proves impossible to view it, then a radical excision should be made. On several occasions I have felt a large gall-stone in a shrunken gall-bladder which required some care to distinguish it from a secondary deposit.

(3) *Locally borderline or inoperable without clinical evidence of metastases.*—It is obviously more easy to form a clinical estimate of a low carcinoma than a high one. In the female a low, deeply ulcerated carcinoma extending into or through the posterior vaginal wall may with reason be assessed as inoperable but yet be properly treated by a diathermy perineal excision, and the same type of operation is often the best thing for a late carcinoma involving the anal canal or fungating outside the anus. In the male, a deep malignant ulcer which has extended through the rectal wall anteriorly and can be felt to be firmly attached to the prostate or urethra is hopeless and requires a palliative colostomy. In the *middle third* of the rectum fixity posteriorly may only indicate that the growth has extended through by direct continuity to the ensheathing pelvic fascia, and may still be removable by a combined excision. Palpable extrarectal induration laterally and anteriorly is of more serious import, and if nodules of growth can be felt high up in the posterior fornix the growth is certainly inoperable. With regard to the *upper third* of the rectum we who have experience in rectal carcinoma must refuse to label advanced or borderline cases as inoperable until they have been proved to be so by laparotomy. We must not be deterred by clinical "bad risk" features such as:

- | | |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------|
| (1) Advanced age—no arbitrary upper age limit should be recognized for radical operation, and I can record a successful perineo-abdominal excision in a man of 84. | |
| (2) High or low blood-pressure—this calls for close co-operation with the anaesthetist and care in the dosage of spinal anaesthesia. | |
| (3) Mild degrees of urinary failure | } These can be much improved by careful preparation. |
| (4) Secondary anaemia | |
| (5) Some degree of intestinal obstruction | |
| (6) Smooth enlargement of the liver—this is not necessarily due to metastases. | |
| (7) Apparent fixity of the growth. | |

other. Thus in radiotherapy, the radiosensitivity of the tumour bed—as these normal tissues may be called—is just as important as that of the cancer cells; for if the tumour bed is also badly damaged by irradiation, the surviving cancer cells have time to recover before they can be dealt with, and the tumour resumes its growth. In their turn, the health of the normal tissues which constitute the tumour bed depends upon the general condition of the body as a whole. Thus, of the three factors concerned in the curability of carcinoma of the rectum, the radiosensitivity of the malignant cells is the least important; next comes the state of the tumour bed, whether relatively healthy or already damaged by sepsis or surgical interference; and most important of the three, the general vitality of the patient.

The first essential in the radiotherapy of inoperable carcinoma of the rectum therefore is to select those cases in which there is a prospect of cure. The advanced cancer cannot be cured and radiation is given only for certain limited objectives—haemorrhage can be stopped, discharge can be lessened, and pain, in so far as it is rectal in origin, can be relieved. Pain due to the involvement of the spinal nerves is better treated by other methods.

This incurability of advanced cancer has been for me best illustrated by recurrent cases; I think surgeons recognize that an advanced case is often best left alone, but for one recurring after operation they always hope that the situation may yet be retrieved. But in most cases recurrence after operation is widespread, and there is often sepsis and a perineal sinus as well. Of the 22 recurrent cases which have been treated by the million-volt X-ray apparatus, 18 have been of this widespread nature, and for them radiotherapy has really nothing to offer, although occasionally worthwhile improvement has been obtained.

TABLE I.—CARCINOMA OF RECTUM, 65 CASES.

| | |
|-------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|
| A. Recurrent after Radical Operation—22. Apparently cured—1. Marked improvement—5. | C. Inoperable primary growth without clinical metastases—32. Marked improvement—14. Growth-free for more than three years—3. |
| B. Inoperable primary growth with liver metastases also—4. Arrest of primary growth—2. | D. Operable primary growth, but operation inadvisable—7. Growth-free following radiation—6. (None of these 7 cases has a colostomy) |

In 32 cases the local extent of the growth made them inoperable, and they were all radically irradiated, that is, cure was aimed at. Cure was attempted because there was no clinical evidence of metastases outside the pelvis, but not a few of these cases I should now recognize as nevertheless too advanced for curative therapy; thus 12 of them died in less than one year after treatment. Leaving aside the question of palliation—always a rather subjective assessment—disappearance of the inoperable carcinoma was obtained in 11 of these 32 cases following X-rays alone, and in a further 3 cases after additional measures: intrarectal radium in 1 case, surgical diathermy in the second, and subsequent perineal excision in the third.

Whether the disappearance is permanent—that is, whether cure will be obtained—it is too early to estimate. 5 of the 14 cases are already dead, but in none was there recurrence in the rectum, and only 1 of the 5 died from metastases. The longest-standing of the 9 still living has now passed her fourth anniversary since treatment, and has had her colostomy closed for the past eighteen months: there are 2 more who have passed their third anniversary, and in one of these there were metastases in the lymph glands as high as the origin of the inferior mesenteric artery when the colostomy was made. Even if these 3 are the only permanent cures out of the 32—and I think their number will certainly be added to—that is still a salvage of 1:10 of otherwise doomed cases.

Finally there are 7 cases in which the rectal growth was operable, but the general condition of the patient precluded surgery. None of these 7 had a colostomy, and in 6 of them the carcinoma disappeared after X-ray treatment. One of these 6 had coronary disease, from which he died twenty-one months after treatment of the carcinoma of the rectum; it was the earliest growth I have yet had to treat by X-rays, being confined to one quadrant of the rectum. The other 5 are all alive and free from growth, and free from a colostomy: one aged 86 is well after two years, 3 aged 81, 77, and 72 respectively for approaching two years, and the fifth, aged 54, was treated only seven months ago.

Of 65 surgically rejected carcinomata of the rectum, 19 disappeared after treatment by million-volt X-rays, or roughly a third of the total. Nothing approaching this has been seen after treatment by 200 kv. X-rays. Several factors probably contribute to the superiority of the million-volt. The gain in depth-dosage, for example, is of the order of 40% for small fields. The isodose curve for a 10-field arrangement at a million-volts shows that for 100 r on each field, the tumour receives 370 r; but at 200 kv., only 250 r (fig. 2). The distribution of the dose is also better with the million-volt, being uniformly high

where it is required in the malignant region, but falling rapidly among the normal tissues beyond the limits of the growth's extension. It is possible that the quantum energy, which increases as the wave-length shortens, that is, as the voltage rises, may also be a factor of some importance. To give a tumour dose of 6,000 r—and something approaching

TABLE II.—CARCINOMA OF RECTUM, IRRADIATED THROUGH
10 SKIN FIELDS, EACH 18 × 8 cm.

| I. A surface dose of 100 r per field gives— | | | II. To give a tumour dose of 6,000 r— | | |
|---------------------------------------------|-------------------|--------------|---------------------------------------|-----------------------------|--------------|
| At 200 KV. | | At 1,000 KV. | At 200 KV. | | At 1,000 KV. |
| 250 r | Tumour dose | 370 r | 2,400 r | Dose per field | 1,620 r |
| 210 r | Maximum skin dose | 210 r | 5,030 r | Maximum skin dose | 3,400 r |
| | | | 67 | Volume dose in megagramme-r | 41 |

(Half-value-layer at 200 kv. = 2.0 mm. Cu)

(Half-value-layer at 1,000 kv. = 9.3 mm. Cu)

this seems to be necessary for the cure of most carcinomata of the rectum—each field must have 1,620 r at 1,000 kv., and the maximum skin dose when the through or exit radiation is added is 3,400 r; but at 200 kv. the field dose has to be 2,400 r, and the maximum skin dose is now 5,030 r, which is at the limit of tolerance and in many cases would result in radionecrosis later. Of still greater importance, perhaps, is the question of volume dose; at 1,000 kv. the volume dose is 41 megagramme-r, a dose which definitely impairs the patient's vitality, but from which recovery occurs in six to eight weeks. At 200 kv. the volume dose is 67 megagramme-r, which is higher than has yet been given in any condition, and which would almost certainly impair the patient's vitality to a critical degree.

I suppose there is nothing more humbling than the daily grappling with the incurable, yet on such radiotherapy has to prove its value, for the surgical results in carcinoma of the rectum are good. If surgical judgment were as perfect as surgical technique, they would be even better, for then there would be fewer recurrences in the operation area. Radiotherapeutic judgment is trained from the other end, as it were, on the advanced and recurrent cases, while radiotherapeutic technique is still developing. Here is the starting point for future advance. In the treatment of carcinoma of the rectum, as of cancer in general, the best interests of the patients can only be served by constant collaboration between surgeon and radiotherapist; with that collaboration we may soon have the early and operable case cured without the deformity of a colostomy.

Mr. A. Dickson Wright: Intractable pain in the later stages of inoperable carcinoma of the rectum, whether from recurrence after removal or from an inoperable primary growth, often appears in patients who have still a considerable time to live and it is really amazing how well these patients look and how much useful work they are still capable of doing before a lethal complication appears. Once the pain has got a grip the patient is quickly useless as a member of the community and a burden and sorrow to those around him, whether from drugs or the lack of sleep undermining his health. It has been very wisely said that surgery directed to the relief of pain is responsible for some of the greatest surgical mistakes because judgment is inclined to weaken under the pressure of the patient's clamour for escape from his agony. It is well, therefore, to exercise the greatest care in the selection of cases for pain-relieving operations. The pain of rectal carcinoma is not a visceral pain as a rule but due to extension to the more sensitive organs in the vicinity such as the anal canal, bladder, prostate and sacral plexus and bones of the pelvis. The pain is generally felt in the latter case in the sacral and perineal regions, in the groins and down the back of the thighs and is of such an unendurable quality that even the strongest characters soon break down under it.

Several procedures have been proposed for the relief of the pain apart from palliative colostomy. (a) The division of the pre-sacral nerve. The opportunity to do this is generally taken at the time of the palliative colostomy, it adds nothing to the risks of the operation and forestalls visceral pain from the rectum and possibly pain which may develop later from vesical and prostatic extensions of the growth. It is no help to somatic pain which may later develop.

(b) The Dogliotto procedure. In my hands this treatment has not achieved the success which on theoretical grounds it should provide. Lumbar puncture is made about the 2nd or 3rd lumbar space with an unbreakable nickel needle and then after the needle is satisfactorily in position the patient is postured by regulating the table so that the posterior nerve roots transmitting the pain lie above the point of the needle. One cubic centimetre or less of alcohol is then introduced slowly to avoid currents and if the injection is satisfactory then a warm sensation is felt in the region where the pain is experienced and by pin-testing this area is found to be anæsthetic. The patient remains in the same position for half an hour to allow the alcohol to be absorbed and is then kept in bed for twenty-four hours. The procedure can be repeated after a few days to stop the pain on the other side. Failure to achieve relief of pain may lead to reckless injections with resultant loss of sphincter control.

(c) Chordotomy is the most satisfactory of all the procedures that I have tried. It is reserved for the younger subjects with good reserves of strength. Laminectomy is done at the summit of the dorsal convexity. The space provided by the removal of the laminae of D. 5 and 6 gives sufficient access to the cord. After exposing the cord two light stay sutures are passed through the ligamentum denticularum after division of two of the denticulations and by traction on these the cord is rotated so as to display its antero-lateral region. The tough pia is sectioned from the denticulate ligament as far round as the anterior roots, i.e. about 45 degrees of the circumference. I prefer to snip the tough pia with fine scissors like de Wecker's iris scissors and then divide the substantia of the cord to a depth of 0.5 cm. with a marked Graefe's knife. The section is repeated an inch higher on the opposite side. After operation, retention of urine develops for a short time and so does some pyramidal weakness, but these disappear very quickly. The relief from pain is instantaneous and if the operation is done under local anaesthesia can be checked at the time before the dura is closed. Following the operation all pain-killing drugs can be dropped and often the patient becomes fit for some kind of work.

Dr. Ffrangeon Roberts: A significant feature of this discussion is that so far no mention has been made of radium, an omission which reflects the general experience that in this part of the body as in so many others radium has proved a failure. X-ray treatment therefore remains the only means left to us of exerting any direct influence upon the growth. In such a slowly growing tumour as rectal carcinoma, where the disability is mainly one of discomfort and inconvenience it should be our aim to enable the patient to live as normal a life as possible and to preserve his working capacity.

The routine performance of colostomy, even when obstruction does not threaten, has long been the established practice on the quite unsubstantiated ground that the rate of growth is thereby slowed up. The resulting disadvantages to the patient hardly need stressing.

The question therefore is, whether X-ray treatment can improve the condition to the extent of making colostomy unnecessary. My results show that it can, provided that the tumour is subjected to a sufficiently large dose, an end which is achieved by the method of multiple small fields which I have advocated for some years past.

Out of 35 cases, of whom 5 could not be followed up, one is still in full working capacity after over six years, his bowels being opened regularly twice a day. Two others are in a similar state after more than five years. A fourth has recently died, aged 72, after completing five years, despite the fact that he had had symptoms for a year before treatment was begun. Taking into account that 10 of the 35 were in an advanced state when first seen these results speak for themselves.

Mr. E. T. C. Milligan: The surgeon measures the extent and the severity of his operative measures against the extent of the disease and the patient's strength. One trained in the operation of perineal excision classifies patients as inoperable when he finds the disease beyond the scope of his operation. The surgeon trained only in the more severe abdomino-perineal operation will consign certain frail and elderly people to inoperability where the less shock-giving operation of perineal excision would be successful. Frail patients who would survive as operation with a 7% mortality, which the perineal resection carries, would succumb to the one with an 18.25% mortality.

To-day there is happily another procedure which will not only reduce the number of inoperable cases in the hands of skilled operators, but will decrease the numbers labelled inoperable: I refer to the operation of simultaneous combined excision of the rectum. Because of the division of the work by two surgeons and consequently the shorter time expended in the operation, the area of the operation can be extended, time-consuming complications overcome, and frailer patients submitted to the procedure. More surgeons throughout the country are now acquiring the skill and practice required by this simpler and shorter procedure. Surgeons at a younger age master its technique without facing the inevitable high initial and occasional mortality. They learn separately the perineal and abdominal parts. More patients will now be submitted to and more will survive the operation. In the beginning the abdomino-perineal operation had a mortality of 40%. It is now 18%.

Palliative removal of the rectum.—Perhaps the worst symptom the patient with carcinoma of the rectum has to face is tenesmus, i.e. continual rectal discomfort or pain, and repeated desire to pass mucus. To relieve or forestall this torment it is good practice to remove the rectum. I believe that great risks should be taken to give this relief even with frail patients. It can be done advantageously where malignant extension in the gland chain or in the liver is beyond the scope of radical removal. Removal of the rectum might well be considered also where the extent of local spread prevents complete local removal of disease. It may be less disturbing to a doomed patient to suffer local

recurrence than to have rectal tenesmus. Removal of the rectum means cutting off the nerve supply as well as stopping the persistent and distressing defaecation desire. Palliative removal is also merciful when the sensitive skin of the lower third of the anal canal and the skin of the anus are involved.

The effect of operability on the patient's mind and life.—Operation means a way of freedom and escape from the fundamental fears of man connected with his security, comfort, vocation and existence, centred round his growth. If we remove the growth we remove these fears although the mind takes longer to heal than the body.

Inoperability.—If a patient knows he has a growth and that it is inoperable, who knows what groundless fears haunt his imagination unless he is willing and has the courage to discuss them? Confidence and trust in the surgeon will help him to bring out unexpressed fears.

Dr. Phillip Flood: In considering a tumour for X-ray treatment there are two factors which influence the dose and plan of treatment: the histology of the tumour and its accessibility. On both these counts carcinoma of the rectum is unfavourable. Histologically, the high degree of differentiation of the tumour cells indicates a relatively low radiosensitivity and, therefore, the necessity of a high total dose to the tumour. This aggravates its second disadvantage, namely its inaccessibility. It is in overcoming the second factor and so rendering a high dose possible, that most progress has been made in recent years.

This progress has been made principally in three ways, *first* higher kilovoltages. Voltages of from 400,000 to a million are now practical and in clinical use, and have considerably increased the dose that can be given to the tumour. *Secondly*, and more important, is greater accuracy in beam direction. A dose of from 6,000-7,000 r in approximately six weeks will be required; if this is to be achieved it must be limited to as small an area as possible; by the use of small posterior fields directed obliquely the area receiving this high dosage can be limited to the site of the tumour, provided the fields are set and directed with accuracy. *The third* direction in which progress has been made is in greater accuracy of dosage measurement which enables a higher dose to be given with safety.

There is another method by which a localized fixed tumour of the rectum may be treated; this is by surgical exposure to render it accessible to irradiation by a tube of short focal skin distance. This method has so far hardly been attempted in this country in rectal cases, although it has been used in the treatment of other deep-seated tumours such as those of the bladder and larynx. Its advantage is that a very big dose can be given and restricted entirely to the tumour. Professor Chaoul published a short series of rectal cases treated in this way before the war, and though his cases would be considered operable cases, his method might be applied to some rectal tumours, especially those considered inoperable on account of their fixation.

Carcinoma of the rectum which is inoperable on account of extensive pelvic metastases, or metastases outside the pelvis, can only be treated palliatively; the principal indication for X-ray treatment in such cases is to relieve pain due to the pressure of tumour masses. Only the minimum dose required to relieve pain should be given, and if relief is not then obtained treatment should not be continued. Again only a small dose is required to arrest or diminish hæmorrhage and discharge. The radiologist should not treat hopeless cases simply as a placebo.

Mr. O. V. Lloyd-Davies: In considering inoperable carcinomata of the rectum every effort should be made to reduce the number of cases at present regarded as inoperable. Mr. Wilfrid Adams has stated that two-thirds of the cases seen at Bristol are inoperable whilst at St. Mark's Hospital approximately 70% are considered suitable for excision.

The solution to the problem lies in the popularization and adoption of the simultaneous or synchronous combined excision. By this method the difficulties are shared by two operators with the result that surgeons of average experience are enabled to perform Combined Excision with a greater assurance of success. In addition surgeons of specialized experience are able to operate on many cases which would be regarded as inoperable by the most expert surgeons working alone.

We have now at St. Mark's a growing number of cases with firmly fixed tumours which have benefited by operation. Moreover it is particularly satisfactory to be able to record that the factor of fixity in the majority of these cases is perirectal inflammation, so that the prognosis is often good.

The growth of team work in all large surgical centres should enable the synchronous combined method to be more extensively used and many more patients will benefit from a radical excision instead of being declared inoperable.

Section of Neurology

President—GEORGE RIDDOCH, M.D.

[May 21, 1942]

DISCUSSION ON TRAUMATIC EPILEPSY

Major Hugh G. Garland: The term "head injury" covers a multitude of different pathological conditions which have trauma as a common denominator but which have little else in common, especially as regards their epileptogenic properties. It is time that some of these problems were settled, for besides their academic importance there is the return of war injuries, the increase in the number of road accidents (which has been followed by a multitude of medico-legal problems), and the new therapeutic approach to this type of epilepsy offered by modern neuro-surgery. A good deal of what is written about traumatic epilepsy is only the result of clinical impression and of speculation. Review of the literature on any aspect of epilepsy is no light task as it seems there have been at least 2,000 papers written in the last twenty years alone.

One fact is clear, namely, certain types of injury are much more likely to be followed by epilepsy than others. Outstanding amongst these is the gun-shot wound (G.S.W.). One of the best recent reviews of epilepsy following a G.S.W. is that of Ascroft (1941), a paper based on a follow-up of 317 cases from the records of the Ministry of Pensions; all these were casualties from the last war. In this group no less than 34 per cent. have developed epilepsy, a finding which is surprising in view of the previously quoted incidence of 4.5 per cent. in 18,000 cases of G.S.W. This latter figure is frequently found in the literature from 1921 onwards (Sargent, 1921; Stevenson, 1931), and is also obtained from Ministry of Pensions records. Ascroft's group was selected by him only to the extent that the notes were complete, and all his cases were presumably included in the larger series. The discrepancy is not due to the late onset of fits, as Ascroft found that most of the cases developed epilepsy within the first few months. Dr. Prideaux (1942) thinks it is partly due to the fact that the less severe cases had their claims settled early (7,600 by 1923) and presumably these cases were at that time suffering neither from epilepsy nor any other serious symptom. If this is the case Ascroft's group is selected to the extent that it consists largely of the more severe injuries; it is, however, almost certainly a maximal figure. Ascroft's series includes a variety of different pathological conditions and he has analysed his results accordingly. The effects of a G.S.W. are frequently localized and it is often the case that a severe local lesion of this type is not associated with loss of consciousness, though there is sometimes such loss after an interval (Eden and Turner, 1941); there may be a scalp wound only, or underlying this there may be severe cerebral contusion with or without fracture or penetration of the dura, or there may be a compound depressed fracture with laceration, in-driven bone, metallic or other foreign bodies, as well as sepsis; the end-result of such injuries also includes such differing conditions as scars, cysts, arachnoid, abscess, &c. Ascroft's figures show that epilepsy occurred in 24 per cent. of those with scalp wounds only, but clearly there must have been brain damage in these cases which was unsuspected at the time. The incidence of fits was twice as high when the dura was penetrated. The presence of foreign bodies did not appear to increase the development of fits (unlike Wagstaffe's findings, 1928), but epilepsy was twice as common after there had been sepsis, whether the dura was opened or not. This high incidence of epilepsy after gross localized lesions is perhaps not unexpected when one thinks of the incidence of epilepsy in other gross and localized brain lesions of non-traumatic pathology. According to Penfield (1939a) fits occur in 44 per cent. of all supratentorial tumours. I think the incidence of epilepsy after recovery from a brain abscess is not less than 50 per cent., just as epilepsy is a common symptom in the acute stages of brain abscess, in fact a frequent presenting symptom in metastatic abscess. We can say at this stage that epilepsy following a G.S.W. is now well known. It is associated with a gross pathology, has opened up a promising surgical therapeutic field, and forms that subdivision of traumatic epilepsy about which there is a maximum of fact and a minimum of speculation.

Epilepsy following other varieties of head injury is much less clearly understood. These include injuries known as "closed" or "blunt" injuries and are those commonly seen

in civilians; they usually result from road accidents, from falling on the head and from blows from falling objects, usually of low velocity. This is again a mixed pathological group but certainly in cases without penetration of the dura there tend to be more diffuse and smaller brain lesions, gross focal lesions being very much less common. At the same time it is probable that a number of focal lesions are overlooked in this group owing to inadequate clinical examination in the acute stages. It is largely on this type of civil injury that most of the figures relating to traumatic epilepsy are based, and, especially in the older literature, there is little attempt to subdivide the cases according to severity or type of injury. That such injuries may be followed by epilepsy is accepted but to what extent the epilepsy is the result of the injury is less certain. The majority of these cases die a long time after the injury and after epilepsy has been present for years: post-mortem examination of the brain is rarely made and is usually inconclusive and, unlike the case of G.S.W., observation of the brain at operation is rare. The problems, therefore, have to be approached in other ways, the most important of which appears to be statistical analysis. Many series of cases have been followed up over long periods, but most of these are unsatisfactory, and it will not be until well-documented records of a large series of cases, such as that of Russell (1932), have been followed over years that accurate figures will be available. One of the most reliable of recent figures is that of Rowbotham (1942), who found epilepsy in 2.5 per cent. of 450 cases of injury of the "blunt" type. He regards this as probably the upper limit, the lower being Feinberg's (1934), who found epilepsy in 0.1 per cent. of a remarkable collection of 47,130 unselected cases of head injury. This latter figure is all the more striking in view of the fact that epilepsy is said to exist in 0.5 per cent. of the population of any country: if this be true Feinberg's figure can only show that a head injury confers a considerable degree of immunity to epilepsy. Russell (1934) found epilepsy in 3.5 per cent. of 200 cases within the first eighteen months of the injury and as Russell's and Rowbotham's cases were obtained from very similar sources this figure of about 3 per cent. is probably somewhere near the truth; but Kinnier Wilson (1940) quotes figures varying from 3.6 per cent. to 21 per cent., while Symonds (1935, 1941) regards epilepsy as being rare after this type of injury. These varying figures can only mean that the cases are in some way selected and that different authors are not discussing the same problem. I think it will be generally accepted that a single cerebral concussion is at least unlikely to be the sole cause of epilepsy, or even to be an important factor in its production, and such cases have no doubt diluted the figures of some observers. In his small series of epileptics Rowbotham found a significant preponderance of severe injuries. Here again, however, there is little agreement as to what constitutes a mild or a severe head injury and for this reason I do not think that further analysis of older published records will be of any value. Suffice it to say that the highest incidence claimed is about 20 per cent. and the lowest a good deal less than that of epilepsy in the general population; and that it is at least very probable that the first figure relates to a selected group of severe injuries and the second is diluted with many trivial cases.

Another method of statistical approach to the traumatic factor in epilepsy is to record the incidence of previous head injury in an unselected group of epileptics. Here again there are difficulties as it is usually impossible to confirm the history and, still more, the details of the accident. Textbooks usually dismiss this subject by saying that a history of injury occurs in less than 5 per cent. of all epileptics. I have analysed a group of 2,600 Service patients; these are selected in that they are all males (in whom a history of injury will be higher than in a mixed group) between the ages of 18 and 50, the majority between 20 and 35; they were all referred to me as falling in the "neuro-psychiatric" group. There were 244 cases of epilepsy, of which 77 per cent. appeared to be idiopathic (Table I). Of the remainder there was a history of head injury prior

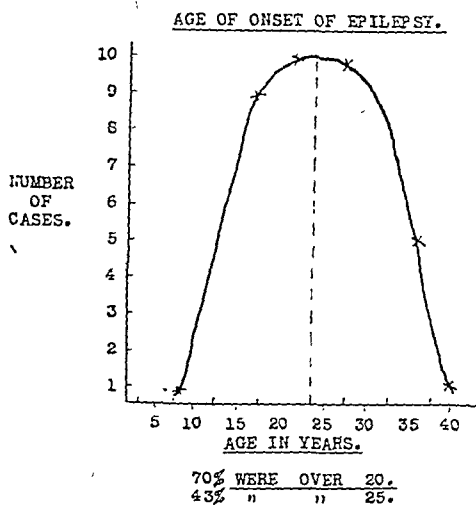
TABLE I.—CAUSATION OF EPILEPSY.

| | Number of cases | Percentage |
|--------------------------------|-----------------|------------|
| Idiopathic epilepsy | 190 | 77 |
| Mixed symptomatic group | 15 | 7 |
| History of trauma | 39 | 16 |
| Total | 244 | 100 |

to the onset of fits in 16 per cent., the remaining 7 per cent. being cases of cerebral tumour, syphilis, or cysticercosis. This is a surprisingly high figure, which would have been lower had not leading questions been asked about previous trauma: unfortunately I have no suitable control group. It does not, of course, follow that the injury is in any way related to the epilepsy, but certain facts suggest that in many cases it was of importance. It is generally accepted that idiopathic epilepsy starts in childhood or adolescence and, while there may be some disagreement as to the upper age limit for

the onset of idiopathic fits, many put it at 20, and probably most at 25. In my group of cases 70 per cent. had their first fit after the age of 20 and 43 per cent. after the age of 25 (Table II). These figures suggest very strongly that most of the cases were not

TABLE II.



of the idiopathic type, and there was no evidence of any pathological condition other than the previous trauma. In idiopathic epilepsy there is a family history of fits in about one case in five; in 34 of my cases where the information appeared to be reliable there was a family history in only 4, but these figures are too small to be significant. Assuming that the injury has been a factor in these cases it is interesting to classify the injuries according to severity. Of the 39 cases one man had been a boxer and had had multiple slight injuries; I have subdivided the remaining 38 into 3 groups, choosing arbitrary limits: a post-traumatic amnesia (P.T.A.) of less than half an hour is called a "slight" injury, a P.T.A. of half to three hours "moderate" and more than three hours "severe" (Table III). It will be seen that there is a strikingly high incidence of

TABLE III.—SEVERITY OF HEAD INJURY.

| Severity | Number of cases |
|---------------------------------|-----------------|
| Slight | |
| P.T.A. under $\frac{1}{2}$ hour | 2 |
| Moderate | |
| P.T.A. under 3 hours | 8 |
| Severe | |
| P.T.A. more than 3 hours | 28 |

severe injuries, which is again at least suggestive that the injury is an aetiological factor. It is generally accepted, and in the case of G.S.W. is certainly proved, that the interval between the injury and the onset of fits may be anything up to a number of years, but here again there is considerable disagreement. Foerster and Penfield (1930) in a short series of cases found an average latent period of five and a half years, with limits of five months and fourteen years. On the other hand Ascroft found the latent interval to be much shorter; he found the commonest onset was within the first month, though there were extremes of a few hours to twenty years, and Russell (1942) thinks the late development of epilepsy in closed injuries is rare, i.e., after two or three years. Table IV

TABLE IV.—LATENT PERIOD.

| Years | Cases |
|---------|-------|
| Under 1 | 24 |
| 1-3 | 4 |
| 4-10 | 4 |
| Over 10 | 6 |

shows the interval in my group of cases: this varied from a few weeks to eighteen years, but the large majority started within the first year. With regard to the longer latent

intervals, it is interesting that Penfield (1939b) has found evidence that cerebral scars may sometimes increase in size over a number of years. I might add that an "average" figure representing the latent period, when taken from a group varying from a few weeks to many years, is of no significance whatever. In reviewing these cases from the point of view of whether the injury was a causal factor or not one can, therefore, say that in the majority of cases the age of onset of epilepsy was later than is usually seen in the idiopathic form, that the severity of the injury was in most cases considerable and that the latent period was usually short; a family history was uncommon and there was in no case evidence of any other further causal factor. It is interesting that trauma in this group of cases was much commoner than all other causes of symptomatic epilepsy, though this, no doubt, was largely the result of the age group under consideration.

Taking traumatic epilepsy as a whole, irrespective of the type of injury, it is interesting to study the clinical varieties of fits. The common idea that head trauma tends to be followed by Jacksonian fits is very wide of the mark and nearly all authors are agreed that much the commonest attack is a generalized convulsion. At the same time there is frequently an aura indicating the focus of onset, as in any other type of epilepsy. It has often been observed that other variants are uncommon, for example, Kinnier Wilson (1940) says: "It is curious how seldom *petit mal* or any epileptic variant is either ascribed to injury or evoked by it." Major fits occurred in all my cases and there was no example of Jacksonian attacks; psychomotor attacks co-existed with major fits in 2 cases and minor with major attacks in 3. The electro-encephalogram (E.E.G.) may ultimately solve this problem. For example I am not sure whether the characteristic E.E.G. of either *petit mal* or psychomotor epilepsy is ever seen after a head injury. If not it may be the case that trauma does not produce fits of these types and that in cases where they occur the trauma may not have been a causal factor, or may perhaps have precipitated fits in a potential idiopathic epileptic; but here again there is disagreement as to the frequency of variants and Symonds (1935) believes the minor attack to be a frequent early manifestation of traumatic epilepsy. This aspect of the problem needs a good deal of further investigation in the light of recent advances, as it may be the case that attacks previously called "minor," which presumably means *petit mal*, may be syncopal, or short attacks of vertigo. Stevenson (1931) actually states that "vertigo" is common in the latent period and Rawling (1922) found that "fainting" occurred in 16 per cent. of cases following G.S.W. The great importance of these diagnostic problems lies in the prognosis in cases where only attacks of a minor nature are occurring, but whatever the final verdict I think there can be no question that by far the commonest attack is a generalized convulsion. This again should not be unexpected as both *petit mal* and psychomotor attacks must be very uncommon in any symptomatic epilepsy. In this respect it is of some significance that such variants have never, I think, been produced by any form of electrical cortical stimulation nor in my experience do they occur after a water-pirressin test.

I have no personal records relating to multiple head injuries. There is no doubt that headache and other common post-traumatic symptoms tend to be more marked and to carry a worse prognosis if there have been previous injuries, and in this respect my one example of epilepsy in a professional pugilist is of interest. The "punch drunk" syndrome seems quite clearly to result from multiple minor injuries and in my case it seemed probable that the epilepsy was so caused.

To assess the influence of trauma in cases of epilepsy following head injury in civilians is to solve the problem whether such are not in fact cases of idiopathic epilepsy, or whether the trauma may have acted only as a precipitating factor. Because of the undoubted lower incidence of epilepsy in these cases, as opposed to G.S.W., it seems clear that pathological processes left behind by such an injury are not so epileptogenic as are the grosser lesions. The development of epilepsy may be entirely a question of the quantity of cerebral tissue damaged, the number of individual lesions, their size, or their position: or it may depend on some other unknown property of the lesion itself. The lesions produced by cerebral cysticercosis are so epileptogenic that there is no recorded case of them existing for very long in the absence of epilepsy (MacArthur, 1942); on the other hand such a gross lesion as disseminated sclerosis has little tendency to produce fits. It has been said that for a head injury to be followed by epilepsy there must be a certain type of brain and that unless the brain is of this type no amount of trauma will result in fits. This may or may not be true but a similar statement would apparently not be true in the case of cysticercosis. Similarly, such differing stimuli as electrical discharge across the frontal lobes, intravenous cardiazol and hypoglycemia can produce convulsions in any type of brain. On the other hand Lennox, Gibbs and Gibbs (1939) state that there are three times more epileptics in the near relatives of all cases of symptomatic epilepsy than there are in those of non-epileptics (though I doubt whether

their cases include cysticercosis); this suggests an inherited tendency to epilepsy in all the symptomatic epilepsies. To sum up, therefore, it appears that in the civilian type of head injury epilepsy follows in about 3 per cent. of all cases, but this figure probably excludes simple concussion, and, the highest incidence follows the most severe injuries. The fits are almost invariably major in type and tend to come on early, the majority starting within the first year. Whether these cases are due solely to the injury or whether there was a previous tendency to epilepsy, of the type of inherited background that occurs in idiopathic epilepsy, is not yet clear. It must always be remembered that both head injuries and epilepsy are common and that their co-existence will sometimes occur by chance. Further, recent work with the E.E.G. suggests an epileptic tendency in 10 per cent. of the population, which might well account for all the cases of epilepsy following civilian head injuries. At the same time in medico-legal work trauma is as important as a precipitant as if it were the only causal factor.

As traumatic epilepsy so commonly appears within a year of a severe injury it is only to be expected that in the majority of cases there will be such bridging symptoms as headache, dizziness and some form of mental incapacity. I have not sufficient information relating to such symptoms in cases where the epilepsy only supervened after years—but I doubt whether their presence or absence is of much diagnostic or prognostic importance; Symonds (1941) finds epilepsy to be more common in those with persistent intellectual impairment or personality disorder, a state of affairs by no means limited to the most severe injuries.

Other important aspects of traumatic epilepsy include the significance of birth injury, which certainly accounts for some epilepsies of early life; the significance *qua* epilepsy of the site of the lesion; the prognosis, which is not always gloomy but which becomes worse the longer the latent period; and the co-existence of other symptoms, such as dementia, both in relation to diagnosis and prognosis.

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Mr. W. Grey Walter: The clinical statistics of traumatic epilepsy are contradictory and confusing, perhaps because the statistical method is not entirely suitable for studying this problem. It may be more profitable to consider in greater detail the physiological phenomena observed in each case. From the study of electro-physiological data, several facts are available. The first is that the electroencephalogram has shown that the convulsive seizure is only a part of the epileptic picture: persistent electrical abnormalities exist in many epileptics both "essential" and traumatic. The second fact is that electrical abnormalities seem to be much commoner in young patients, being almost invariable in children who have suffered birth injury. Thirdly, the electroencephalogram changes during the convulsion are always the same, irrespective of the cause of the fit. Records of the *petit mal* type are very rare in the traumatic cases, and it seems most probable that the true *petit mal* attack is never directly or entirely attributable to injury.

Apart from the short term, and temporary effects of head injury, there is no evidence of any specific effect of trauma in combination with convulsions on the electro-encephalogram, and there seems little hope, therefore, of distinguishing between the latent epileptic and the person in whom the injury was the direct cause of the condition—if such exists.

Further evidence is available from the electrical convulsion therapy, in the course of which it is possible to measure quite accurately with alternating current the convulsion threshold of thousands of patients. This threshold varies very widely from patient to patient, but is fairly constant in each individual. The variation is of the order of 1,000 per cent. Moreover, the threshold can be greatly raised by administering the con-

ventional anti-convulsant drugs. These facts suggest that convulsion thresholds vary enormously in each individual, and there is some reason for supposing that this is another aspect of E.E.G. data, leading to the idea of specific epileptic liability.

Two cases may illustrate this view. One is a woman who complained of attacks of twitching in the left arm and face which had developed after a comparatively trivial blow on the right side of the head. This persisted despite surgical removal of a part of the cortical area identified through the course of the abnormal electrical discharge which could be observed, during the attack, by the E.E.G. (fig. 1). The histopathological report

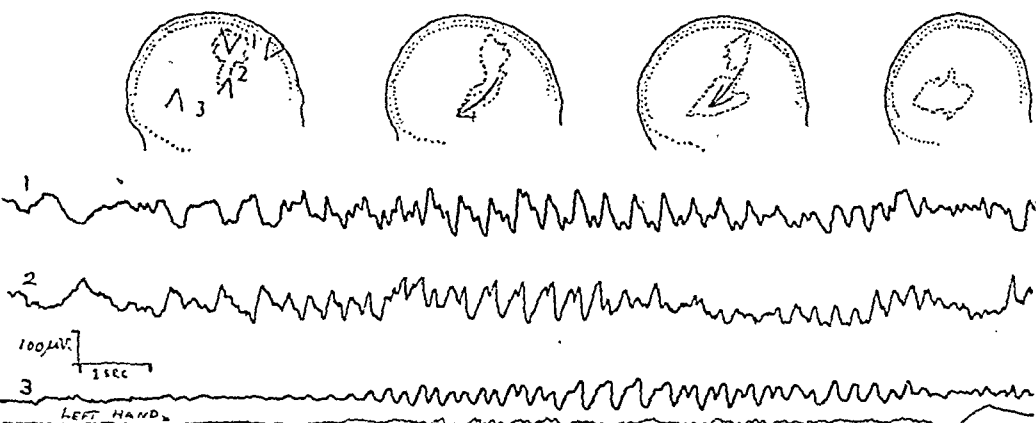


FIG. 1.

on the specimen removed suggested that the phenomena here might be due to minor interference with the blood supply to the area by perivascular gliosis, resulting in interference with oxygenation and removal of metabolites. The inference here is that, in spite of the local pathology, the genetic background was such as to discourage spreading of the electrical abnormalities beyond the neighbourhood of the mechanical interference, and, therefore, no generalized convulsion could develop. This would be the tentative explanation of so-called continuous partial epilepsy.

The second case had had a series of major fits in 1938; an E.E.G. at that time had shown bilateral synchronous slow waves of the epileptic type. In 1940 the patient was readmitted in status epilepticus, and shortly died. The last series of attacks were Jacksonian in type, starting in the right hand and face. At autopsy a small glioma was found in the left frontal lobe. Here the explanation would be that a strongly epileptic inheritance favoured the development of generalized convulsions at the earliest stage of the new growth.

Section of Psychiatry

President—A. A. W. PETRIE, M.D., F.R.C.P.

(April 14, 1942)

DISCUSSION ON TESTING INTELLECTUAL CAPACITY IN ADULTS

Major F. J. S. Esher: *Experience in testing the intelligence of dull military adults* (Précis).—The ideas in this paper were developed as the result of testing in an E.M.S. hospital, psychoneurotic soldiers who seemed dull or defective. The work was carried out by my colleague E. G. J. Bradford of the Sheffield University, and myself.

It was noticed that amongst inferior adults educational attainments or their lack tended to influence the scoring on some tests, that the personality of the testee required some investigation as it affected the relations between tests, and that the age of the testee had to be taken into account as older men scored, on the whole, poorer scores than the younger. I, therefore, propose to adopt in this paper the viewpoint that to test intelligence alone is of less worth in the case of the adult than it is in the child, whose personality has not yet crystallized.

Procedure.—All men admitted to the psychoneurosis centre were given the Progressive Matrices immediately on admission. Sets A B C D and E were used with unlimited time. School and employment histories were taken.

The following other tests were given: *Terman* (Stanford Revision). *Performance Battery* consisting of Passalong, Form Boards (graded series), Kohs' designs and Knox cubes—all designed or modified by E. G. J. Bradford.

Theoretical considerations.—A man's efficiency in the Forces depends on factors other than intelligence alone. We considered the main factors that made for success to be:

(1) Intelligence. (2) Knowledge. (3) Personality.

Discussion.—(1) We felt that intelligence was the most important of the three and the only one we could hope to measure. This is substantially so even now but I feel sure we are in a better position to speak of the effect of the other two. Intelligence enables a man to analyse his perceptions and to modify his behaviour in accordance with the analysis of the moment. Better intelligence makes for better analysis, though it is, of course, truer to say that the man with the better analytic power has the better intelligence.

(2) Knowledge is the accumulated results of analyses made through the agency of one's own intelligence or that of someone else (e.g. teacher, parent, instructor, &c.), by which it is possible to solve problems similar in their essence to previous ones (not necessarily one's own). The solution of a problem by knowledge rather than by intelligence appears to need the expenditure of less mental energy and is therefore a biological economy. It is an illustration that habitual actions are easier to carry out than new ones, but it must be noted that this method of solving a problem can only be developed if the individual has undergone sufficient relevant experience in the past; hence the immense value of education.

(3) Personality may be thought of as reflected in the attitude a person adopts to his environment (this includes objects real and unreal, animate and inanimate as well as himself). In his attitude he may attempt to mould the environment to suit himself, an aggressive one which may be either constructive or destructive. He may allow the environment to mould him, a passive dependent one; on the other hand he may create an imaginary and false environment because he cannot cope with his difficulties in the real world (difficulties largely within himself).

Each of these three factors plays an important part in the solution of intelligence tests —factors other than intelligence must materially influence the scoring of tests such as those of the Binet type which include not only material which can be solved by intelligence alone, but social situations, educational problems and questions requiring verbal comprehension, searches and so forth.

Such tests may test the intelligence of children with success if it is assumed that all children of normal intelligence are interested in and have experience of subjects taught at school, in simple social situations, in the meaning of words, in the use of numbers, &c. It is probably true for the majority, but for some children it is unquestionably not the case, e.g. some are blind or deaf, some chronically ill, and others are encouraged or

ventional anti-convulsant drugs. These facts suggest that convulsion thresholds vary enormously in each individual, and there is some reason for supposing that this is another aspect of E.E.G. data, leading to the idea of specific epileptic liability.

Two cases may illustrate this view. One is a woman who complained of attacks of twitching in the left arm and face which had developed after a comparatively trivial blow on the right side of the head. This persisted despite surgical removal of a part of the cortical area identified through the course of the abnormal electrical discharge which could be observed, during the attack, by the E.E.G. (fig. 1). The histopathological report

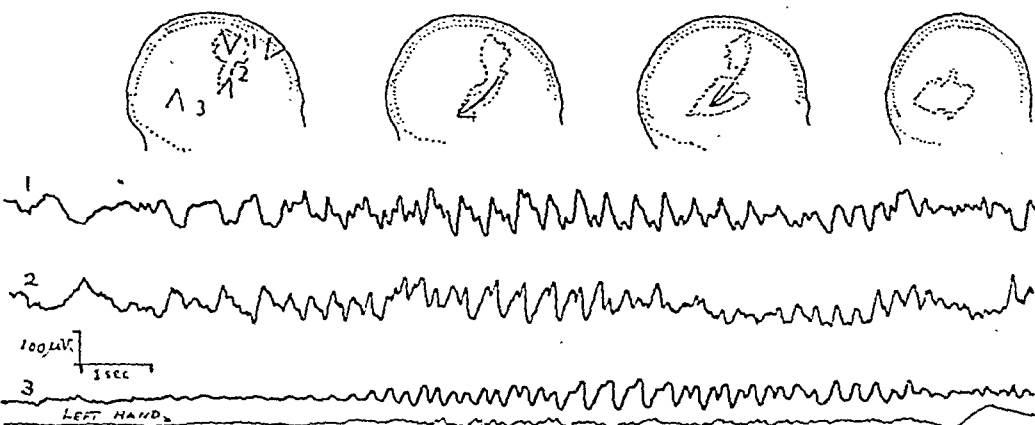


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develop some of their many symptoms during testing as soon as they realize they are facing a difficulty, namely, the increasing difficulty in solving the test problems; such people may complain that the booklet, the diagram or the room began to spin round after a time, or that they felt giddy, dizzy or faint, or got headache or palpitations. When these symptoms occur they are unable or unwilling to put their best efforts into the test.

When tests are timed, e.g. the Army's 20-minute version of the matrix test, careful and meticulous obsessives are apt to be penalized for their care and precision. Such men often waste much time checking and rechecking their results before proceeding to the next subtest.

Personality makes its effect more evident in performance tests. In these tests the testee can be observed in the process of carrying out a difficult task under defined conditions; one may see the nature of his mistakes and the way he behaves when he realizes he is balked and is failing. The tester is more truly an observer when carrying out performance tests than verbal ones. As my colleague, E. G. J. Bradford pointed out in *Occupational Psychology*, July 1941, 15, 116: "In Binet testing, the initiative is in the hands of the tester, whereas in the performance tests it is mostly left to the subject . . . the tests themselves measure the power of sustained effort in a way that oral tests do not, and hence they have a value apart from that of discovering intellectual weakness."

Scatter in a test has long been known to be suggestive of instability. The same may be said about a correlation between one test and another that is lower than expected. A measure of the homogeneity between scores is the "mean square contingency coefficient". If it is small much scatter and instability may be suspected.

The contingency "C" was 0.55 between two measures of intelligence applied to M.D.s but only 0.05 between the same two measures of intelligence among neurotics. This strongly suggests that maladjustment of personality affects the accuracy of tests as measures of intelligence.

Individuals express their intelligence differently using mainly verbal, abstract-symbolic or practical (visuo-spatial) media. Tests should therefore be chosen to give testees the opportunity of displaying their abilities in the above three media.

It may be well to note here that test conditions should be satisfactory. Disturbance by noise or interruptions, fatigue, hunger, ill-health or unpleasant associations with the testing room, may interfere with a testee's ability to put his best into the test.

In assessing the testee's scores notice must be taken of the fact that men, as they get older, have increasing difficulty in passing tests. Wechsler, for example, incorporates this principle in his tests by having different norms for different ages over 25 years.

In my own figures a preliminary survey showed that it was well marked in Kohs' designs and Passalong tests in men of over 30 years of age. The effect was less in Form Board, Binet, Vocabulary and Matrix tests.

Taking 20-25 age-group as standard, older age-groups scored less and their scores may be expressed as percentages of the scores obtained by the standard group (20-25 years).

26-31 age-group:

Scored 92% in Matrix.
97% in Form Boards.
97% in Passalong.
52% in Kohs.
91% in Binet.

32-37 age-group:

Scored 83% in Matrix.
79% in Form Boards.
70% in Passalong.
44% in Kohs.
100% in Binet.

This age effect is marked in men of the lower levels of intelligence and is probably less so among the most intelligent. Among the dumbest it probably sets in earlier in life. If this is so, it is in keeping with the known fact that mental defectives mature slowly and senesce rapidly and early; thus at 25 years the defective may look 17 whereas at 35 he looks 45.

These observations suggest that the estimation of a man's ability in the Army or industry must be based not only on intelligence but also his standard of knowledge and his personality. Tests, therefore, which are strongly affected by each of the three factors will be more accurately prophetic than tests of intelligence alone. They should also test ability in the various media described above and due allowance should be made for the effect of age.

In the case of adults a figure or value for intelligence alone is unsatisfactory. One must have something that measures or describes the individual as a whole. Ideally, this estimate should enable one to assess a man's ability in any particular sphere.

Mr. J. C. Raven: A psychological test is designed to reveal individual differences of response to a standard test situation. The problems remain constant and a testee's responses are usually evaluated in terms of the mean and dispersion of responses for a

discouraged to do school work. It seems obvious to me that such children will be handicapped in their attempts to pass the tests. Burt pointed out long enough ago that in certain areas 30% of all children attending elementary schools were backward (i.e. educationally below a level 15% less than their mental age).

Dull adults, on the other hand, seem to fall into two main groups. The one contains people who have slowly learned the lessons of years and who can successfully bring lengthy training and tested experience (i.e. knowledge), unlikely in a child, to their own assistance when faced with intelligence tests such as those of the Binet type. The other group contains people whose schooling and subsequent training were of poor quality and who therefore failed to develop what intelligence they had; such folk often not merely stagnate, they lose what little knowledge they derived from school (e.g. reading and writing) and in addition lose the power of exercising their intelligence unless they are urged or helped by someone else.

The first group tend to obtain better scores on some intelligence tests than their real native abilities warrant, and the second tend to fail miserably when they should be able to do better.

Children may therefore be said to acquire knowledge under ordinary circumstances in close proportion to their intelligence and for this reason tests solved by knowledge rather than innate ability are still quite good measures of intelligence. In adults, on the other hand, knowledge which, according to circumstances, may or may not have been acquired, may disturb test scores, hence one's assessments of their intelligence, because it is less clearly related to intelligence than in children. This is especially so in tests of the Binet type, though in other tests, e.g. Passalong, training and experience seem to have less effect.

One has only to think of such subtests as "The difference between president and king", or "Searching for a ball in a field", giving "Difference between abstract words", solving "Arithmetical problems", "Reading and report", Problems of fact and many others, to realize what a help (often an essential help) knowledge is to their satisfactory solution.

The mere fact that, owing to circumstance, some intelligent adults are illiterate makes it impossible for them to attempt certain tests, such as "Reading and report", "Coding a message", "Dissected sentences", &c. We have seen many such men who were often sent to the hospital as mental defectives on grounds of military inefficiency or inability to learn the training given in the Unit. They were clearly different from the typical dull and sluggish mental defectives and were usually found able to pass normally in performance test; in my reports they were described as Scholastic Defectives, and one might say they were Burt's "educationally retarded children" grown up. They constantly gave histories of loss of education for various reasons, the more frequent being chronic sickness, nomadic or difficult parents, problematic behaviour, infantile psychoneurosis or cerebral trauma.

Though many such men were sent for examination as mental defectives (and their school history, unsatisfactory employment and "untrainability" in the Army suggested it) we were seldom in doubt about their diagnosis. On the whole, they were bright in their replies and quick in their movements, they had good knowledge of everyday events and presented a more normal appearance than is seen in "typical mental defectives".

In testing, they scored M.A.s of $9\frac{1}{2}$ to 12 years in the Binet tests, scored normally in the Performance Battery and tended to score normally in vocabulary tests if they were successful in their employment and social contacts, but badly if they were unsuccessful. The socially unsuccessful scholastic defectives were so unable to manage themselves normally that they were easily certifiable as mental defectives under the Mental Deficiency Acts.

Personality expresses itself in the solution of all intelligence tests, indeed any other test. The person whose attitude towards a task is aggressively constructive is, other things being equal, more likely to succeed in his task than, shall we say, the person of dependent type.

In childhood, a certain dependence upon adults is considered natural and is therefore no detriment to the child. In adults, however, an independent and more mature personality is expected and a dependent personality is a real handicap. This is, I feel, an important reason why group tests are relatively unsatisfactory with many adults and why individual tests are essential in such cases for the formation of more accurate assessments of their intelligence.

Inability to persist at a task is a frequent source of failure in testing both adults and children. This is often seen in the hospital when matrix tests have been given. Scores for A and B series may be good, but those for C, D and E far below expectations considering the A and B score. The same effect is also seen in hysterics; they tend to

obtained can be surprisingly reliable but it must be remembered that a test of this type is essentially an individual test, that it requires approximately forty minutes to administer, that Binet worked with children and that the method and concepts he employed are really only suitable for work with children or with adults who remain relatively childish.

(2) For the purpose of detecting intellectual inferiority amongst adults, choice of test depends largely upon the number of people to be examined and the available number of competent psychologists.

Burt (1937) pointed out that dull children are usually disproportionately backward in school subjects and, in general, non-verbal tests are more suitable than verbal tests. For individual examination performance tests are useful. Where large scale group testing is necessary a simple form of matrix test is useful. The reliability of the results obtained depends upon the type of test chosen. Individual tests are in general more reliable than group tests.

(3) For the purpose of indicating a person's general fund of information and clearness of intellectual judgment verbal tests of intelligence are suitable.

Both individual and group verbal tests are fairly reliable and results naturally correlate well with scholastic ability and ability to follow instructions. A verbal test of intelligence has the defect that one does not know how far success is due to scholastic training, verbal fluency or genuine intellectual ability. A good vocabulary test together with a good non-verbal test of intellectual ability gives more clearly defined results but at present there is no really satisfactory vocabulary test available for group testing.

(4) For the purpose of selecting candidates for special training the type of test employed depends upon the type of training.

Ability to benefit from any training which is more than training in the execution of a routine task depends essentially upon a person's ability to reason by analogy and intellectual ability in the sense of capacity for quick, clear, accurate judgment is of primary importance both for the acquisition of knowledge in any class of work and for the appropriate utilization of the knowledge acquired. Where men are to be selected and trained to hold positions in which it is necessary to make decisions or give instructions to other people this is particularly true. I would even argue that clarity of judgment was the essential capacity necessary for holding the respect of other people.

The best method of assessing intellectual superiority in the sense of superior capacity for quick, clear, accurate judgment is to provide an opportunity for a person to acquire a logical method of reasoning and subsequently to test the rate and accuracy with which he is able to apply the method he has acquired. Although intellectual capacity is of primary importance, ability to acquire any particular form of training depends also upon physique, temperament, aptitude, environment and previous training—in so far as these terms can be adequately defined—as well as upon intellectual ability as defined in this paper.

¹Progressive Matrices 1938 was designed to cover the widest possible range of intellectual development. It was not designed to differentiate clearly between individuals of any one level of mental ability. Used with people of sub-normal ability a relatively large chance factor is involved and the reliability of the results obtained is correspondingly low.

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Dr. C. J. C. Earle: What is measured by so-called intelligence tests is the level of organization of behaviour in a standard situation. The ability to behave intelligently requires a mixture of endowment and acquirement of sensorimotor elements as well as imaginal, of non-cognitive as well as cognitive. In the case of adults we face the added difficulty of the enormous modification of behaviour by training, both social and scholastic, by specialized knowledge, habits of thought and many other factors. For this reason tests devised for and standardized on children are quite useless: we must obtain our standards from homogeneous and comparable groups.

Despite these difficulties it is possible to obtain a good working measure of what is commonly called a man's general level of intelligence. But even the theoretical "potential native endowment" of intellect depends on at least three psychoneural properties: the complexity of neural organization; the degree and speed of variability of neural pattern; and the capacity for selective inhibition, or mental synergy. These three processes may be presumed to be more efficient in intelligent than in unintelligent people. But it is a matter of neurological fact that they vary independently even in the normal. *Intelligence, however conceived, is not a unit variable.* And when one adds all the various factors of acquirement, it is obvious that what is measured by tests of "general intelligence" or by aggregated scores from a battery is a mere conglomeration. Therefore, while it is undoubtedly valuable to have some working knowledge of the general level of intelligence, it is not worth while to spend much time or skill in getting it: this is the only justification for the use of group tests.

The analogy may be drawn between level of intelligence in the psychological field and stature in the physical. Even in very careful physical examinations we do not measure stature accurately because it is not a unit variable, and its accurate measurement gives no important information. If we were to classify on the basis of stature we would find that a mongol, an infantile, a kyphotic and a man with both legs amputated, would all fall in the same category. What is needed, rather than an accurate measure of total height, is some idea of the proportions which go to make up that total; and also of what other facts about the physical make-up may throw light upon the problem. Accurate knowledge of stature, without knowledge of its components, is practically useless.

The position is the same with general intelligence. To know the level, however

given population of individuals. In some tests the responses are judged or marked by the psychologist as, in his opinion, revealing characteristic qualities or satisfying a certain criterion of correctness. In other tests a person's actions, judgments or choices are recorded and evaluated without the personal opinion of the psychologist being involved.

Both forms of test are open to criticism. The first type can only be given satisfactorily by a competent psychologist. Results obtained with the second type are apt to be vitiated by influences which pass unobserved. It must also be recognized from the outset that responses of the greatest interest to the psychologist are responses which from the essential uniqueness of their nature cannot be evaluated in terms of the mean or dispersion of responses given by other people.

Dr. Earl once said that no one has yet succeeded in giving a satisfactory psychological definition of "mental defect". I would say no one has yet succeeded in giving a satisfactory psychological definition of "mental ability". Even the word "intelligence" may mean "a piece of information" or "clearness of mental apprehension".

In his work on "The Nature of Intelligence and Principles of Cognition" Spearman (1927) drew a sharp distinction between "reproductive" and "educative" mental processes, that is, between mental retentivity and intellectual ability.

Intellectual ability may be defined as *ability to reason by analogy from awareness of relations between experienced characters*. Conversely *intellectual defect* may be defined as *a permanent inability to form comparisons and reason by analogy*. "Intellectual defect" defined in this way is not necessarily synonymous with "general mental defect" but it is probably one of the chief causes of proneness to accidents and social failure (Raven, 1942).

The ideal test of intellectual ability is a situation which records under standard conditions either the rate or the accuracy with which a person is able to apprehend the characters of immediately presented experiences, deduce relationships between the characters apprehended and conceive logical correlates of the characters apprehended and the relations deduced.

We have to infer a person's intellectual ability from a record of his intellectual activity. Past intellectual activity is recorded in a person's general fund of knowledge and information. Present intellectual activity is recorded in the number and kind of problems he is able to solve.

Neither a person's fund of information nor intellectual activity at any given time provides a completely satisfactory method of inferring his real intellectual ability. A person's fund of information depends upon retentivity of memory, fluency of recall, upon past experience and education as well as upon his intellectual ability. Deductions concerning a person's intellectual ability from his mental activity at a given time in a given test situation involve all the practical and theoretical problems of "sampling".

A person may do well under test conditions and fail under other conditions, or he may do badly under test conditions and be able to make perfectly sound intellectual judgments under other conditions. Available experimental data indicate that, provided we regard a person's response to a test situation as essentially a response of the whole personality to a single situation, the principles of "sampling" can be applied to psychological testing, but problems are involved which still need to be investigated and in the absence of adequate data we must be prepared to reserve judgment.

Apart from questions of "sampling" as a psychological method, practical problems exist:

(1) Any test used must record a person's intellectual activity under standard conditions. In psychological work this is surprisingly difficult. Even if it were possible to keep the entire test situation constant, people would still vary in their attitude towards the test and in their willingness to co-operate. It is usually necessary to sacrifice some degree of accuracy in the control of a test situation to secure a relatively uniform co-operative attitude on the part of the person being tested.

(2) We cannot record intellectual activity. We can only record a person's actions, judgments or choices (Raven, 1939). Even the simplest judgment involves more than purely intellectual activity, it involves a decision. A decision involves cognitive control and cognitive control involves temperamental disposition. A choice, as used in mental testing, involves search and search involves remembering what one is looking for. If one does not remember what one is looking for, choice becomes chance (Miller and Raven, 1939).

(3) We can only measure the rate or the accuracy of a person's judgments. Rate and accuracy of judgment vary with health, with practice, with the material used in the test and with distracting or facilitating influences at the time of testing. Even if we are prepared to accept the belief that innate intellectual ability is a constant quality of a person capable of quantitative measurement we are not justified in assuming that the rate or the accuracy of a person's judgments in a given test situation, however carefully designed and controlled, provides an adequate measurement of the person's entire intellectual ability which in its usual meaning includes processes other than those tested.

In choosing a mental test we should bear in mind the purpose for which we want a test and the reliability of the results we expect to obtain.

(1) For the certification and classification of mental defectives' tests of the Binet type are useful. Tests of this type are really standard psychological interviews by means of which a psychologist is able to indicate in the form of a mental age and intelligence quotient the approximate level of general mental development which a person has reached and the rate at which development is taking place. In the hands of a competent psychologist the results

whether the subject reacts more markedly to situations of novelty—as do the immature and excitable types—or to those involving intellectual difficulty—which sometimes completely disorganize the behaviour of apparently phlegmatic people. We can see whether such reactions as there may be take the form of excitement or of inhibition. We note that some people lower their scores through carelessness, others through over-caution; that some fail because they are afraid to try, and others because they do not realize the futility of their effort.

The purely cognitive evidence is reinforced by that from the psychomotor field. The essential importance of motor evidence has been amply proved by the work of Luria in Russia, and of Bills and others in America: the contributions of Kretschmer and of Enke are also highly important; yet this qualitative observation of performance test behaviour is at present the only clinically practicable method of assessing this essential factor.

It is obviously impossible in a brief paper to discuss the exact differential diagnostic significance of the various findings. But it is quite certain that the components of behaviour in the individual personality under test are presented for inspection in the test situation. There is the degree and directedness of the subject's striving; there is his excitability or its opposite; his reaction to his success or failure; and his preferred mode of approach. I agree that these tests do not give an accurate measure of the "g" factor or of the power of educing relationships or of abstract thinking, or of any other succinctly definable scientific abstraction. For the matter of that clinical mental testing does not give a really accurate *measure* of anything—a fact which is raised as an objection to such a mode of approach. But it does give an adequate picture of the behavioural component, it does allow a trained examiner to analyse intellectual process, and it does show the place occupied by intelligence within the personality. Most important of all it does allow of an adequate prediction of behaviour.

[May 19, 1942]

Heredity in the Psychoneuroses (*Summary*)

By FELIX W. BROWN, D.M.

THE part played by heredity in the development of the psychoneuroses is one of the fundamental unsolved problems of psychiatry. Psychotherapy entails so much consideration of the environmental situations that it is easy to ignore the possible hereditary aspect of the problem one is dealing with. But the chief difficulty is to define the condition the heredity of which one is attempting to trace. A psychiatric diagnosis for a psychoneurosis can still only be made on symptoms, rather than on aetiology. This introduces the same difficulties as if one were to try to investigate the heredity of a symptom such as cough. There is no neat blood test, as in hæmophilia, or well-established syndrome as in Huntington's chorea, to help in the diagnosis of a psychoneurosis. There is by no means as valid a series of diagnostic criteria as in manic-depressive psychosis. The psychoneuroses are inefficient and escapist reactions of qualities which are inherent in human nature, and which, when not exaggerated, are biologically useful.

For the present purposes the psychoneuroses were considered in three groups, which were defined as follows:

Anxiety states.—Psychoneuroses characterized by some of the somatic symptoms of palpitations, shaky feelings, giddiness, indigestion, tight feelings in the chest, with the mental symptoms of an affect of anxiety, without a preponderance of depression, and various phobias such as fear of diseases, fear of closed places, open spaces, &c.

Hysteria.—A psychoneurosis characterized by definite physical conversion symptoms such as paralysis, anaesthesia, fits, vomiting, blindness, aphonia, and certain mental symptoms such as an affect of indifference and periods of amnesia. This reaction is biologically akin to the possum reaction of a captured weasel or young lapwing.

Obsessional state.—A psychoneurosis characterized by ritualistic acts and thoughts, which the patient recognizes as absurd, and which are not associated with a preponderant affect of depression. This is related to habit formation in childhood and to mankind's primeval desire for ritual and sympathetic magic.

These definitions do not of course ensure that one is dealing with homogeneous material. One may be investigating the heredity of several different conditions under each of these headings.

The other great difficulty is that of separating the part played by heredity from that of environment. A series of statistics might be produced, complete with mean deviations.

accurately, is of little use. We need to know its make-up. We need to know not only *how intelligent a man is*, but also *how is a man intelligent*. The accurate measure of intellectual level, alone and of itself, is merely misleading. What is wanted is an analysis of the actual intellectual function in that individual case, and also of the degree and nature of the integration of intellect with the personality as a whole.

A fully objective or mechanical analysis of intellectual process is not yet possible, for there is no test which cannot be solved in more than one way. This has been clearly shown by Weisenberg and McBride in their study of aphasics. So-called non-verbal tests were not in fact non-verbal for all the cases. The question of what is verbal and what is not can only be settled by careful individual examination. There are wide differences also in the verbal tests themselves. For one man words are pure symbols, for another they are very intimately connected with auditory and kinæsthetic imagery. Many quite intelligent people are curiously bad at pure logical verbal analysis. In the performance field too, even the Vigotsky block test, designed as the perfect measure of pure concept formation, has recently been shown to be soluble on largely perceptual lines, and many highly intelligent people solve it in this way. The analysis of the intellectual process is, therefore, a highly individual affair. Fortunately its really accurate analysis is rarely necessary in clinical work and probably never in personnel work.

In dealing with the integration of the intellect with the personality the influence of acquirement by education and special habits of thought is of great importance. When any cognitive capacity is completely integrated it ceases to be a true measure of intellectual efficiency, for no ability for new learning is needed for its reproduction. For a normal average adult practically no intelligence need be exercised to name the months of the year; what is required here is the product of intelligence which has been exercised in the past. Tests which measure productive intelligence in the child or in the illiterate do no more than measure reproductive activity in the average normal man.

Vocabulary tests are undoubtedly the best measure of reproductive intelligence. An adult's knowledge of the meaning of words is fairly fully stamped in. Vocabulary tests, therefore, give an index of the best level to which an individual has attained—whatever may have happened to it since. Interference with vocabulary only occurs in very grave disorders. The test is limited in value for morons, dullards, those having or having had special reading disabilities, and bi-lingually educated people such as certain Welsh country folk: all of whom score far below their potential. To a greater or lesser extent the same thing is true of all verbal tests. The degree to which they measure productivity, as against reproductivity, is always doubtful, and I know of no verbal test which is a safe measure of ability for new learning in an adult. Such tests, therefore, need care and discrimination in their application.

Performance tests demand new learning and productive intelligence and are an essential for an adequate examination. A combination of verbal and performance tests gives us the relative ability of the individual in the two main psychoneural fields: the verbal-symbolic and the spatial-objective. But performance tests give far more than that, for it is with these tests that we can analyse, albeit subjectively, the intellectual process itself and also estimate the degree and mode of integration of intelligence with the total personality and the degree to which behaviour is influenced by emotional factors.

Performance tests demand a more complete stage of behaviour than do verbal tests. They demand the taking of decisions and the release and control of motor activity; while if they are difficult enough they show the reaction to difficulty and failure. Crudely speaking, the comparison between verbal and performance ability is an index of the degree of interference in intellectual process. But it tells us nothing of the nature of the interference; it does not differentiate between dementia and neurosis; nor does it tell us at what point in the test the interference occurred.

If we are to know about the efficiency of functioning of the intelligence, or to predict behaviour, we must know these things. We must therefore use clinically adequate performance tests, which allow of a really effective observation of behaviour, for such observation is the only present means of filling the gap between the pure mental process which we think or hope to measure and the score on the mental test. The observation of test behaviour allows one to judge not only the degree but the nature of disturbance of function. Overt behaviour is a function of the total organism. The test, though it stresses the cognitive aspect, is in fact always a personality test, and the subject projects his personality into the situation for the examiner to read. We can see at what point, for instance, he uses the conceptual approach, and at what point he thinks perceptually: we can see him turn from planning to trial and error; and we can note whether he profits from the chance successes of that method or whether he uses it blindly. Moreover we can usually form a fairly valid judgment as to why these changes occur. We can note

of their not being desperately ill. Only 31 controls were taken, but even these are sufficient for some purposes. It was not as easy to investigate the controls as the psychoneurotics for several reasons: (1) I was not the patient's physician and the same contact was not always able to be made. (2) For some curious reason these patients and their immediate relatives knew and cared less about their remoter relatives than the psychoneurotics. (3) These patients and the other informants seemed to have less insight into personality traits than the psychoneurotics and could not give as good a history of other relatives. In some cases, however, I happened to pick on the one normal member of a psychiatrically very abnormal family, and usually here a good history was obtained. This may of course mean that if a lot is known about a family, psychiatric abnormalities will always be found, but my impression is that psychoneurotics and their relatives give a better account of personalities than the normals. These controls cannot of course be regarded as typical of the general population, because they were all ill, but it was hoped that in respect to psychoneurotic incidence they could be regarded as at least something with which to compare my results. The illnesses from which these patients were suffering were conditions such as tuberculosis, heart failure, &c. in which there is considered to be no neurotic trait. An interesting point emerges not directly concerned with the present subject. This is the part played by psychobiological situations in determining the onset of a physical disease. Many of the patients gave histories of difficulties which they had encountered similar to those with which we are familiar in taking histories of psychiatric cases. However, instead of reacting by a psychoneurosis, they fell victims to a physical disease or their rheumatic heart became decompensated. This is far from the same as saying that these diseases are psychological in origin, but is merely a way of stating the platitude that the chain breaks at its weakest link.

For instance, when in a run-down state after three self-induced abortions in five years and when she was trying to get a divorce from her husband who had deserted her, a 28-year-old woman developed rheumatic arthritis. A pot shopkeeper with a pancreatic cyst developed diabetes after a period of worry and insomnia following the opening of Woolworth's next door, although he had had the cyst for some years previously.

STATISTICAL RESULTS

Owing to space limitation it is practicable here to show only some of the tables of results. *Influence of position in family.*—The positions of psychoneurotics in the sibships of the propiiti were charted, so that the number of first children, only children, youngest children, &c. could be investigated. It was seen that the psychoneurotics occur apparently scattered at random in any position in the family. There were but 4 only children among the propiiti. Dr. Lewis Fanning has kindly confirmed the random occurrence of these cases statistically. Thus according to the series of cases here examined, the position in family is of no significance in determining whether or not a person will develop a psychoneurosis (the full proof of this cannot be published here).

Incidence of abnormalities in the parents of patients and controls.—This is shown in Table III. It will be seen that 46.8% of the parents of anxiety states are normal,

TABLE III.—INCIDENCE OF PSYCHIATRIC ABNORMALITIES IN PARENTS OF PSYCHONEUROTIC PATIENTS AND CONTROLS.

| | Anxiety states | | | | Hysteria | | | | Obsessional state | | | | Controls | |
|--------|----------------|-------|-------|--------|----------|------|-------|--------|-------------------|-------|-------|--------|----------|------|
| | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % |
| N | 59 | 46.8 | 33.8 | ± 7.7* | 17 | 40.5 | 40.1 | ± 9.6* | 20 | 50.0 | 30.6 | ± 9.4* | 50 | 80.6 |
| An | 27 | 21.4 | 21.4 | ± 5.4* | 4 | 9.5 | | | 3 | 7.5 | 7.5 | ± 3.4* | 1 | 1.6 |
| Hy | 2 | 1.6 | | | 8 | 19.0 | 17.4 | ± 5.6* | 3 | 7.5 | 7.5 | ± 3.4* | | |
| O | 7 | 5.6 | | | | | | | 3 | 7.5 | 7.5 | ± 3.4* | | |
| D | 1 | 0.8 | | | | | | | | | | | | |
| S | 1 | 0.8 | | | | | | | | | | | | |
| P | 22 | 17.5 | 4.6 | ± 5.7 | 6 | 14.3 | 1.4 | ± 6.8 | 13 | 32.5 | 19.6 | ± 8.2* | 1 | 1.6 |
| AP | 2 | 1.6 | | | 3 | 7.1 | | | | | | | 8 | 12.9 |
| Alc | 2 | 1.6 | | | | | | | 1 | 2.5 | | | 1 | 1.6 |
| Ep | 3 | 2.4 | | | 4 | 9.5 | | | | | | | 1 | 1.6 |
| PP | | | | | | | | | | | | | | |
| Totals | 126 | 100.1 | | | 42 | 99.9 | | | 40 | 100.0 | | | 62 | 99.9 |

* = statistically significant figure.

significantly different from the control figure of 80.6%. The 21.4% of parents suffering from anxiety state is also significant.

Significant figures for hysteria are 40.5% normal parents, and 19% hysteric parents. The obsessional show 50% normal, and 7.5% obsessional parents. This is a much lower figure than Lewis obtained, in whose series, in 1936, 37% were obsessionals. This difference is probably due to many cases being counted as obsessionals in his series which would

which would only show that children are capable of imitating their nervous parents. This difficulty cannot be escaped except by extensive twin investigation, or by study of children who have been brought up away from their parents. As this latter situation does, however, in itself provide a great environmental stress, this method also is invalid.

SELECTION OF CASES

A small group of cases has been taken and investigated thoroughly, concentrating on reliability of data rather than on numbers. 104 cases have been taken comprising 63 anxiety states, 21 hysterics and 20 obsessional. The age and sex distribution is shown in Table I. These cases have not been selected in any way. They were taken entirely at random from the out-patients satisfying the diagnostic criteria at the Maudsley

TABLE I.—AGE AND SEX CHART OF PROPOSITI AND CONTROLS.

| Age-group | Anxiety state | | | Hysteria | | | Obsessional state | | | All psychoneuroses | | | Controls | | |
|-----------|---------------|----|------------|----------|----|------------|-------------------|---|------------|--------------------|----|------------|----------|----|------------|
| | ♂ | ♀ | Both sexes | ♂ | ♀ | Both sexes | ♂ | ♀ | Both sexes | ♂ | ♀ | Both sexes | ♂ | ♀ | Both sexes |
| 10-14 | | | | | | | | | | | | | | | |
| 15-19 | | | | | | | | | | | | | | | |
| 20-24 | 2 | 1 | 3 | | 1 | 1 | 1 | 1 | 2 | 3 | 3 | 6 | 2 | 2 | 4 |
| 25-29 | 7 | 8 | 15 | | 6 | 6 | 3 | 1 | 3 | 10 | 14 | 24 | 1 | 1 | 2 |
| 30-34 | 2 | 7 | 9 | 2 | 4 | 6 | 2 | 1 | 3 | 6 | 12 | 18 | 5 | 5 | 10 |
| 35-39 | 10 | 5 | 15 | 1 | 4 | 5 | 1 | 1 | 2 | 12 | 10 | 22 | 3 | 3 | 6 |
| 40-44 | 2 | 1 | 3 | 1 | 1 | 2 | 1 | 4 | 5 | 6 | 15 | 21 | 4 | 4 | 8 |
| 45-49 | | | | | | | | | | 6 | 8 | 14 | | | |
| 50-60 | 1 | 1 | 2 | | 1 | 1 | 1 | | 1 | 2 | 2 | 4 | 2 | | 2 |
| Totals | 34 | 29 | 63 | 5 | 16 | 21 | 15 | 5 | 20 | 54 | 50 | 104 | 19 | 12 | 31 |

Out-patient Department, Guy's Hospital, and the Cassel Hospital, so that the cases are not confined to one social class. I knew nothing of the patient's family history before investigating a case. I may add, before starting this work I had a prejudice against the significance of heredity in the psychoneuroses. With the exception of the cases at the Cassel, I was myself responsible for the psychotherapy of the patients. Not only was the detailed family history often helpful in the treatment of the patient, but also the fact that I was treating the patient helped me to obtain excellent co-operation from the patients and their relatives. I did not have the services of a social worker, and all the interviewing of relatives was done by myself. Though much time was thus taken up, the advantage was that the human factor was simplified, the human error being my own, not my own and that of a social worker. In all about 500 relatives were interviewed, and information obtained concerning 2,288 relatives of the patients. In each case a diagnostic summary of the patient's case was prepared, then a family history from at least one other relative as well as the patient. Details were requested for each first and second degree relative, but needless to say a full account was not always available for each relative. Having obtained the case-histories the next step was to classify all the relatives seen and those about whom accounts were taken, according to their psychiatric abnormalities (Table II).

TABLE II.—KEY TO ABBREVIATIONS IN CLASSIFICATION OF RELATIVES.

| | |
|-----|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| N | Psychiatrically normal. |
| An | Definite anxiety state, amounting to social or work incapacity at some time. |
| Hy | Definite hysteria, paralyses, amnesic fugues, hysterical fits, with social or work incapacity at some time. |
| O | Definite obsessional psychoneurosis, with ritualistic acts or thoughts, causing some incapacity. |
| D | Manic-depressive psychosis, with suicides, mental hospital admissions, depressive stupor, &c. with incapacity for work at some time. This includes involutional depressive states and puerperal depressions. |
| S | Schizophrenic psychosis. |
| P | Psychosis of unspecified nature, including general paralysis, senile dementia, and various cases not able to be accurately diagnosed from the data. |
| AP | Anxious personality, including timid apprehensive personality, excessive worrying, phobias not amounting to definite psychoneurosis; obsessional personality, with <i>folie de doute</i> , excessive worry over details; depressive personality with well-marked mood swings. In well-marked cases it is possible to distinguish between these groups, but usually these personality traits blend into one another, and often all are present in the same relative. |
| Alc | Alcoholism. |
| Ep | Epilepsy. |
| PP | Psychopathic personality, including odd, eccentric people, paranoid psychopaths, impulsive and quarrelsome people, wanderers unable to settle to any job with any success, unstable psychopaths. |
| MD | Mentally defective. |

CONTROLS

A series of controls was taken of roughly the same age-groups as the psychoneurotic patients. The controls were selected among the medical patients in the wards at Guy's. They were selected only by accessibility of relatives and information, and by the fact

of their not being desperately ill. Only 31 controls were taken, but even these are sufficient for some purposes. It was not as easy to investigate the controls as the psychoneurotics for several reasons: (1) I was not the patient's physician and the same contact was not always able to be made. (2) For some curious reason these patients and their immediate relatives knew and cared less about their remoter relatives than the psychoneurotics. (3) These patients and the other informants seemed to have less insight into personality traits than the psychoneurotics and could not give as good a history of other relatives. In some cases, however, I happened to pick on the one normal member of a psychiatrically very abnormal family, and usually here a good history was obtained. This may of course mean that if a lot is known about a family, psychiatric abnormalities will always be found, but my impression is that psychoneurotics and their relatives give a better account of personalities than the normals. These controls cannot of course be regarded as typical of the general population, because they were all ill, but it was hoped that in respect to psychoneurotic incidence they could be regarded as at least something with which to compare my results. The illnesses from which these patients were suffering were conditions such as tuberculosis, heart failure, &c. in which there is considered to be no neurotic trait. An interesting point emerges not directly concerned with the present subject. This is the part played by psychobiological situations in determining the onset of a physical disease. Many of the patients gave histories of difficulties which they had encountered similar to those with which we are familiar in taking histories of psychiatric cases. However, instead of reacting by a psychoneurosis, they fell victims to a physical disease or their rheumatic heart became decompensated. This is far from the same as saying that these diseases are psychological in origin, but is merely a way of stating the platitude that the chain breaks at its weakest link.

For instance, when in a run-down state after three self-induced abortions in five years and when she was trying to get a divorce from her husband who had deserted her, a 28-year-old woman developed rheumatic arthritis. A pot shopkeeper with a pancreatic cyst developed diabetes after a period of worry and insomnia following the opening of Woolworth's next door, although he had had the cyst for some years previously.

STATISTICAL RESULTS

Owing to space limitation it is practicable here to show only some of the tables of results.

Influence of position in family.—The positions of psychoneurotics in the sibships of the propiiti were charted, so that the number of first children, only children, youngest children, &c. could be investigated. It was seen that the psychoneurotics occur apparently scattered at random in any position in the family. There were but 4 only children among the propiiti. Dr. Lewis Fanning has kindly confirmed the random occurrence of these cases statistically. Thus according to the series of cases here examined, the position in family is of no significance in determining whether or not a person will develop a psychoneurosis (the full proof of this cannot be published here).

Incidence of abnormalities in the parents of patients and controls.—This is shown in Table III. It will be seen that 46.8% of the parents of anxiety states are normal,

TABLE III.—INCIDENCE OF PSYCHIATRIC ABNORMALITIES IN PARENTS OF PSYCHONEUROTIC PATIENTS AND CONTROLS.

| | Anxiety states | | | | Hysteria | | | | Obsessional state | | | | Controls | |
|--------|----------------|-------|-------|--------|----------|------|-------|--------|-------------------|-------|-------|--------|----------|------|
| | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % |
| N | 59 | 46.8 | 33.8 | ± 7.7* | 17 | 40.5 | 40.1 | ± 9.6* | 20 | 50.0 | 30.6 | ± 9.4* | 50 | 80.6 |
| An | 27 | 21.4 | 21.4 | ± 5.4* | 4 | 9.5 | | | 3 | 7.5 | 7.5 | ± 3.4* | 1 | 1.6 |
| Hy | 2 | 1.6 | | | 8 | 19.0 | 17.4 | ± 5.6* | 3 | 7.5 | 7.5 | ± 3.4* | | |
| O | 7 | 5.6 | | | | | | | | | | | 1 | 1.6 |
| D | 1 | 0.8 | | | | | | | | | | | 8 | 12.9 |
| S | 1 | 0.8 | | | | | | | | | | | 1 | 1.6 |
| P | 22 | 17.5 | 4.6 | ± 5.7 | 6 | 14.3 | 1.4 | ± 6.8 | 13 | 32.5 | 19.6 | ± 8.2* | | |
| AP | 2 | 1.6 | | | 3 | 7.1 | | | | | | | | |
| Alc | 2 | 1.6 | | | | | | | 1 | 2.5 | | | 1 | 1.6 |
| Ep | 3 | 2.4 | | | 4 | 9.5 | | | | | | | 1 | 1.6 |
| PP | | | | | | | | | | | | | | |
| Totals | 126 | 100.1 | | | 42 | 99.9 | | | 40 | 100.0 | | | 62 | 99.9 |

* = statistically significant figure.

significantly different from the control figure of 80.6%. The 21.4% of parents suffering from anxiety state is also significant.

Significant figures for hysteria are 40.5% normal parents, and 19% hysteric parents.

The obsessional show 50% normal, and 7.5% obsessional parents. This is a much lower figure than Lewis obtained, in whose series, in 1936, 37% were obsessionals. This difference is probably due to many cases being counted as obsessionals in his series which would

be included in the present series as obsessional personalities (AP). There are 7.5% of manic depressives in the parents of obsessionals, a significant figure.

Thus in all the three psychoneuroses, there seems to be some evidence of breeding true some connexion between obsessional state and manic-depressive psychosis, and less between anxiety state and depression. There proved to be no significant difference in incidence between mothers and fathers (table not shown here).

Abnormalities in siblings over 15.—These are shown in Table IV. The same tendency appears. In the anxiety states and obsessionals, the proportion of normals is significantly

TABLE IV.—PSYCHIATRIC ABNORMALITIES IN SIBLINGS OVER 15 OF PSYCHONEUROTIC PATIENTS AND CONTROLS.

| | Anxiety states | | | | Hysteria | | | | Obsessional state | | | | Controls | |
|--------|----------------|-------|-------|---------|----------|-------|-------|--------|-------------------|-------|-------|--------|----------|------|
| | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % | Diff. | S.E. | No. | % |
| N | 140 | 61.4 | 28 | ± 5.07* | 52 | 80 | 0.4 | ± 5.3 | 36 | 64.3 | 25.1 | ± 6.2* | 110 | 89.4 |
| An | 28 | 12.3 | 12.3 | ± 3.03* | 3 | 4.6 | | | 3 | 5.4 | | | | |
| Hy | 5 | 2.2 | | | 4 | 6.2 | 5.4 | ± 2.5* | | | | | 1 | 0.8 |
| O | 2 | 0.9 | | | | | | | 4 | 7.1 | 7.1 | ± 2.4* | | |
| D | 2 | 0.9 | | | | | | | 1 | 1.8 | | | | |
| S | 1 | 0.4 | | | 1 | 1.5 | | | | | | | | |
| AP | 38 | 16.7 | 7.8 | ± 3.88* | 4 | 6.2 | | | 11 | 19.6 | 10.7 | ± 5.3 | 11 | 8.9 |
| Alc | 2 | 0.9 | | | | | | | | | | | | |
| Ep | 1 | 0.4 | | | | | | | | | | | 1 | 0.8 |
| PP | 3 | 3.5 | | | 1 | 1.5 | | | | | | | | |
| MD | 1 | 0.4 | | | | | | | 1 | 1.8 | | | | |
| Totals | 228 | 100.0 | | | 65 | 100.0 | | | 56 | 100.0 | | | 123 | 99.9 |

lower, 61.4% and 64.3%, as compared with 89.4% in the controls. The 12.3% of anxiety states in sibs of anxiety states, and 7.1% of obsessionals in sibs of obsessionals is also significant. The AP group is also significant in the sibs of anxiety states.

First-degree relatives.—Table V shows the incidence in the most important abnormalities for all first-degree relatives, i.e. parents, children and siblings of psychoneurotic

TABLE V.—INCIDENCE OF CHIEF ABNORMALITIES IN FIRST-DEGREE RELATIVES.

| | Anxiety states | | Hysteria | | Obsessional state | | All psychoneurotics | | Controls | |
|------------------|----------------|-------|----------|-------|-------------------|-------|---------------------|------|----------|------|
| | No. | % | No. | % | No. | % | No. | % | No. | % |
| N | 208 | 57.0 | 69 | 64.5 | 60 | 59.4 | 337 | 58.8 | 164 | 86.8 |
| An | 55 | 15.1* | 7 | 6.5 | 3 | 3.0 | 65 | 11.3 | | |
| Hy | 8 | 2.2 | 12 | 11.2* | | | 20 | 3.5 | 2 | 1.1 |
| O | 2 | 0.5 | | | 7 | 6.9* | 9 | 1.6 | | |
| AP | 61 | 16.7 | 10 | 9.3 | 25 | 24.8* | 96 | 16.8 | 19 | 10.1 |
| D | 9 | 2.5 | | | 4 | 4.0 | 13 | 2.3 | | |
| Other conditions | 22 | | 9 | | 2 | | 33 | | 4 | |
| Totals | 365 | | 107 | | 101 | | 573 | | 189 | |

patients. There is significant evidence of similar inheritance, of 15.1% in anxiety states, 11.2% in hysterics, and 6.9% in the obsessionals. There is also a significant incidence of anxiety states, in the relatives of hysterics and obsessionals, and of depressive psychosis in the relatives of obsessionals and anxiety states.

Second-degree relatives.—Table VI shows the incidence of the more important abnormalities in all second-degree relations, i.e. uncles, aunts, grandparents, nephews, nieces, half-

TABLE VI.—INCIDENCE OF CHIEF ABNORMALITIES IN SECOND-DEGREE RELATIVES.

| | Anxiety states | | Hysteria | | Obsessional state | | Controls | |
|------------------|----------------|-------|----------|------|-------------------|-------|----------|------|
| | No. | % | No. | % | No. | % | No. | % |
| N | 533 | 72.4 | 207 | 79.3 | 178 | 71.2 | 251 | 89.9 |
| An | 20 | 2.7 | 5 | 1.9 | 1 | 0.4 | 2 | 0.7 |
| Hy | 3 | 0.4 | 4 | 1.5 | 2 | 0.8 | 1 | 0.4 |
| O | | | | | 2 | 0.8 | | |
| AP | 87 | 11.8* | 24 | 9.2 | 42 | 16.8* | 17 | 6.1 |
| D | 20 | 2.7* | 4 | 1.5 | 11 | 4.4* | 1 | 0.4 |
| Other conditions | 73 | | 17 | | 14 | | 7 | |
| Totals | 736 | | 261 | | 250 | | 279 | |

sibs, grandchildren. The data of course are not as reliable as those for first-degree relatives. The significant figures are those for depression in second-degree relatives of anxiety states (2.7%), and of obsessional states (4.4%) and for anxious personality in relatives of anxiety states (11.8%) and of obsessional states (16.8%). The high incidence of depression in second-degree relatives is probably due to the fact that they are on an average fifteen years older than the first-degree relatives who have not had time to develop their depressions.

Twins.—There were four pairs of fraternal twins among the propoiti, only one member of each pair being psychoneurotic. One pair was studied in detail. Physically they resembled one another very closely, but one was an hysteric and the other a normal healthy woman. They were brought up together but their biographies showed a gradual divergence of personality. Their finger-prints show no resemblance at all, in spite of the physical resemblance. One can only conclude that they were fraternal twins closely resembling one another physically but not in temperament, although they shared approximately the same environment. This is in itself an argument for an hereditary factor in the psychoneuroses. It also suggests that this factor is not in any way linked with any physical traits; that personality and physique can vary independently. Of the other twins, one pair were of opposite sexes, the other two pairs were of the same sex but dissimilar.

Family histories of Service psychoneurotics.—Table VII shows the incidence of psychiatric abnormality in the parents and siblings of 30 consecutive cases of soldiers, sailors and

TABLE VII.—INCIDENCE OF ABNORMALITIES IN PARENTS AND SIBLINGS OF 30 CONSECUTIVE SERVICE CASES.

| | Parents | | Siblings | |
|---------------|---------|-------|----------|-------|
| | No. | % | No. | % |
| N | 37 | 61.7 | 125 | 87.4 |
| An | 3 | 5.0 | 6 | 4.2 |
| "Shell shock" | 3 | 5.0 | | |
| D | 4 | 6.7 | 1 | 0.7 |
| P | 1 | 1.7 | | |
| AP | 11 | 18.3 | 9 | 6.3 |
| MD | | | 1 | 0.7 |
| Alc | 1 | 1.7 | 1 | 0.7 |
| Totals | 60 | 100.1 | 143 | 100.0 |

airmen who were failing to adjust for psychoneurotic reasons. The data were hastily collected in the course of ordinary work and among Service cases in E.M.S. hospitals. There is a relatively high incidence of abnormality in the parents, especially of depression. The siblings are not very abnormal.

Follow-up of cases.—A postal follow-up was done recently on all these 104 psychoneurotic patients. Of the 63 anxiety states, replies were obtained concerning 28. Of these all had returned to work, 11 were completely recovered, 12 better but with some symptoms, 3 were the same, and 2 had died, 1 of duodenal ulcer and 1 of a stroke. Four were grade 1 in the Services. Of the obsessional 11 replies were obtained. One had completely recovered, 6 better but still had symptoms, 3 were worse, and 1 had committed suicide. The 1 who recovered attributed her recovery entirely to halibut oil. Two were in the Forces graded B, 6 were at work, but in 1 case a schoolmaster had become a fire watcher, 2 were incapacitated. Of the hysterics, 14 replies were obtained, 3 were recovered, 5 much improved, 3 unchanged, and 3 were worse. One was grade A.1. in the Army, 1 had been boarded out of the Army. Six were at work and 6 were incapacitated. In none of these cases did the family history provide a reliable guide to the prognosis of the patient. Some with the worst family history had done best. All these cases had received some psychotherapy, some of them quite intensively. The cases that had done badly had attended most of the available psychiatrists, one bad obsessional had received shock therapy with no improvement. The interesting point about this follow-up is the good prognosis of the anxiety states. The degree of conversion seems to be an index of bad prognosis. It is of course impossible to generalize from such a small follow-up, especially as those who did not reply may all be in mental hospitals, but I doubt it. The obsessionals and hysterics who had done badly did not hesitate to describe their miserable state.

CONCLUSIONS

In this series, without detracting from the importance of the environmental factor or of psychotherapy, a case is made out for the significance of heredity in the development of the psychoneuroses. The random occurrence of psychoneurosis in the sibships and the cases of fraternal twins are probably the strongest arguments for heredity playing a significant part.

Assuming then that there is a hereditary factor, what is its nature? It is easier to answer this negatively than positively. It is not a recessive, only one case of consanguinity was found. Simple dominance is also excluded; the ratio of first-degree relatives affected, in the aggregate, does not suggest simple dominance, though at times individual family trees can be found where the inheritance looks simple dominant.

There is a great probability that the inheritance of many commonplace human characters is on the lines of variable dominance, where the environment also plays a large part.

be included in the present series as obsessional personalities (AP). There are 7.5% of manic depressives in the parents of obsessionals, a significant figure.

Thus in all the three psychoneuroses, there seems to be some evidence of breeding true some connexion between obsessional state and manic-depressive psychosis, and less between anxiety state and depression. There proved to be no significant difference in incidence between mothers and fathers (table not shown here).

Abnormalities in siblings over 15.—These are shown in Table IV. The same tendency appears. In the anxiety states and obsessionals, the proportion of normals is significantly

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| | Anxiety states | | | | Hysteria | | | | Obsessional state | | | | Controls | |
|--------|----------------|-------|-------|---------|----------|-------|-------|--------|-------------------|-------|-------|--------|----------|------|
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| N | 140 | 61.4 | 28 | ± 5.07* | 52 | 80 | 9.4 | ± 5.3 | 36 | 64.3 | 25.1 | ± 6.2* | 110 | 89.4 |
| An | 28 | 12.3 | 12.3 | ± 3.03* | 3 | 4.6 | | | 3 | 5.4 | | | | |
| Hy | 5 | 2.2 | | | 4 | 6.2 | 5.4 | ± 2.5* | 4 | 7.1 | 7.1 | ± 2.4* | 1 | 0.8 |
| O | 2 | 0.9 | | | | | | | 1 | 1.8 | | | | |
| D | 2 | 0.9 | | | | | | | | | | | | |
| S | 1 | 0.4 | | | 1 | 1.5 | | | | | | | | |
| AP | 38 | 16.7 | 7.8 | ± 3.88* | 4 | 6.2 | | | 11 | 19.6 | 10.7 | ± 5.3 | 11 | 8.9 |
| Alc | 2 | 0.9 | | | | | | | | | | | | |
| Ep | 1 | 0.4 | | | | | | | | | | | 1 | 0.8 |
| PP | 8 | 3.5 | | | 1 | 1.5 | | | | | | | | |
| MD | 1 | 0.4 | | | | | | | 1 | 1.8 | | | | |
| Totals | 228 | 100.0 | | | 65 | 100.0 | | | 56 | 100.0 | | | 123 | 99.9 |

lower, 61.4% and 64.3%, as compared with 89.4% in the controls. The 12.3% of anxiety states in sibs of anxiety states, and 7.1% of obsessionals in sibs of obsessionals is also significant. The AP group is also significant in the sibs of anxiety states.

First-degree relatives.—Table V shows the incidence in the most important abnormalities for all first-degree relatives, i.e. parents, children and siblings of psychoneurotic

TABLE V.—INCIDENCE OF CHIEF ABNORMALITIES IN FIRST-DEGREE RELATIVES.

| | Anxiety states | | Hysteria | | Obsessional state | | All psychoneurotics | | Controls | |
|------------------|----------------|-------|----------|-------|-------------------|-------|---------------------|------|----------|------|
| | No. | % | No. | % | No. | % | No. | % | No. | % |
| N | 208 | 57.0 | 69 | 64.5 | 60 | 59.4 | 337 | 58.8 | 164 | 86.8 |
| An | 55 | 15.1* | 7 | 6.5 | 3 | 3.0 | 65 | 11.3 | | |
| Hy | 8 | 2.2 | 12 | 11.2* | | | 20 | 3.5 | 2 | 1.1 |
| O | 2 | 0.5 | | | 7 | 6.9* | 9 | 1.6 | | |
| AP | 61 | 16.7 | 10 | 9.3 | 25 | 24.8* | 96 | 16.8 | 19 | 10.1 |
| D | 9 | 2.5 | | | 4 | 4.0 | 13 | 2.3 | | |
| Other conditions | 22 | | 9 | | 2 | | 33 | | 4 | |
| Totals | 365 | | 107 | | 101 | | 578 | | 189 | |

patients. There is significant evidence of similar inheritance, of 15.1% in anxiety states, 11.2% in hysterics, and 6.9% in the obsessionals. There is also a significant incidence of anxiety states, in the relatives of hysterics and obsessionals, and of depressive psychosis in the relatives of obsessionals and anxiety states.

Second-degree relatives.—Table VI shows the incidence of the more important abnormalities in all second-degree relations, i.e. uncles, aunts, grandparents, nephews, nieces, half-

TABLE VI.—INCIDENCE OF CHIEF ABNORMALITIES IN SECOND-DEGREE RELATIVES.

| | Anxiety states | | Hysteria | | Obsessional state | | Controls | |
|------------------|----------------|-------|----------|------|-------------------|-------|----------|------|
| | No. | % | No. | % | No. | % | No. | % |
| N | 533 | 72.4 | 207 | 79.3 | 178 | 71.2 | 251 | 89.9 |
| An | 20 | 2.7 | 5 | 1.9 | 1 | 0.4 | 2 | 0.7 |
| Hy | 3 | 0.4 | 4 | 1.5 | 2 | 0.8 | 1 | 0.4 |
| O | | | | | 3 | 0.8 | | |
| AP | 87 | 11.8* | 24 | 9.2 | 42 | 16.8* | 17 | 6.1 |
| D | 20 | 2.7* | 4 | 1.5 | 11 | 4.4* | 1 | 0.4 |
| Other conditions | 73 | | 17 | | 14 | | 7 | |
| Totals | 736 | | 261 | | 250 | | 279 | |

sibs, grandchildren. The data of course are not as reliable as those for first-degree relatives. The significant figures are those for depression in second-degree relatives of anxiety states (2.7%), and of obsessional states (4.4%) and for anxious personality in relatives of anxiety states (11.8%) and of obsessional states (16.8%). The high incidence of depression in second-degree relatives is probably due to the fact that they are on an average fifteen years older than the first-degree relatives who have not had time to develop their depressions.

Section of Otology

President—F. W. WATKYN-THOMAS, F.R.C.S.

[March 6, 1942]

A Study of the Activities of the Intratympanic Muscles. [Summary]

By F. W. KOBRAK, M.D.

In 1925 the author described a series of cases in which deafness of internal-ear origin was associated with middle-ear signs; in this series there were cases of Ménière's syndrome and of pontine and circum-pontine lesions such as bulbar palsies, disseminated sclerosis and encephalitis lethargica. As a result of these observations it was suggested that in the control of the tonus of the intratympanic muscles there was an element provided by the vestibular mechanism. After this preliminary work clinical observations showed that in some cases of genuine cochlear deafness there were signs of middle-ear disease and that such cases were often improved by treatment of the middle ear.

Following the experimental work on animals of H. Kobrak (1930), Hallpike (1935) and others on the intratympanic muscles (1930-1935) the earlier clinical work was supplemented by the administration of drugs which were known to have effect on impaired muscle tonus; special attention was paid to tetrophan (Schering) a coal-tar derivative. As a result of these observations a clinical relationship was detected between alterations of middle-ear muscle tonus and some varieties of otosclerosis (the "spasmophile" form) and "middle-ear inefficiency" in some cases of cochlear deafness (1938). Further information was given by a new method of testing hearing, which was intended primarily to extend the use of tuning forks in regard to loudness rather than intensity. The results obtained support the possibility of a muscular factor in some cases of deafness. This work (*J. Laryng. & Otol.*, 1940, 55, 405) may be summarized thus: If a tuning fork is struck with a force of constant intensity it is heard for X seconds. It is then struck again, and after a lapse of X seconds is held to the ear. In the normal ear it is generally heard only for a short period, Y seconds. Again the fork is struck, and after X + Y seconds is held to the ear for the third time. In the normal subject it is seldom heard at all; if it is, the third "Z" period of hearing is very short indeed, even shorter than the "Y". In many pathological cases the "steppage" of the periods is quite different, and additional periods are often found.

This "protraction of the hearing period" cannot be ascribed to fatigue; it strongly suggests an active inhibition of hearing, and such an inhibition could be produced most easily by the action of the intratympanic muscles.

Hitherto the action of the tympanic muscles has been regarded as a damping effect against sounds of dangerous intensity—a protective mechanism. But the animal experiments show an interesting discrepancy; the full effect of the muscle activities, as registered by the contractions, do not come into force until the damping, as shown by the Wever-Bray effect, has passed its climax or is nearly over. This suggests a dual muscular effect—a linked reflex of "protective" and "corrective" damping. It seems possible that the protective damping is more dependent on vestibular stimulation, the corrective damping more on cochlear stimulation. It is not suggested that corrective damping in any way resembles visual accommodation; it is not an adaptation for "near hearing", but it is a mode of response to quickly changing sound intensities. Damping adaptation is slowly developed during the first months of life, and with increasing auditory experience reaches a stage of muscular automatism. For the quickly changing sound intensities of speech and music to be appreciated, a ready flow of change in damping intensities seems indispensable to give elasticity in hearing; and such muscular automatism must be correlated with the vestibular-balanced tone of the intrinsic muscles. It will be realized that there are essential differences between testing hearing for speech, which is more dependent on elasticity of hearing and therefore on the integrity of the tympanic musculature, and testing for hearing of pure tones. Without some corrective type of damping, apart from the gross, protective damping, it is difficult to see how basic perception of music is possible. In orchestral music coinciding piano and fortissimo would only be perceptible, if protective damping changed to corrective damping in some 20 milliseconds.

With the psychoneuroses, however, we are not dealing with definite diseases, but only reaction types. The clinical classification of psychoneuroses into anxiety, hysteria, and obsessional is arbitrary, though convenient. This classification is supported by the observation that these conditions to some extent breed true, about 15% in anxiety states and about 7% in hysteria and obsessional states. These three conditions also seem to be related genetically to one another and the obsessional state, and to a less extent the anxiety state, and much less hysteria, to the manic-depressive psychosis. They also seem to be related to a more indefinite personality deviation which I have called anxious personality which is in itself not definitely abnormal, but yet which is possessed by most of our patients. To postulate a gene for anxiety state, one for hysteria and one for obsessional, even a variable dominant gene, seems much too simple to fit the facts. Another theory would be that psychoneuroses occur by a certain shuffling of the kaleidoscope of factors responsible for normal personality. The fact that psychoneuroses occur in the relatives of normals to some extent would support this. In some families it seems as though the psychoneuroses have arisen as a result of combinations of factors not in themselves pathological, though the combination proves to be. For instance, a combination of overconscientiousness with low intelligence may well predispose a patient to develop a psychoneurosis, though these qualities separately may not be beyond the range of normality. This theory, however, would not account for the observed greater incidence in the relatives of psychoneurotics, and it is probably not the whole story. I would suggest, however, that the development of, say, an obsessional state rather than an anxiety state depends more on the commonplace personality factors, which are probably themselves variable dominants, than on the specific psychoneurotic factor, if it exists. It may perhaps be that there are one or more pathological variable dominant factors, of the order of constitutional emotional sensitivity, determining whether or not a psychoneurosis can develop in a suitable environment.

The high ratio of similar inheritance obtained in some of the figures for first-degree relatives suggests in fact that the inheritance of the underlying diathesis responsible for psychoneurosis in general is not excessively complex. It may even be a simple variable dominant. If this were so, then the particular type of psychoneurosis may depend on the particular grouping of contributory personality factors, not necessarily in themselves pathological. Some such combined inheritance is most likely to fit such data as exist. It is possible to make a scheme of multi-factor inheritance to fit almost any genetic ratio, but it would be wiser at this stage to let the figures speak for themselves rather than to speculate further. The environment in which these psychoneuroses developed was the peace-time environment, and the stresses were the familiar ones of family and sexual relationships and work difficulties. Psychoneuroses developing in these conditions may be more endogenous than those arising in the acute and extraordinary stresses of war time. It is moreover probable that many of the environmental situations of these psychoneurotics arise as a result of their characters, rather than that their characters are a result of their environment. To split the influence of environment from heredity decisively is at present impossible, but nevertheless from such evidence as we have, heredity plays quite as important a part as environment in the development of the psychoneuroses.

This work was done with the aid of a grant from the Medical Research Council, whom I wish to thank, especially Sir David Munro. I also wish to thank Dr. R. D. Gillespie for his help at Guy's and for initiating this research, Drs. Anderson and Rogerson for their help at the Cassel, and to acknowledge the help of the late Professor Mapother who allowed me to use the Maudsley material. I particularly wish to thank Dr. Lewis Fanning of the Institute of Hygiene for his invaluable assistance with the statistics, and Dr. Elliot Slater for some very useful advice.

A motor-driver had always been very deaf. His upper tone was lowered both by air and bone. Here again there was a drop at 4096—quite a stepping down at that level.

A riveter, aged 27, had been engaged on this trade for three years. His history was that while he was riveting he suddenly noticed that his right ear became deaf. He is now completely deaf in that ear. There is also a drop at 4096 in his left or "good" ear. Is that a danger signal? Should he give up this work?

A boy of 14 had a blind right eye due to congenital hæmorrhage. He complained of a tendency to deafness, but heard conversation at 20 ft. with both ears. Bone conduction was normal. But here again there was the extraordinary drop at 4096. The boy's father complained of much the same thing, but the drop was less.

The next case was a lady of 67, who had had some deafness for many years. She heard spoken words at 12 ft. Again there was this drop at 4096.

The last case is another soldier, aged 30, who complained of slight deafness in the left ear. He heard with the right ear conversation at 20 ft., with the left, conversation at 18 ft. His lower tone limit was normal, his upper tone limit on the left side greatly reduced. Here again there was a large drop at 4096.

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It is clear that the participation of the intrinsic muscles in hearing has not only a theoretical but also a clinical significance, especially in some forms of progressive deafness hitherto found refractory to treatment. If there is any interference with the activity of the reflex arc which controls the intrinsic muscles some effect on hearing is inevitable. Even apart from the question of corrective damping, hyperexcitability of the arc would bring the protective damping mechanism into action for intensities far below the normal threshold of stimulus; this might account for the protracted period of hearing. Moreover cochlear hypo-excitability would be deleterious. Instead of the muscles being thrown into a premature spasm (vestibulogene), the cochlea would be deprived of a valuable protection, and fatigue would come on far more quickly and last longer.

Routine measures of treatment provide a field for research by analysing the detailed effects of such therapy. The assessment of the improvement of hearing by inflation in cases of cochlear deafness without any concomitant middle-ear change needs further consideration. The same applies to the results sometimes obtained in cases of dry perforations by the use of an artificial drum.

The effect of medication as well as of mechanical treatment in such cases needs investigation. So far no direct evidence is available as to changes in the structure of the cochlear nerve in cases of deafness by therapy in the way of arrest of or recovery from the degenerative process, or of regression in the otosclerotic changes in the labyrinthine capsule; but it is hoped that clinical evidence may be afforded by an application of varied forms of the numbing effect. Audiometer tones of highest intensity failed to produce conclusive results. Thereupon, the numbing effect was investigated in the following way: The tuning fork c' (Edelmann) was hammered close to the ear over a period of some thirty seconds, until there was a definite sensation of giddiness and pressure in the ear. Air conduction figures of the audiogram pointed to lesions in the organ of Corti. However the figures of bone conduction were not analogous to those of air conduction; they pointed to a second extracochlear numbing effect, which seems to improve, relatively, the figures of bone conduction against those of air conduction. Here, once more, damping activities of the tympanic muscles are suspected. Dual intracochlear and extracochlear effects are suggested to be in operation not only with the experimental shock stimuli of numbing, but also with the mitigated "numbing effects" of ordinary hearing, on which further research is now in progress.

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Deafness Due to Central Lag

By W. M. MOLLISON, M.Ch.

THERE is a type of deafness found especially at the age of 50-60 which is not revealed by the usual tests of spoken words. Slowly spoken sentences may be heard well, but rapidly spoken sentences are not grasped. The first word or two are heard and the rest is lost. The conducting and perceiving apparatus appears to be normal, but the central receptive apparatus is sluggish; that is to say, there is a central lag.

One case is that of a highly intelligent man aged 57, who complained of deafness, saying that he could not hear what was said at committees of which he was chairman. I found he heard quietly spoken words at 18 ft. on each side, but whole sentences he heard only at about 6 or 8 ft. Although he had been working hard he was not at all an exhausted subject. In the audiogram the lowest part of his scale was at 4096.

These cases are characteristic of quite a large number of people. At a Section meeting (*Proc. Roy. Soc. Med.*, **35**, 245) papers read by representatives of the R.A.F. pointed out that a great many flying personnel had this loss at 4096. After that discussion I looked at some of my audiograms and found that exhausted cases had a drop at 4096.

A lady aged 54 heard spoken words at 16 ft. on one side and 20 ft. on the other. Here again there was an enormous drop at 4096—very little drop above that.

A man aged 25, a soldier, heard spoken words at 20 ft. on the right side and 18 ft. on the left, but he could not hear his watch on the left side, and he had a marked drop at 4096 in the left ear and a slight drop in the other.

A man aged 40 had some tinnitus, the note of which fluctuated up and down the scale, and certain noises vibrated in his ears. For instance, the music of a band heard with the right ear was quite distorted, but, heard with the left, it was normal. His audiogram, again, shows a drop at 4096, and not much drop until that point is reached.

A motor-driver had always been very deaf. His upper tone was lowered both by air and bone. Here again there was a drop at 4096—quite a stepping down at that level.

A riveter, aged 27, had been engaged on this trade for three years. His history was that while he was riveting he suddenly noticed that his right ear became deaf. He is now completely deaf in that ear. There is also a drop at 4096 in his left or "good" ear. Is that a danger signal? Should he give up this work?

A boy of 14 had a blind right eye due to congenital hæmorrhage. He complained of a tendency to deafness, but heard conversation at 20 ft. with both ears. Bone conduction was normal. But here again there was the extraordinary drop at 4096. The boy's father complained of much the same thing, but the drop was less.

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[May 1, 1942]

DISCUSSION ON BRAIN ABSCESS

Mr. D. W. C. Northfield: *The management of cases of brain abscess.*—This series of cases of abscess of the brain comprises only those in which the infection has apparently spread from the ears or from the paranasal sinuses. Consideration of difficulties of diagnosis and treatment is followed by an outline of the method of treatment now adopted, and which is giving more hopeful results than former methods.

In the first group of cases in which differential diagnosis has proved difficult, the presence of acute suppurative meningitis has been a stumbling block. Lumbar puncture easily confirms its presence, but whether or not an abscess is also present may be uncertain. During the early stages of abscess formation the cerebrospinal fluid commonly contains a high cell count (predominantly polymorphonuclear) and a moderately raised protein content, but the chlorides are not reduced. The fluid is sterile, and the pleiocytosis steadily diminishes with a change to lymphocytes. There is no general meningeal infection although this may develop in unfavourable cases, with a corresponding increase in cells and fall in chlorides. Complementary to these changes in the cerebrospinal fluid is the clinical picture. In all expanding or space-occupying lesions in the cranium a detailed and accurate history is of great importance, in order to detect the chronological sequence of symptoms indicating increased intracranial pressure and spreading focal disturbance of neurological function. Neurological examination yields the necessary objective evidence of a localized brain lesion. Headache, vomiting and diplopia are common to meningitis and abscess, but drowsiness and mental obtusation suggest an abscess, and restlessness, delirium and severe toxæmia usually accompany meningitis. Persistent and early vomiting is often a symptom of an abscess in the posterior fossa. There may be little or no pyrexia or increase of pulse-rate, and indeed a bradycardia is a valuable sign of abscess, but in meningitis the temperature and pulse-rate are usually much raised. The importance of aphasia, hemianopia and other localizing paralytic signs need not be stressed. A leucocytosis in the blood is seldom present in an uncomplicated case of brain abscess, although usually considerable in meningitis.

When "focal signs" are found in a case of meningitis, if they are not due to an intracerebral collection of pus, they may be caused by a locus of leptomeningeal fluid.

In a girl of 20 radical mastoidectomy was performed for left chronic otitis media; the lateral sinus was opened and had to be plugged. Drowsiness and dysphasia rapidly developed and three days later the wound was reopened and two drams of pus were aspirated from within the cranium in the left temporal region. When she came under my care, poor co-operation prevented a satisfactory examination, but slight right-sided pyramidal signs developed and the cerebrospinal fluid was purulent and contained streptococci. The temporal region was again explored: no pus was found within the brain and the ventricle was easily tapped making a brain abscess highly unlikely. There was, however, a gush of purulent cerebrospinal fluid from between the brain and the cranium. She died; autopsy revealed no abscess but diffuse suppurative leptomeningitis. The "focal signs" in this case were clearly due to trapping of the cerebrospinal fluid around the temporal lobe.

In another case, in which a cerebellar abscess was suspected owing to the presence of localizing signs, an exploratory operation revealed no abscess, but a considerable quantity of cerebrospinal fluid was evacuated from the region of the cerebellopontine angle, and the boy made a rapid recovery.

Adams McConnell (1937) has described similar cases of loculation of cerebrospinal fluid in the posterior fossa; in his cases, symptoms of raised pressure and "focal signs" were associated with a cerebrospinal fluid of normal content.

Otitic hydrocephalus—now familiar to us by reason of the writings of C. P. Symonds—has been rare in my experience. It is usually to be distinguished from abscess by the comparative well-being of the patient, in spite of headache and perhaps vomiting, and by the absence of abnormal neurological signs apart from papilloedema. Changes in the cerebrospinal fluid are variable but generally of a mild order.

Girl aged 13 years: left chronic otitis media had been present for a year, and symptoms of increased intracranial pressure for about a week. There was papilloedema but no convincing focal signs. The cerebrospinal fluid contained 20 lymphocytes per c.mm. and 40 mg. of protein per 100 c.c. Compression of the right jugular vein gave a rise in cerebrospinal fluid pressure, but on the left side there was no rise. Lateral sinus thrombosis with otitic hydrocephalus was diagnosed, but the patient's continued deterioration led me to explore the temporal lobe, and no abscess was found. Mr. Keogh explored the mastoid and found an extradural abscess and thrombosed sinus and again explored the temporal lobe with negative findings. Recovery followed a stormy illness, complicated by a brain fungus and a cerebrospinal fistula through the mastoid wound, giving rise to a quadrantic hemianopia. The papilloedema subsided after a about a month.

In another group of difficult cases, the pathology does not yet seem satisfactorily explained. The clinical picture mimicks closely that of cerebral abscess, but no abscess is found on exploration, and the patients usually recover spontaneously. These may be cases of brain oedema due to thrombosis of cortical veins, although in a case under my care there was no abnormality of those veins exposed by craniotomy. Dott in 1940 described to the Section of Neurology similar cases which he considered were due to a non-suppurative encephalitis. Cortical venous thrombosis can be recognized with greater certainty when the affected veins drain the central areas of the cortex, the characteristic feature being epilepsy with sudden hemiparesis. The cerebrospinal fluid may contain an excess of cells and protein—and in severe cases it may be blood-stained.

At times it may be difficult to distinguish between an abscess and a tumour; the presence of otorrhœa may be misleading.

Female aged 40 years: right otorrhœa since childhood; several years bouts of severe headaches and vomiting with right homonymous visual hallucinations. In one such attack there was stupor for several days, when a mastoid operation was undertaken in order to exclude an extradural abscess. When she came under my care, there was drowsiness, neck rigidity and a positive Kernig: no papilloedema and the visual fields could not be tested; other neurological signs had no localizing value: cerebrospinal fluid, 440 mm. pressure, 5 cells per c.mm. and 60 mg. protein per 100 c.c.—compatible with a chronic abscess. Ventriculography indicated a right frontal tumour, and a meningioma arising from the sphenoidal ridge was removed at subsequent operation.

More common has been the finding of an abscess when it has been little or not at all suspected. There have been six cases of temporal and two of cerebellar abscess in which there was no clinical evidence of ear disease. Three of these died: at autopsy, in two there was golden fluid in the middle ear of that side only on which the abscess was situated, suggesting an old infection; and in one unmistakable evidence of bone disease on the same side as the abscess. Of the cases that lived one showed radiologically opaque mastoid air cells. The possibility of abscess should always be entertained in cases of suspected tumour if the history is short and if the cerebrospinal fluid gives any support, whether or not there is any evidence of a causative septic focus.

The significance of certain physical signs deserves greater emphasis. An abscess may not give rise to papilloedema; in my experience papilloedema has been absent in two out of three cases, and in cases of acute abscesses and in the early stages its absence is the rule rather than the exception. Neck rigidity and Kernig's sign are constant accompaniments of meningitis, but these signs must not be considered to indicate of necessity the presence of that condition. They are common findings in cases of cerebral tumour and their causation is related to brain herniation—through the hiatus tentorii and through the foramen magnum. An abscess causes similar dangerous herniations of the brain, so that nuchal pain and rigidity and Kernig's sign do not necessarily indicate meningitis; but those signs should always be viewed with gravity, and especially if associated with severe headache or drowsiness do they indicate the urgency of the situation.

During the last six years there have been under my care twenty-four cases of abscess secondary to ear or paranasal infection. Various methods of treatment have been tried and I have finally chosen the closed method which we owe to Clovis Vincent. In the following analysis are set out the results of the various procedures:

| ANALYSIS OF TREATMENT OF 24 CASES OF BRAIN ABSCESS. | | | | |
|-----------------------------------------------------|------------------------------------------|----------|-----|----------------------------|
| Cases | Treatment | Survived | | |
| 9 | Drained | ... | ... | 2 |
| 3 | Drainage only | ... | ... | 0 |
| 3 | Aspiration + drainage | ... | ... | 1 |
| 1 | Decompression + drainage | ... | ... | 0 |
| 2 | Aspiration + decompression + drainage | ... | ... | 1 |
| 15 | Not drained | ... | ... | 10 |
| 3 | Aspiration only | ... | ... | 2 |
| 4 | Decompression + aspiration | ... | ... | 2 (died later of epilepsy) |
| 2 | Aspiration + enucleation | ... | ... | 2 |
| 5 | Decompression + aspiration + enucleation | ... | ... | 4 |
| 1 | Enucleation only | ... | ... | 1 |

Various methods of draining an abscess have been employed and the results can be fairly described as disastrous, but the figures of survival for treatment by closed methods are much more favourable.

An analysis of the post-mortem findings is instructive, for it throws light upon the problems of treatment. Extensive and massive œdema of the brain was the most frequent finding, and pronounced brain herniation was noted in some of these cases. Diffuse leptomeningitis was the next commonest finding, and less often ventriculitis. Multiple abscesses were found in one case causing a honeycombing of the temporal lobe. In another case the abscess could not be detected by the needle. Pulmonary embolism caused the death of one patient. Another died in status epilepticus whilst temporarily discharged from hospital to await final enucleation of the abscess, which had been aspirated several times; the autopsy revealed a shrunken thick-walled abscess with no recent spread of the infection. Thus it would appear that treatment usually failed on account of massive œdema with brain herniation or because of acute meningitis (including ventriculitis).

Of the ætiology of brain œdema there is little knowledge, and indeed there is no unanimity of opinion as to the pathological criteria of its lesser degrees, nor of the precise mechanism of its development and spread. Its presence may be assumed if there is marked drowsiness, lack of improvement as a result of treatment (in the absence of meningitis), and the neurological signs of brain herniation. The treatment of œdema

in both abscess and tumour of the brain constitutes perhaps the most urgent and the most formidable problem in neuro-surgery. Dehydration methods have had and are still having a great vogue, but in my experience have proved disappointing. Indeed, I have been frequently impressed how in some cases œdema persists and spreads in spite of treatment, in a manner best described as fulminating and malignant.

By reason of the observations at autopsy of the displacements of the brain caused by œdema, and of the results of treatment, my opinion is hardening that the only hopeful line of attack is surgical decompression. This is by no means always successful, probably because it is not resorted to sufficiently early; and if advanced œdema is an irreversible change—this is almost certainly true. Such decompression for brain abscess is obtained by the Clovis Vincent procedure, and some measure of relief of tension results from the mere aspiration of the abscess.

The avoidance of meningitis and the treatment of the established condition *vis* with brain œdema in difficulty and importance. Chemotherapy helps, but other methods must be employed at the same time. Diffuse meningitis may occur at the same time as the spread of infection into the brain, or it may be due to the surgeon's activities. During drainage, it is very liable to occur as a direct spread over the surface of the brain, and the two-stage method was introduced to cope with this. Infection may also spread centrifugally in the brain, probably as a result of the dislocation of the infected area towards the drainage opening in the skull and the consequent disruption of the delicate newly-formed natural barriers to micro-organisms. Operative, autopsy and experimental studies show that at the deepest pole of an abscess its wall is thinnest, and it is in this area that secondary loculi commonly form. From this poorly walled-off pole of an abscess, there is a most important pathway of infection to the subjacent ventricle, for œdema softens the intervening white matter and the ependyma of the underlying ventricle may rupture into this softened area. Ventriculitis has been quite clearly caused in this manner in two cases. Finally, as is well recognized, the ventricle tends to wander as a diverticulum through the softened white matter towards the brain fungus which tends to develop as a result of drainage, and the diverticulum can easily rupture on its surface. The accepted treatment of acute suppurative meningitis uncomplicated by abscess is chemotherapy, maintenance of the patency of the cerebrospinal fluid channels by frequent lumbar puncture and the free administration of fluids—"forced drainage". If an abscess is present, the subarachnoid spaces are inevitably obstructed by the swelling of the brain and by the herniation of brain into the various cisterns. Thus attempts to drain cerebrospinal fluid by lumbar puncture lose effectiveness, and, indeed, are dangerous by accentuating herniation and compression of the mid-brain or medulla. Some decompressive measure is, therefore, as logical a form of treatment for the meningitis as for the œdema.

Successful treatment depends upon accurate diagnosis; this must include precise localization, an estimate as to the "age" of the abscess, and the presence or otherwise of meningitis. So essential is such information that no ancillary method should be ignored. A diagnostic burr hole and tapping through a clean area of skin may be all that is necessary for confirmation in some cases; on the other hand, one should never hesitate to employ ventriculography. If ventriculography confirms the diagnosis—an osteoplastic flap is reflected forthwith and the abscess contents evacuated through a wide-bore blunt needle, the operation field being swamped with a bactericide such as proflavine, to avoid its infection. If tension is not sufficiently relieved, the dura mater may be opened so as to provide an orthodox osteoplastic subtemporal decompression and, provided no pus has been spilt, there is apparently little or no risk of infection. Even if the dura mater is not opened, bone should be removed from the base of the flap so as to provide a gradual decompression which results from the stretching of the dura mater. A burr hole is made in the bone flap over the most superficial part of the abscess through which it may be subsequently aspirated. When a diagnostic tap reveals an abscess conveniently accessible, and if there is but little swelling of the brain, decompression may not be needed, or may be postponed. In cases of cerebellar abscess, provided the patient's condition allows of it, the dura mater of the posterior fossa is fully exposed in every case; the abscess can be aspirated more easily than through a burr hole behind the mastoid process. But the dura mater should probably never be widely opened owing to the proximity of the cisterna magna, and the consequent ease with which diffuse meningitis can occur. The aspiration of a cerebellar abscess affords much greater relief of tension than aspiration of a hemisphere abscess, owing to its strategical position in causing an obstructive hydrocephalus.

At the time of aspiration, the cavity of the abscess is irrigated with electrolytic sodium hypochlorite (milton). Using small quantities (less than the amount of pus obtained), 50% milton is allowed very gently to flow into the cavity and is then aspirated; large quantities of ropy pus can usually be obtained, which would otherwise be too viscid to

pass through the needle. When the fluid returns fairly clear, it is valuable to leave in the abscess a small quantity of 5% milton or of 1:1,000 buffered solution of proflavine. A bactericide in such a closed cavity appears to aid in the destruction of organisms (two abscesses have become sterile under treatment) and to accelerate the localization of infection and the maturation and contraction of the abscess wall. It is also a prophylactic against infection of the needle track. At first it may be necessary to repeat aspiration every few days, but these intervals gradually lengthen. The patient's condition, the amounts of pus obtained and the resistance of the abscess wall are all factors which are weighed in making a decision to re-aspirate.

Thorotrast (1-2 c.c.) injected into the abscess at one of the early aspirations is of great help. It gradually becomes "fixed" in the wall of the abscess and the size and the position of the abscess can be demonstrated radiologically. Thorotrast may fail to depict the whole of an abscess if there are several loculi. This happened in one case; over-confidence in the X-ray appearance led me to forget the possible presence of a deeper loculus, which was thus inadvertently ruptured during enucleation, and the patient died of a diffuse meningitis.

What criteria guide one as to when to enucleate the abscess? They are the subsidence of acute inflammation and the formation of a tough wall. This is indicated by the disappearance of toxæmia and of meningitis, reduction of intracranial pressure—and consequent softening of the decompression—and diminution in the neurological signs of a focal cerebral lesion. The toughness of the abscess wall can be estimated by the blunt aspirating needle, and by the radiological appearance of the thorotrast shadow which diminishes in size and acquires a crisp crenated outline. There is a well-defined optimum period for successful enucleation. The above pathological criteria must be satisfied for obvious reasons, but if operation is unduly postponed, it becomes technically more difficult owing to the formation of an excessive degree of scarring around the abscess, leading to an unnecessarily wide excision of tissue. Secondary loculi or separate deeply placed abscesses may develop if a case is left too long. During the optimum period, the abscess wall is well defined, is sufficiently tough to "handle", and separates cleanly from the surrounding white matter. In this series, the age of the "youngest" abscess has been six weeks, and of the "oldest", six months, estimating age from the first symptom of intracranial disorder. The third month (i.e. between eight and twelve weeks) appears to be the "time of election" for enucleation.

Enucleation is facilitated by uncapping the wall of the abscess, excising with that portion of brain the adherent area of dura mater which constitutes the scarred track of the aspirating needle. When dissecting around the abscess, one must be ever alert for any prolongation of the abscess in a totally unexpected direction. The deeper the dissection the greater the care to be exercised to avoid rupturing its wall, and if the abscess is large and tense it is helpful to aspirate partially its contents, taking precautions against contaminating the field and the instruments, and the small hole must be closed by sutures. When the abscess is adherent to the wall of the ventricle, an opening into the latter may be unavoidable. In cerebellar abscess, the dissection is more difficult owing to the proximity of cranial nerves, and the abscess may be adherent to the dura mater covering the posterior surface of the petrous bone.

It is well recognized that epilepsy often follows the healing of an abscess, and enucleation may very likely avoid or diminish the frequency of this unfortunate sequel. But until the abscess has been finally eradicated an anticonvulsant should be given. In two cases fatal status epilepticus occurred after the patient left hospital; in one case the abscess had been drained, and had healed completely; the other has already been referred to. In neither case did an autopsy reveal any recent exacerbation of infection.

If the results of closed methods are examined again, it will be seen that they reflect the morbidity of the disease at its different stages. Some cases die in the early stages; extensive œdema was present at autopsy in the case which died following aspiration: the fatal issue might have been prevented by decompression, but there is no good reason to suppose that drainage would have been more successful. Of the three that died after aspiration and decompression, in one only was there extensive œdema. Only one case died out of eight in which enucleation was carried out, and this roughly indicates the relatively low risk of a fatal issue provided the early dangers can be safely overcome.

(That portion of the paper dealing with treatment is a summary of one delivered before the Society of British Neurological Surgeons, and is published fully in the *Journal of Neurology and Psychiatry*, 1942, 5, 1.)

Reference—McCONNELL, A. A. (1937), *Brain*, 60, 315.

Mr. Terence Cawthorne: An abscess of the brain is always secondary to a focus of infection elsewhere in the body, and the seat of this primary infection is to be found more frequently in the ear than anywhere else.

That there may be a direct connexion, even a track, between the ear and the brain

abscess which has arisen from it is often the case. But to look upon this track as a channel along which pus can flow either from the abscess to the ear or from the ear to the abscess is an oversimplification of the problem that may have led to the belief that in the presence of intracranial symptoms it is less unfavourable to find copious than scanty discharge from a diseased ear.

Nearly all otogenic brain abscesses are adjacent: that is to say the infection spreads upwards into the temporal lobe or less frequently backwards into the cerebellum, but from time to time cases are reported of abscesses in parts of the brain not directly in contact with the affected petrous bone. These distant abscesses have been found mainly in the frontal lobe and are the subject of a monograph by Nielsen and Courville who suggest that the infection spreads via the venous sinuses. Such abscesses may be difficult to localize as I found in the case of a woman with a history of a discharging left ear for many years who, following a chill, developed a severe headache and gradually became irritable and drowsy. Mastoidectomy revealed extensive disease with granulations on the lateral sinus. Exploration of the posterior fossa revealed no abscess either then or three days later, when in addition, the temporal lobe was also explored with negative results. Eventually, in the terminal stages, the opposite temporal lobe was explored and a large abscess found, but unfortunately too late to save the patient.

The neglected chronic suppurating ear is a much more frequent cause of brain abscess than the acute ear, and most authorities find that in 80% or more of abscesses there is a history of long-standing ear discharge. One reason for this lies in the fact that in the acute ear the prodromal symptoms caused by the infection eroding through the tegmen tympani are sufficient to demand exploration so that the process is arrested before a brain abscess can develop. On the other hand, in the chronic ear there may have been a very gradual erosion of the tegmen tympani, leaving the dura exposed to the disease with no symptoms other than a discharging ear and occasional bouts of headache. Such a state of affairs may be present for years, and may go unsuspected until an acute upper respiratory infection causes a flare up of the chronic process in the ear which spreads through the exposed dura to the brain.

I have noted erosion of the tegmen tympani by disease in nine cases of temporal lobe abscess secondary to chronic ear discharge, and in eight of these the increased thickness of the dura suggested that the erosion was of long standing; in all of them part at least of the exposed dura was sloughing. On opening the dura there was, in four cases, a subdural collection of pus with an underlying superficial ulceration of the brain tissue, but no deep abscess. In the remaining five, the abscess was separated from the surface by a narrow area of inflamed brain.

The invasion of the brain may be unsuspected because the otitis media is so fleeting, or again it may follow accidental damage during the course of a mastoid operation or as the result of exploring the brain for a supposed abscess.

Four methods of spread of infection from ear to brain may be recognized by the naked eye and are as follows:

- (1) Spread to an adjacent or distant lobe with no obvious change in the overlying dura.
- (2) Direct extension with obvious alterations in the appearance of the overlying dura.
- (3) Direct extension with dural changes, subdural abscess and superficial cortical ulceration.
- (4) Implantation abscess due to accidental or deliberate trauma.

In the first type it seems probable that the method of spread is by the perivascular spaces or by the blood-stream, either as an embolic or a thrombotic process, especially the latter, and in the distant abscesses it is more than likely that the extension has taken place via the venous sinuses. This is the most likely method of spread in the abscess that follows an acute ear infection, especially if the latter is fleeting.

The second type is, in my experience, the commonest and is most usually seen in abscesses secondary to chronic suppuration. In the cerebellar abscesses I have seen, the spread has been by this way, either via the bone between the sinus and the labyrinth (3 cases), via the labyrinth (1 case), or via the lateral sinus (1 case).

The third type, where the abscess is on the surface of the brain in association with a subdural collection of pus, is possibly more common than is generally realized and is not distinguishable clinically from the other types of abscess. It deserves attention because it responds to local drainage without exploration of the brain. In fact exploration of the brain is likely to do more harm than good and I content myself with free incision of the dura and nothing more.

The fourth type is probably the least frequent and requires no explanation except that it serves as a warning against the casual exploration of the brain through an infected area.

In otogenic cases the classical features of brain abscess, namely headache, vomiting, stupor, slow pulse and subnormal temperature, are often modified by the acute infective process in the ear and adjoining intracranial space, and the only general symptoms that

I have found to be constant are headache and slow cerebration. I have not yet met a case in which headache was not a prominent feature.

Nominal aphasia is constantly present in abscesses of the temporal lobe on the left side. It has been said that in left-handed persons it might be expected to be present if the abscess were on the right side. The only right-sided temporal lobe abscesses that I have seen have been in right-handed persons and they did not have nominal aphasia.

I have, however, seen a left temporal lobe abscess in a patient who was left-handed and he had nominal aphasia. The case in question was a real problem because both ears had been discharging for years and both were acutely inflamed and there was nothing to choose between either of them as the cause of the meningitis and brain abscess from which the patient appeared to be suffering. Although semi-delirious and drowsy, he was sufficiently co-operative to exhibit well-marked nominal aphasia and as he was left-handed it was decided to explore the right ear first of all. Mastoidectomy revealed extensive disease in the tympanum and mastoid antrum but no erosion of the tegmen tympani and a healthy dura. Nothing more was done on the right side and the left mastoid was opened, when an eroded tegmen tympani was found with a sloughing dura, and a subdural and superficial cortical abscess were drained. It has been suggested that this patient, a young man of 24, was really right-handed and that his apparent left-handedness was due to imitating his elder brother.

The management of brain abscesses secondary to ear disease presents many problems, and a study of the literature reveals a wide variety of opinions as to who should treat them, how and when and by what route they should be drained and in what manner the drainage should be maintained; but on one aspect of the treatment opinion seems to be unanimous, namely that the abscess should be drained or evacuated in such a way as to avoid spread of the infection to healthy tissues in the cranium.

With this object in view, it is urged by many authorities that an abscess should be allowed to localize and become encapsulated before it is drained. This is probably true of many brain abscesses, but in those secondary to chronic otitis media, especially when, as is so frequently the case, there is an acute flare up of a chronic ear, I think that unless the condition of the patient forbids it, the mastoid should be drained without delay. This enables the infecting focus to be eradicated, and prevents further spread of infection from the ear to the intracranial space. It also allows for the inspection of the dura adjacent to the petromastoid and the evacuation of any extra- or sub-dural collection of pus. In the case of the subdural abscess that may be accompanied by superficial cortical ulceration or at any rate some degree of encephalitis, this drainage of the infecting focus is usually sufficient to arrest the process. A policy of waiting in this type of case is a mistake. The only way of knowing that such a state of affairs is present is by exposing the dura at the site of infection.

When an abscess within the brain appears ready for evacuation, the question arises as to the route by which it should be drained. As the principal object is to drain an abscess without encouraging the spread of infection, advantage should be taken of any pre-existing track along which the abscess may be drained. Besides being shut off from the remainder of the intracranial space it has the additional advantage, in the case of adjacent abscesses, of being the shortest distance between the abscess and the surface.

When an abscess is thought to be adjacent to an infecting ear, the first step should be a thorough exposure of the dura adjoining the ear by means of a radical mastoid operation. An extradural or subdural abscess can be drained and if there is evidence of a track leading either to the middle or to the posterior fossa, the abscess can be drained along this track. If not, and the only way of being certain of this is to expose the dura via the mastoid, it may be wiser to explore the brain through a separate, surgically clean, approach.

The President said that the mastoid should always be opened and thoroughly explored and the dura uncovered. After that he would wait for the abscess to localize.

Had any member used the method of drainage through the track (Lemaître's method)—the introduction of a fine catheter with gradual dilatation along the track?

Was there such a thing as a non-suppurative encephalitis, or, alternatively, did every brain abscess start with a non-suppurative encephalitis?

Lastly, there was the importance of headache. No patient with an *uncomplicated* mastoid, however much pain he might have, should have a headache.

Mr. Cawthorne had asked about distant abscesses. He had seen one in the opposite temporal lobe and this he had discovered too late.

Mr. W. O. Lodge said that if they were to wait until their patients came before them with meningitis they were bound to have a very high mortality, and of the few patients who recovered a certain number would suffer later from epilepsy. Therefore a more aggressive attitude should be cultivated. The commonest cause of brain abscess was chronic otitis media. In 1941 2% of candidates for the Services were rejected on account of chronic otitis media, which should therefore be made a notifiable disease.

Mr. Thacker Neville was surprised at Mr. Northfield's insistence on excision of the abscess cavity. He would puncture the abscess with a needle and put in two minute drainage tubes and aspirate through them. Mr. Northfield had not converted him to excision by his figures.

Mr. R. G. Macbeth said that the best results were obtained in cases of brain abscess by a neuro-surgical team. It was important to differentiate between the chronic and the acute type of brain abscess. Though otologists had obtained their successes in the chronic type most of them had been disappointed with their results in the acute type. If neurosurgeons handled the acute cases, there was at least a hope that some of these would recover. Where there was acute middle-ear suppuration, this should be dealt with first.

Mr. Holt Diggle said that neuro-surgery had revolutionized the treatment of brain abscess in general. But he still thought that so far as their own specialty was concerned—and they were dealing more largely with otitic brain abscess than with abscesses of the frontal lobe—they would as a group prefer to continue the old teaching of Macewen to drain the brain abscess through its stalk of infection.

Mr. Cawthorne had said that in all cases of chronic infection a radical mastoid should be performed, but when one had to deal with an acute hæmorrhagic condition it was difficult to do a good radical mastoid and the patient might be in a moribund condition when seen. Of late it had been his practice to do a Schwartz operation in such cases and deal with the intracranial condition as revealed. Since then he had had better results. Subsequently he had dealt with the chronic condition by performing the radical operation after the acute symptoms had subsided.

Mr. I. G. Robin said, with reference to aphasia, he was reminded of a patient who went into an L.C.C. hospital two years ago in coma. She had had an otitis media for five years. She had hemiplegia and true aphasia, which cleared up, and he saw her one and a half years later with hemiplegia again and no aphasia. The patient was right-handed, and this was a left-sided abscess, 3 in. inwards and forwards with multiple loculi. It had a very dense capsule, with the track leading forward. Injection showed several cerebral loculi far forward. He could feel no abscess wall to these loculi, and he thought there must have been an acute spread within the last month.

The President, in closing the discussion, said that in the absence of a neuro-surgeon he was perfectly willing to do decompressions to any size and if need be drain the abscess, but if it came to a question of enucleating the abscess, that was entirely a neuro-surgeon's job, and fortunately even in wartime a neuro-surgeon was usually available.

Mr. D. W. C. Northfield (in reply) said that with regard to the treatment given to the ear, he had always been able to work with an ear surgeon. Some cases that had come to him from other hospitals had already had the ear disease attacked in one way or another, and there had been no necessity to do anything further. In cases in which abscess had arisen as the result of chronic ear disease, when he had successfully dealt with the abscess, he referred the case to an aural surgeon, or, if the ear condition had appeared to be rather more urgent, he dealt first with what appeared to be the predominant condition. It was wrong to operate on the ear in a patient who might be moribund not because of the ear disease, but because of the abscess. Then the treatment of the abscess should take precedence over anything else. After one or two aspirations, which was now almost his routine method, if the patient was improved, and if then a stage was reached in which the ear condition was predominant he would say: "Get on with the ear operation and do whatever is necessary." Each case must be taken on its own merits.

Left-handedness or right-handedness would appear to depend on which of the two hemispheres was the dominant one—usually it was the left. A left-handed person usually had a dominant right hemisphere. On the other hand, he might be fundamentally still dominant in his left hemisphere but have developed left-handedness. In such a case he would expect the left-handed person to have aphasia only from the left temporal region. Usually a left-handed person had aphasia only from the right-sided lesion, although he had seen such an unusual case as a right-handed person having aphasia from a right-sided lesion. Such a person was dominant in his right hemisphere, but he grew up using the right hand.

Headache was a conspicuous feature of brain abscess.

As to the necessity of enucleating an abscess he had had three cases in which aspiration only was undertaken and appeared successful, but he felt nervous lest these cases should flare up again; in one of these a ventriculogram was done and another was watched carefully, but no sign of returning abscess was discovered; but he always felt happier when the abscess was removed as if it were left there was always a risk that loculi would form.

As to aspiration and drainage by a fine tube, this might be all right in given cases. Each surgeon should proceed with those methods which in his hands had proved most successful. In his own hands drainage had not been successful.

Hammering in a case of brain abscess must accentuate the condition. It was a point which Mr. Hugh Cairns had raised at a meeting of the Section some years ago, and he quoted Macewen who said that he never used a hammer but always a curette or burr.

Sections of Otology and Laryngology

COMBINED MEETING HELD AT OXFORD

[July 17, 1942]

OTOLOGICAL SESSION

Chairman—F. W. WATKYN-THOMAS, F.R.C.S.

(President of the Section of Otology)

The Effect of Certain Cerebral Lesions Upon the Caloric Responses¹

By GERALD FITZGERALD and C. S. HALLPIKE

(From the Research Unit, National Hospital, Queen Square, London)

ACCORDING to Bauer and Leidler (1911) the removal of one cerebral hemisphere in the rabbit results in the exaggeration of the nystagmic response to a cessation of rotation towards the opposite side. For example, removal of the left hemisphere causes an exaggeration of the nystagmic response to a cessation of rotation to the right.

According to Ewald's law, the horizontal canals which are chiefly involved respond preponderantly to ampullo-petal flow of endolymph. Now cessation of rotation to the right causes an ampullo-petal flow of endolymph in the left horizontal canal, and Bauer and Leidler, accepting Ewald's law, argued that the enhancement of the nystagmic response to this stimulus was due to an increased sensitivity of the left labyrinth; and from this were led to the general conclusion that ablation of a cerebral hemisphere gives rise to an increase in sensitivity of the homolateral labyrinth.

These observations were repeated in 1923 by Dusser de Barenne and de Kleyn. Following removal of one hemisphere (e.g. the left) there was observed as before an exaggeration of the nystagmic response to cessation of rotation to the right, so confirming the finding of Bauer and Leidler. The effect was then tried of caloric stimulation. With hot caloric stimulation there was found an exaggeration of the response from the left labyrinth, a result which seemed to confirm Bauer and Leidler's notion of an increased sensitivity of the labyrinth on the side of the missing cerebral hemisphere. This was surprisingly contradicted, however, by the results obtained with cold water, which revealed an exaggeration in the response of the *right* labyrinth. These findings were explained in the following way: The three stimuli of which the effect was observed to be enhanced were

(1) Cessation of rotation to the right. (2) Hot caloric stimulation of the left labyrinth. (3) Cold caloric stimulation of the right labyrinth. All of these have it in common that they give rise to nystagmus to the left, and Dusser de Barenne and de Kleyn therefore concluded that the effect of removing one cerebral hemisphere, in this case the left, was a central facilitation of all and any labyrinthine impulses which would normally result in nystagmus to the same side. According to the terminology then adopted, the phenomenon was described as "nystagmusbereitschaft" to the side of the ablated cerebral hemisphere. These findings had an obvious application to the clinical problem of localization of cerebral lesions in man and were so applied by de Kleyn and Versteegh, who reported some results in 1927. Hot and cold caloric tests were carried out according to the threshold method of Kobrak (1918) in 36 subjects with cerebral lesions and a "nystagmusbereitschaft" was found in 11. No information was, however, given as to the nature or position of the lesions shown by clinical examination, operative or post-mortem findings except in one instance in which a large abscess of the frontal lobe was correlated with a "nystagmusbereitschaft" to the same side.

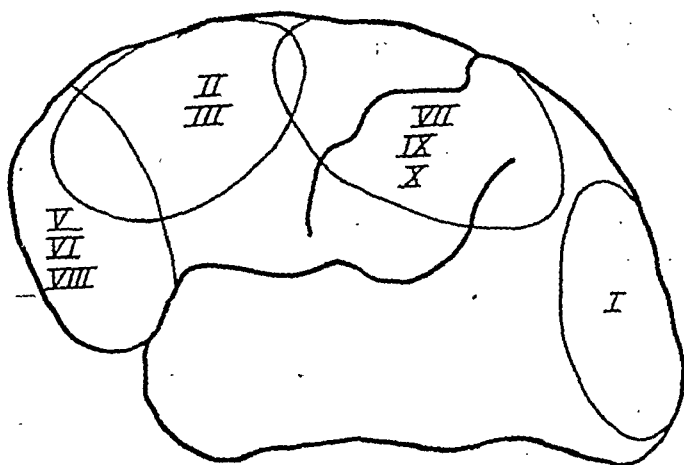
In the present work further evidence has been obtained of the occurrence in the human subject of "nystagmusbereitschaft" attributable to cerebral lesions, and it has further been possible to identify the area of the cortex, disease of which determines the appearance of this phenomenon. As an English translation of "nystagmusbereitschaft", the term directional preponderance of induced vestibular nystagmus has been devised and will be adhered to. It has the particular advantage of describing the essentially directional character of the disturbance.

MATERIAL

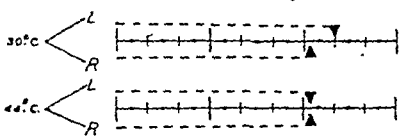
This was provided from the wards of the National Hospital and consisted in all of some 50 subjects in whom there was clinical and often other evidence of supratentorial

¹ Abridged version. For a full account of this work see *Brain*, 1942, 65, 115. Studies in Human Vestibular Function: I. Observations on the Directional Preponderance ("Nystagmusbereitschaft") of Caloric Nystagmus Resulting from Cerebral Lesions. The figures are reproduced by permission of the Editor of *Brain*.

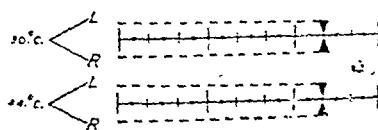
TABLE I.



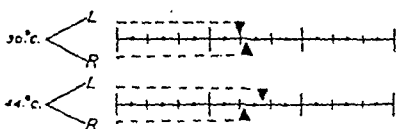
Temporal lobes not involved. Caloric reactions normal.



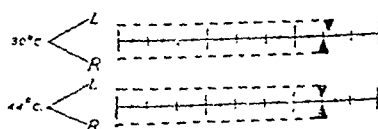
Case 1.—M. G.



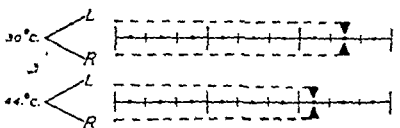
Case 6.—F. L.



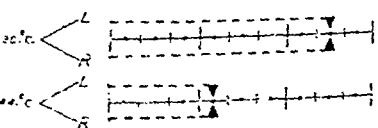
Case 2.—D. F.



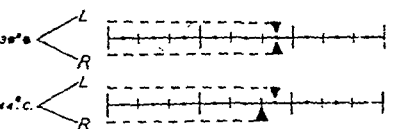
Case 7.—A. L.



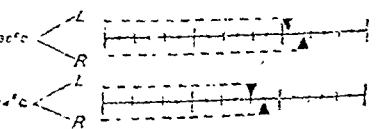
Case 3.—F. F.



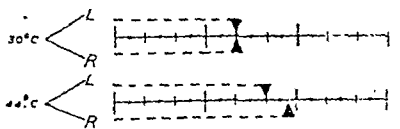
Case 8.—G. P.



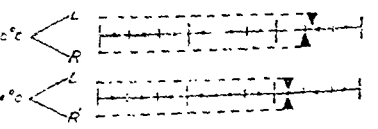
Case 4.—K. A.



Case 9.—A. S.



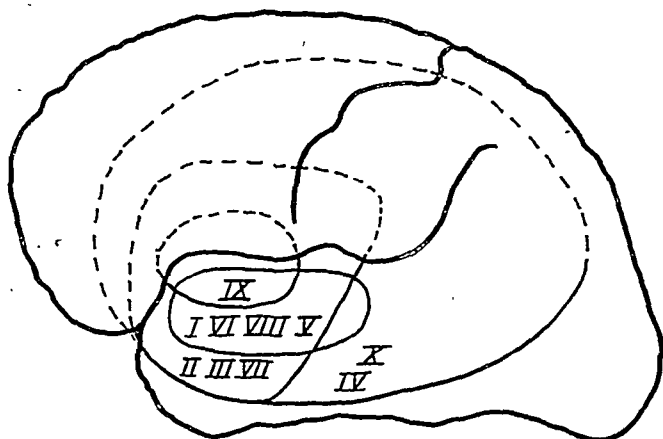
Case 5.—T. H.



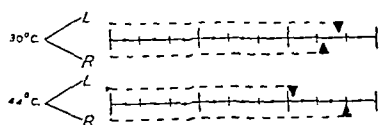
Case 10.—S. T.

lesions of the central nervous system. In none of these was there any evidence of aural disease or any spontaneous nystagmus. Hearing was normal in all. Hot and cold caloric tests were carried out with certain modifications of the usual procedure. In some cases the responses revealed directional preponderance, in others it was absent, and it soon became apparent that its occurrence was dependent upon the localization of the lesion. Final conclusions were based upon 20 subjects in whom the existence and localization of the lesions could be considered as securely established by the clinical, operative or post-mortem findings. For the most part the lesions were actively growing tumours, but some long-standing vascular lesions were also included.

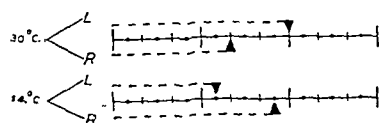
TABLE II.



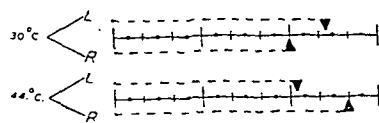
Cases 1-5.—Lesions involve right temporal lobe. Directional preponderance of caloric nystagmus to the right.



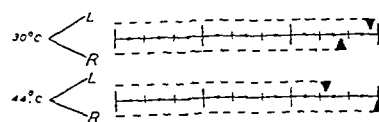
Case 1.—E. D.



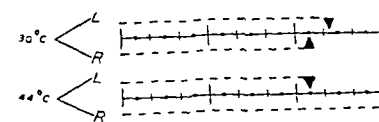
Case 2.—A. E.



Case 3.—L. G.

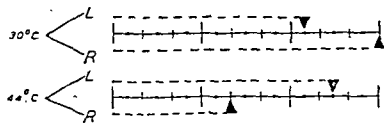


Case 4.—R. S.

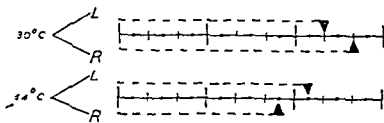


Case 5.—A. W.

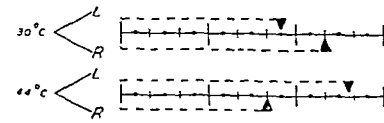
Cases 6-10.—Lesions involve left temporal lobe. Directional preponderance of caloric nystagmus to the left.



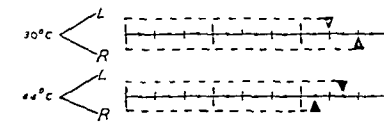
Case 6.—A. F.



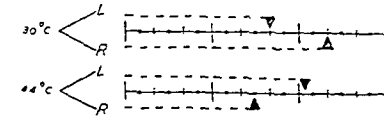
Case 7.—F. M.



Case 8.—T. M.



Case 9.—F. O'S.

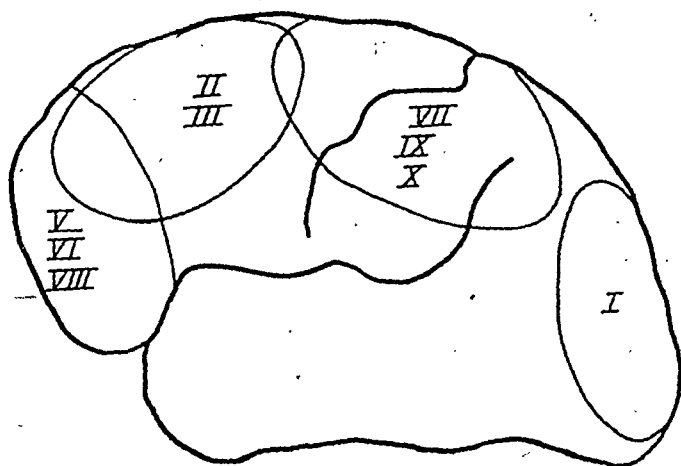


Case 10.—G. S.

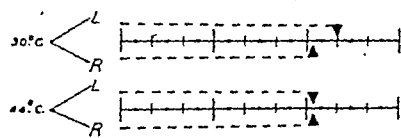
METHODS

To obtain maximum efficiency of the caloric tests certain technical modifications were found necessary. For the details and rationale of these modifications reference should be made to *Brain*, 1942, Part 2, p. 115, wherein are also discussed the limits of variation in the results obtained in normal subjects. The form adopted for expressing the results is shown in fig. 1, which gives the average result obtained in normal individuals. Each continuous line represents a 3-minute period, subdivided into intervals of minutes, 20 seconds and 10 seconds. L. and R. denote the reactions of the left and right labyrinths at 30° C.

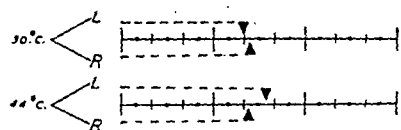
TABLE I.



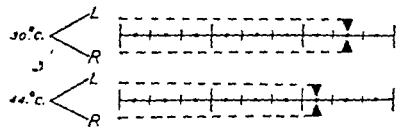
Temporal lobes not involved. Caloric reactions normal.



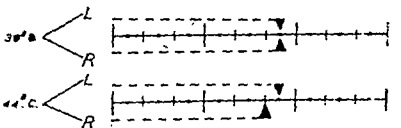
Case 1.—M. G.



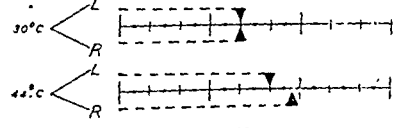
Case 2.—D. F.



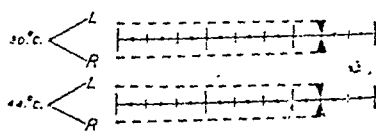
Case 3.—F. F.



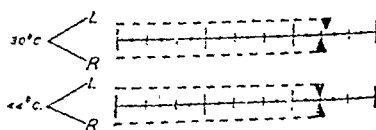
Case 4.—K. A.



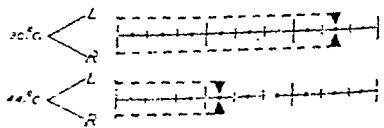
Case 5.—T. H.



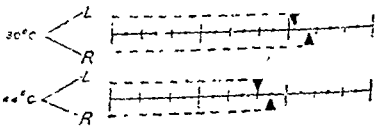
Case 6.—F. L.



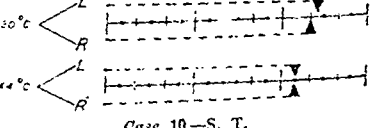
Case 7.—A. L.



Case 8.—G. P.



Case 9.—A. S.



Case 10.—S. T.

lesions of the central nervous system. In none of these was there any evidence of aural disease or any spontaneous nystagmus. Hearing was normal in all. Hot and cold caloric tests were carried out with certain modifications of the usual procedure. In some cases the responses revealed directional preponderance, in others it was absent, and it soon became apparent that its occurrence was dependent upon the localization of the lesion. Final conclusions were based upon 20 subjects in whom the existence and localization of the lesions could be considered as securely established by the clinical, operative or post-mortem findings. For the most part the lesions were actively growing tumours, but some long-standing vascular lesions were also included.

[July 17, 18, 1942]

LARYNGOLOGICAL SESSION

Chairman—E. D. D. DAVIS, F.R.C.S.

(President of the Section of Laryngology)

DISCUSSION ON INJURIES OF THE FRONTAL AND ETHMOIDAL SINUSES

Major C. A. Calvert, R.A.M.C.: The frontal and ethmoidal air sinuses insinuate themselves so intimately into the bony framework of the skull that, under the stress of injury and disease, they can be at times a very real anxiety to the neurologist and neuro-surgeon. The bony partition between the mucosal lining of the sinuses and the dural covering of the brain is very thin and brittle, particularly over the ethmoidal cells, and it is easily disrupted when severe grades of violence, such as those incidental to motor vehicle and aircraft accident, impinge on the frontal and fronto-temporal regions or, less frequently, on the face which juts downward prominently from the anterior part of the base of the skull.

The material on which my study of the problem of fractures of the frontal and ethmoidal sinuses is based has been obtained mostly from a military hospital for head injuries, all the cases treated there being included, and also from Brigadier Cairns' own series of pre-war cases.

The total number of cases in the series is 128. Of these, 103 were treated at the Military Hospital and 25 were pre-war cases of Brigadier Cairns. In trying to arrive at an idea of the incidence of frontal and ethmoidal sinus fractures in cases of head injury in general, I shall confine myself to the series in the Military Hospital. Out of a total of 1,731 cases of head injury admitted since the hospital opened, 655 or 37% had fractures of the vault or base of the skull. Out of these 655, 103 or 15% had fractures involving the sinuses now under discussion. This is a higher percentage than in a comparable General Hospital because more members of His Majesty's Forces travel rapidly on motor-cycles or in motor-cars or aeroplanes than do civilians. Reference to Table I, in which the nature of the accident is tabulated in percentages for the hospital series of 103 cases, shows that in 69% the patient had been travelling at speed at the time of his head injury, whilst in only 9% was the injury due to his being knocked down when walking along the road. This differs greatly from conditions in civilian life in which the latter cause is much more frequent.

TABLE I.—NATURE OF ACCIDENT.

| | | |
|--------------------------------------|----|-------|
| Motor-cycle crash | 40 | } 69% |
| Motor-car collision | 44 | |
| Aeroplane crash | 19 | |
| Bomb, shell, or gunshot wound | 6 | |
| Knocked down by motor-car | 8 | } |
| | 9 | |

In 70% the fractures were compound not only into the nose, but also through the skin of the forehead. Thus, an open wound demanding prompt excision frequently exists, and the important question arises how much should be done at operation in the acute stage for the fracture underneath. This will be referred to later.

Anosmia.—The state of the olfactory sense was known in nearly all of the cases. In 35% of these, the sense of smell was completely lost in one or both nostrils. Provided the nasal passages are clear, the presence of complete anosmia either unilaterally or bilaterally has a certain implication, for dural tearing was more than twice as common in such cases as in patients with only slight or no impairment of smell whatever.

Apart from gross brain damage, which is no more inimical in the frontal lobes than elsewhere in the brain, the chief hazard in cases of frontal or ethmoidal sinus fracture is laceration of the dural barrier and spread of infection intracranially from the nose. The question whether or not the dura is torn is therefore of the utmost concern. The knowledge that it is torn at once gives the case a more sombre complexion. The two unequivocal signs of dural rupture are leakage of cerebrospinal fluid from the nose and the presence of an intracranial aerocele. With either of these complications, the patient

and 44° C. The stimulus extends in all four over the first 40 seconds and the dotted lines indicate the duration of the responses. For reasons given elsewhere this is measured from the application of the stimulus.

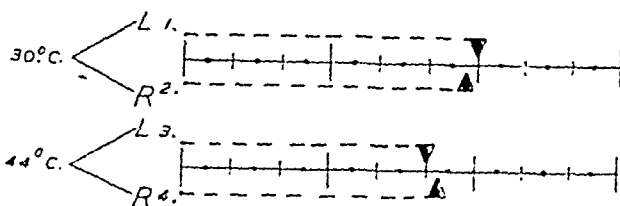


FIG. 1.—Normal.

Fig. 2 shows the response pattern characteristic of directional preponderance to the right (the effect obtained by Bauer and Leidler in the rabbit by removal of the right cerebral hemisphere). There is an exaggeration of the left cold and right hot responses, both of which consist of nystagmus to the right. The right cold and left hot responses, both of which consist of nystagmus to the left, are diminished.

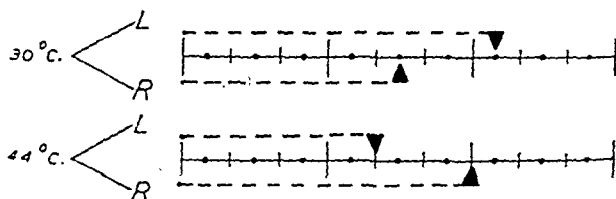


FIG. 2.—Directional preponderance to the right.

Fig. 3 shows the response pattern characteristic of directional preponderance to the left (the effect obtained by Bauer and Leidler in the rabbit by removal of the left cerebral hemisphere). There is an exaggeration of the right cold and left hot responses, both of which consist of nystagmus to the left. The left cold and right hot responses, both of which consist of nystagmus to the right, are diminished.

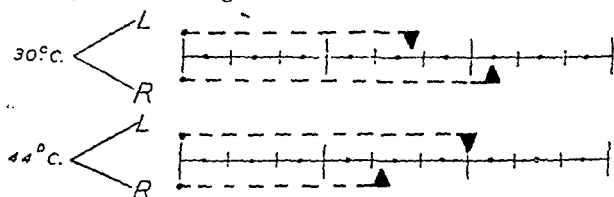


FIG. 3.—Directional preponderance to the left.

RESULTS

Table I shows the localization of the lesions and the caloric responses in 10 cases in which the responses were normal (the effect obtained by Bauer and Leidler in the rabbit by removal of the right cerebral hemisphere). In some of these the lesions involved the left hemisphere and in some the right. For the purpose of illustration they are all shown as being projected upon the lateral surface of the left hemisphere. The lesions are scattered throughout the hemisphere from the frontal to the occipital poles, with the exception of the temporal lobe. The caloric responses vary in their general magnitude but there is no alteration of their normal pattern.

Table II shows the localization of the lesions and the caloric responses in 10 cases in which the latter showed directional preponderance. In Cases 1—5 the lesions involved the right temporal lobe with directional preponderance of the caloric responses to the right. In Cases 6—10 the lesions involved the left temporal lobe with directional preponderance of the caloric responses to the left.

CONCLUSIONS

(1) The findings of Dusser de Barenne and de Kleyn are confirmed that directional preponderance of induced vestibular nystagmus (caloric) does occur in association with some lesions of the human cerebrum.

(2) The matter has been carried a stage further by the identification of the temporal lobe as the site of the nervous mechanism the involvement of which determines the occurrence of this phenomenon.

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 DUSSER DE BARENNE, J. G., and DE KLEYN, A. (1923), *Arch. f. Ophthalm.*, 111, 374.
 KOBRAK, F. (1918), *Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres*, 10, 214.

In order to estimate the incidence of spread of infection intracranially and the death-rate following fractures of the skull involving the frontal and ethmoidal sinuses, I shall have to limit myself to the hospital series. Out of the total of 103 cases, there were 5 deaths from meningitis or brain abscess. Two further cases developed a brain abscess secondary to the sinus fracture, but recovered with excision, and one case of meningitis survived after intensive M & B therapy and was then successfully grafted. This represents a morbidity rate of 8% and a death-rate of 5% from spread of infection intracranially in the frontal and ethmoidal sinus fractures in our series. For several reasons these figures probably do not give a true representation of the outlook in cases of fracture involving the paranasal sinuses. First, a number of our cases with dural laceration, and certainly most of the severe ones, were protected by a fascial graft. Secondly, as the hospital has been in operation for only about two and a half years, the follow-up period is very short, and it would be too optimistic to expect that all the cases with positive evidence of dural tearing, untreated by fascial grafting, would escape a fatal meningitis in the future. Thirdly, it is probable that some of the cases with severe dural tears actually develop meningitis and succumb without having recovered sufficiently from their cerebral damage to be fit for transfer to a head injury centre. In many cases of frontal sinus fracture, for example those in which it can be shown radiologically that the fracture is limited to the anterior wall of the frontal sinus, the question of dural repair does not arise, and a number of cases fall into this group.

In the cases of cerebrospinal rhinorrhœa and aerocele (operated and unoperated cases inclusive) the morbidity rate in respect of intracranial infection was 23%, and the death-rate 12%. In the cases of cerebrospinal rhinorrhœa and aerocele treated conservatively, the morbidity rate due to intracranial infection was 50%, and the death-rate 25%. Judging from these figures it seems that, unless there are very definite contra-indications on general grounds, all cases with a history of cerebrospinal rhinorrhœa or an intracranial aerocele should be operated upon and have their dural injury repaired.

Some surgeons, however, consider that dural repair is unnecessary in those cases of cerebrospinal rhinorrhœa in which the leakage has been slight and has ceased within a few days of the accident. Our figures show that this course is accompanied by some risk, and we have records of patients who have developed brain abscess or meningitis several months after the subsidence of a slight and evanescent cerebrospinal rhinorrhœa. It is generally granted that the presence of an intracranial aerocele indicates the necessity for dural repair.

Including Brigadier Cairns' cases, intracranial infection occurred in 6 patients in the series in which no history of rhinorrhœa was forthcoming, and in which the presence of an aerocele had been excluded by X-ray. In other words, intracranial infection occurred in 6 cases in which there was no clinical evidence of dural laceration. Two of these patients developed brain abscess; one died, and the other recovered after excision of the abscess. Of the remaining 4 who developed meningitis 3 died. The fourth patient recovered with intensive sulphanilamide therapy. He subsequently had a fascial graft operation carried out successfully for repair of a hole in the dura over the posterior ethmoidal cells.

The question therefore arises whether there are any other indications, apart from rhinorrhœa and aerocele, which point to the likelihood of laceration of the dura in relationship to the fractured cranial wall of the frontal and ethmoidal sinuses and thus indicate the advisability of operative intervention. The most important investigation in this respect is the radiological one, and I would like to acknowledge my indebtedness to Major Davies for his help in the difficult radiological aspect of the problem. The views that have been found most useful, apart from the lateral and stereoscopic P.A. films, are an occipito-mental projection and oblique orbital views taken from either side to show the optic foramina, orbital roofs, and the region of the crista galli and ethmoidal cells.

A wide gap in the posterior wall of a frontal sinus or any considerable displacement of bone in that situation, a fracture running down in the posterior wall of the frontal sinus to the anterior ethmoidal cells and widening rapidly as it descends, a fracture passing from one anterior cranial fossa across the cribriform plate to the opposite side of the base of the skull, a fracture associated with the projection intracranially of a sharp fragment from the back of the frontal sinuses or the medial part of the orbital roof, which would be likely to penetrate the dura, would all have to be regarded as likely to be complicated by dural laceration and therefore probably best treated by operation. The same applies to the inverted U-shaped fracture resulting from a blow on the side of the head in the region of the base of the external angular process of the frontal bone. The posterior limb of this fracture passes down into the anterior temporal region, whilst the anterior

must be considered to be in grave jeopardy. An analysis of the cases in the present series will sufficiently demonstrate this point.

Cerebrospinal rhinorrhœa.—Of the total of 128 cases, there were 21 with cerebrospinal rhinorrhœa, of whom 11 were treated conservatively and 10 were submitted to operation. In most of the conservatively treated cases, operation was advised but rejected by the patient. In a few, operation was for one or another reason not undertaken, either because of the patient's advanced age or other disadvantageous circumstance, or because the cerebrospinal fluid leak, of very short duration and never profuse, had long ago ceased by the time the patient presented for treatment, and, judging by the X-ray appearance of the fracture, it was likely that the meningeal tear had been firmly sealed off. The results in this series are summarized in Table II.

TABLE II.—CASES OF CEREBROSPINAL RHINORRHŒA.

| | |
|---------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 11 cases treated conservatively | 7 well to date—no intracranial infection. |
| | 4 { 3 developed meningitis, of whom 2 died. 1 developed brain abscess—excision—recovery. |
| 10 cases treated by dural graft | 0 recovered without incident. 1 died of meningitis—G.S.W. with penetration of dura and both walls of frontal sinus. F.B. removed. Hole in dura repaired with small muscle graft. Rhinorrhœa recurred—treated conservatively—meningitis—death. |

Intracranial aerocele.—This condition was present in 9 of our cases. Six of them were treated conservatively and 3 by repair of the dura. All of the 3 cases operated upon recovered. Of the 6 patients treated conservatively 4 recovered and are well up to date. The remaining two developed meningitis and one of them died (see Table III).

TABLE III.—CASES OF INTRACRANIAL AEROCELE.

| | |
|--------------------------|-----------------------------------------------------------------------|
| 6 treated conservatively | 4 recovered without incident. 2 developed meningitis, of whom 1 died. |
| 3 treated by dural graft | All recovered without incident. |

In a further 13 cases in which there was neither a history of rhinorrhœa nor radiological evidence of aerocele, the presence of a dural laceration was either demonstrated at operation or proved to have been present in certain cases that succumbed as a result of meningitis or brain abscess.

Cases with positive evidence of dural tear.—Adding the later 13 cases to the previous groups gives a total of 43 cases with positive evidence of a dural tear complicating frontal and ethmoidal sinus fractures. A study of the combined group is instructive.

Twenty of these cases were treated conservatively, and of these, 9 recovered without incident and 11 developed either meningitis or brain abscess, of whom 6 died.

Twenty-three had a dural repair operation, and of these 20 recovered without incident and 3 died. The 3 cases terminating fatally were all operated on during the acute stage.

One patient was subjected to excision of the scalp wound over the fractured frontal sinus within twelve hours of the accident, the fracture itself being left untouched. He had a stiff neck and a positive Kernig sign after operation, and two days later he developed a temperature of 103° F. The onset of meningitis was suspected and therefore the anterior and posterior walls of both frontal sinuses were removed and a large dural laceration at the back of the frontal sinuses repaired by a fascial graft. Unfortunately the patient developed a severe bronchopneumonia which terminated fatally. Post mortem the dural repair was entirely satisfactory. There was a thin layer of subdural clot and evidence of cerebral fat embolism, but none of meningitis. The reaction produced by clot and cerebral fat embolism had been mistaken for incipient meningitis, and had an extensive operation for dural repair not been undertaken then, but rather deferred until the patient's condition was less critical, it is possible that he might have survived.

The second death in the operated series was that of a patient who had received a gunshot wound in the centre of the forehead eight hours before admission to hospital. There was no amnesia. He was quite conscious and rational. The situation of the foreign body was localized carefully by X-rays and was found to be almost exactly in the mid-line about the position of the genu of the corpus callosum. One hour after admission to hospital, the entrance wound in the centre of the forehead was excised, exposing small, punched-out defects in the anterior and posterior walls of the frontal sinus. The macerated brain tissue presenting through the rent in the dura at the opening in the posterior wall of the frontal sinus was removed by gentle suction, and the metallic foreign body extracted with a pair of sinus forceps without difficulty. The operation was completed by the application of a small muscle graft over the dural opening, and the wound was closed. The patient did well afterwards, apart from a slight leakage of cerebrospinal fluid from his nose. This was treated conservatively, and then, rather suddenly, about five weeks after the accident, he developed a pneumococcal meningitis which ended fatally twelve days later. The conclusion in this case must be that the operative treatment was inadequate, and that the situation might still have been saved had the leakage of cerebrospinal fluid led to prompt intervention and effective repair of the hole in the dura.

The third patient was admitted to hospital eight hours after the accident with a small wound in the forehead, above the supra-orbital margin, through which pulped brain matter was escaping. The forehead wound was excised, and a good deal of comminution of the underlying bone was found. A small right frontal osteoplastic flap was turned, and revealing two small rents in the dura behind the fractured frontal sinus. The dura was opened, exposing an area of lacerated frontal pole, 3.5 cm. in diameter and 3 cm. deep, which was excised. The dura was then closed watertight and the back of the frontal sinus explored extradurally, down towards the orbital roof. The dura here was intact, but a fracture running in the roof of the orbit was seen. As there was no apparent indication for further interference, the operation was terminated by leaving a small gutta-percha drain down to the orbital plate for twenty-four hours. The early post-operative recovery was satisfactory, but eighteen days after the accident the patient developed a pneumococcal meningitis which proved fatal. At post mortem it was found that the entrance of the infection had been through a second dural tear over the ethmoidal cells, posterior to the area of operative exposure.

acute stage, whether there was leakage of cerebrospinal fluid or not. He made a small incision at the upper medial angle of the orbit and removed that part of the orbital roof forming the floor of the frontal sinus. Blood-clot was then sucked out, but, unless rhinorrhœa existed or cerebrospinal fluid was found in the sinus, nothing further was done except to leave a drain of rubber tubing, the inner end of which was made to lie accurately over the upper end of the fronto-nasal duct, so that air forced up through the latter from the nose would have a ready means of escape to the exterior. This drain was left in place for a week. When there was a complicating rhinorrhœa, the anterior wall of the frontal sinus was sacrificed to an extent sufficient to give good exposure of its posterior wall, which was in turn ablated to permit of repair of the underlying dural tear. There are certain objections to Teachenor's method. In the first place, operation on the injured sinus is unnecessary in the majority of uncomplicated frontal sinus fractures, for the blood-clot within is rarely the starting point of a sinusitis, especially if sulphanilamide therapy is employed, injunctions against blowing the nose are followed, and interference with the nostrils in order to get rid of blood-clot is avoided. In the second place, the exposure obtained by the bone removal he recommends is inadequate, should the dural tear lie well back in the cribriform plate region, which is not infrequently the case. Lastly, drainage of the sinus to the exterior sometimes results in a troublesome fistula.

Unless there is gross comminution and depression of both walls of the frontal sinuses, however, in which case Cone's operation may be employed, the most satisfactory method of approach when operation appears indicated, even during the acute stage, is by reflection of a small frontal osteoplastic flap just above the level of the frontal sinuses, as is usually employed in cases of chronic cranio-sinus fistula.

A history of preceding chronic frontal or ethmoidal sinusitis in a patient with traumatic cerebrospinal rhinorrhœa should be regarded as an indication for immediate repair of the dural defect.

The usual course, when operation is carried out during the chronic stage, is to turn a small frontal osteoplastic flap, which should be made to extend across the middle line when the fracture involves the posterior wall of both frontal sinuses or spreads back in the roof of the orbit on either side. The site of the dural tear may then be approached extradurally or intradurally. The method which Brigadier Cairns has used (*J. Laryng. & Otol.*, 1937, 52, 589) and which has been employed at our military hospital is the extradural approach. The dura is first separated from the posterior wall of the frontal sinus and then backwards along the roof of the orbit until the tear is reached. A fringe of dura sufficiently wide to permit of water-tight suture or the secure application of a fascial graft, must be exposed all around the tear in the membrane.

The intradural approach recommended by Julian Taylor for the treatment of a chronic fistula and reported by Major Eden in the *British Journal of Surgery* (1942, 29, 299) may also be employed. It is claimed that thereby (1) a more ready localization of the dural defect and an easier repair of it with fascia is possible than by the extradural method; (2) that, if the necessity should arise, closure of a rent on the other side of the middle line can be readily effected; and (3) that direct contact with the fracture line leading into the sinus, which may possibly be infected, is avoided.

The final complication of these sinus fractures to which I will refer, and that only very briefly, is sinusitis. In the hospital series there have been only 3 proven cases of frontal sinusitis, and intracranial infection did not occur in any of them. In all, the infection was very mild: one case responded within a few days to sulphanilamide therapy and inhalations; another to an intranasal operation; and a third to drainage of a small abscess which appeared at the upper inner angle of the orbit.

These fractures of the air sinuses are of more serious moment and more often productive of grave complications than the literature on the subject would lead one to expect.

Brigadier H. Cairns: At the outbreak of war our view was that practically all fractures into the frontal and ethmoidal sinuses should be operated on. We believed that there was usually a dural tear and, consequently, a considerable risk in every case that meningitis would occur as an immediate or remote complication. When the dura is broken there is usually considerable separation of the edges and these unite by a layer of fibrous tissue which is thinner than the dura itself: this scar has proved in a number of cases inadequate to prevent the spread of infection from the accessory sinuses to the meninges. In a series of cases reported to this Section five years ago (Cairns, 1937) there were some in which fatal meningitis ensued two or more years—in one case six years—after the original injury. Our belief was—and so far I know of no evidence against it—that thorough repair of the dura by suture or by fascia lata provides an adequate barrier against infection.

limb descends across the forehead to enter the frontal sinus on the same or opposite side, ending in the roof of the orbit. This is a dangerous type of fracture, especially when associated with much displacement at the fronto-zygomatic synostosis or with tilting of the crista galli over to the side. Either of these displacements suggests the likelihood of distortion in the neighbourhood of the ethmoidal cells with fracture of their thin brittle roof. Any one of these fractures, especially if accompanied by gross impairment of smell, would be regarded as a strong indication for operation, apart altogether from the presence of rhinorrhœa or intracranial aerocele.

Operation.—The majority of cases have wounds in the scalp leading down to the line of fracture. Excision of this wound, as in cases of compound fracture elsewhere, is therefore essential as soon as shock has been controlled. If there is cerebrospinal fluid coming from the nose, or if there is cerebrospinal fluid or brain tissue escaping from the forehead wound or the fracture is such that it is likely that there is an underlying dural tear, it has to be decided whether or not a long operation is to be undertaken to repair the dura during the acute stage. If the patient's general condition is good, the wound relatively clean, and little or no evidence of brain damage, and if adequate X-ray studies can be made to show the ramifications of the fracture without detriment to the patient's general condition, and all the requisite neuro-surgical facilities are available, the best course may well be to proceed at once, turn an osteoplastic flap if necessary, and repair the dural defect. In many cases some or perhaps all the attendant circumstances are disadvantageous: the wound is grossly contaminated, the general condition is such that to turn the patient about in an endeavour to obtain satisfactory X-ray films would prejudice his chances of survival, or he is perhaps so rebellious and unco-operative that nothing short of general anesthesia will suffice to control him during operation. In the majority of these, I believe the best course in the first instance is merely to excise the scalp wound thoroughly, having first obtained portable X-ray views so as to have some idea of the condition of the skull underneath. Even this limited interference should be deferred until the patient has recovered from shock, the scalp in the meantime having been shaved, the skin cleansed up to the edge of the wound, and a sterile pad sprinkled with sulphanilamide applied. The operation should when possible be carried out under local anesthesia. The skin edges having been excised, all foreign bodies, blood-clot, fragments of devitalized tissue, and any small loose fragments of bone are removed, and the operation should be terminated by dusting the wound with sulphanilamide and suturing it carefully in two layers with interrupted sutures of waxed silk, and without drainage. It is wrong to prise up and pull out in haphazard fashion large depressed portions of bone through an inadequate incision and then to retire, leaving unrepaired a dural tear now in more easy communication with the nose or the air sinuses than it was prior to the bone removal. Subsequently, the patient is given adequate doses of one of the sulphonamide drugs. I think chemotherapy is of value and should be employed in every case with blood-clot in the sinuses. No attempt should be made to wash clots out of the nose. If the patient is unconscious, he is kept well over on his side to facilitate the escape of blood and secretions from the air passages. As soon as he is sufficiently recovered to co-operate, he is warned of the risk of blowing his nose or sneezing. Then, when the immediate dangers incidental to his brain injury are past, a thorough X-ray study of the fracture should be undertaken. Now I believe is the time when, with all the data relating to the patient well worked out and with the probable situation of his dural lesion in mind, operation for the repair of the defect should be undertaken, the approach most likely to give an adequate exposure of the hole in the membrane being selected. In all cases with cerebrospinal rhinorrhœa, it is important that the patient should be nursed in a separate room, that his attendants should wear a mask, and that no one suffering from a head cold should be allowed to visit him.

In cases with gross comminution and depression of both walls of the frontal sinuses and almost certain dural laceration directly underneath the fracture a complete removal of the anterior wall, the posterior wall, and the mucosal lining of both frontal sinuses may be necessary during the acute stage. Then the dura is repaired and the wound is closed with a drain passing through the fronto-nasal duct into the nose, as recommended by Cone. The skin of the forehead subsequently becomes adherent to the underlying dura and the exenterated sinuses are thus permanently obliterated. The supra-orbital margins should be spared as far as possible during bone removal, but, notwithstanding this, the operation is a very disfiguring one; and though the cosmetic appearance can be improved by bone-grafting later, the deformity may have some psychological effect on the patient.

Teachenor recommended operation in every case of frontal sinus fracture during the

A New Forehead Flap for Nasal Reconstruction

By JOHN MARQUIS CONVERSE, M.D.

Plastic Surgeon to the American Hospital in Britain.

This paper was illustrated by a film, demonstrating the operative steps and showing results obtained in a number of patients.

FOLLOWING a correct diagnosis of the nasal deformity and an accurate estimation of the tissue loss some mode of repair must be considered. In some cases this is possible by the means of a local tissue shift to remedy a small defect. In many cases it is possible to replace a superficial defect by means of a free skin graft. However, when tissue loss is considerable, necessitating the reconstruction of the tip or the alæ of the nose, and in many cases of superficial defects unsuited for free grafting it is necessary to use a skin flap.

In a few favourable cases a flap from the surrounding tissue, of the nose itself or of the cheek may be used; in most cases, however, it is necessary to call upon a skin flap from a distance. The transfer of flaps from the chest, the arm, or the abdomen, means multiple stages and often an uncomfortable position for the patient. Furthermore the skin of these regions usually does not have the texture that permits the proper shaping of the nose. Their greatest disadvantage is the disparity in colour, which exists from the start and is often accentuated in the course of time.

A favourite method of repair in defects of the nose is by means of a forehead flap. This flap used in the ancient Hindu Rhinoplasty is one of the oldest of surgical procedures. Numerous modifications of this flap were made during the nineteenth century. In general an oblique forehead flap, giving added length was employed. Further lengthening of the flap was obtained with the introduction of Gillies' "up-and-down" modification.

The advantages of the forehead flap can be enumerated as follows: (1) Its contiguous availability. (2) Its good match in colour and texture. (3) Its excellent blood supply. These advantages follow the general principle that the reconstruction of a part can best be performed by using the part itself or the tissues in its immediate vicinity. It is particularly important in the reconstruction of a partial loss of the nose that the newly constructed part be blended with the remainder of the nose into a homogeneous feature.

The disadvantages of the forehead flap are twofold: (1) The secondary deformity produced on the forehead. (2) The fact that most forehead flaps are open flaps presenting a raw surface open to infection and fibrosis. With the idea of diminishing these disadvantages I have devised a forehead flap, the steps of which are illustrated. The advantages of this flap are the following: (1) Scarring on the forehead is reduced to that portion of the flap which will serve in the reconstruction. The incisions made for the "carrying" portion of the flap are placed entirely behind the hairline. (2) The flap may be closed by folding it upon itself, thus avoiding the danger of infection. Fibrosis is reduced to a minimum. The flap can be unfurled easily and replaced into its original site without difficulty. (3) An abundant blood supply is provided by the wide base of the flap.

The disadvantage of this flap is the large raw area created after its elevation. It is minimized, however, by the use of a temporary Thiersch graft skin dressing which is removed before the return of the flap.

Operative procedure.—The portion of the flap which is destined to reconstruct the nasal loss is outlined on the forehead as far laterally as possible. It is then carefully dissected off of the frontalis muscle without injury to the latter (fig. 1). In this manner the normal motility and wrinkling of the forehead are preserved. A coronal incision is then made as far as, but respecting, the anterior branches of the temporal vessels on the opposite side.

The frontalis muscle is split along the medial edge of the permanent defect (fig. 2). The forehead flap is raised with the galea aponeurotica and the remainder of the frontalis muscle. In this way the chances of injuring the essential afferent and efferent vessels are minimized. The nasal portion of the flap is sutured into position. The forehead flap is then folded upon itself (fig. 3) so as to be completely closed (fig. 4). A full thickness graft removed from behind the rim of the auricle and as far back as the hairline will suffice to cover a moderate-sized defect. It gives the best type of repair. An intermediate thickness skin graft is applied to the retro-auricular region and gives a good secondary skin replacement. The remaining raw area on the head is covered by Thiersch graft skin dressings (figs. 5 and 6). A pressure dressing is applied taking care to avoid pressure over the folded flap (figs. 7 and 8).

Later the attachment of the forehead flap to the nose is divided, the skin dressings removed (fig. 9), and the closed flap is unfolded without difficulty (figs. 10 and 11) and returned to its original site (fig. 12) leaving a laterally situated defect covered by a good colour-matching full-thickness graft.

After the outbreak of war, with the establishment of the Military Hospital for Head Injuries, we soon found that fractures into the accessory sinuses were much more common than we had supposed. I do not think that this means that the frequency of these injuries has increased in war time, for as Major Calvert has shown nearly half of them are due to motor-cyclists' injuries, and the number of these is no greater now than it was before the war. It is merely that now, for the first time, we have the patients segregated, and also they are probably much more thoroughly examined by X-rays than they were before. It is on X-ray examination that the diagnosis depends. Our pre-war ideas about treatment had been based on the selected cases of rhinorrhœa and aerocele. Many of the military cases prove to be simple fissures of the anterior or posterior walls of the frontal sinus: there is no rhinorrhœa or aerocele, and it may be extremely difficult to infer from X-ray examination whether the dura is torn and separated, or whether it is intact. In one case where X-ray showed a fracture with some separation of its edges in the posterior wall of the frontal sinus I found at operation that the dura was caught in the fracture line, but was intact. Either it had never been torn or, if torn, its edges had not separated and had united firmly.

I think there is general agreement that the dura should be repaired in cases of rhinorrhœa and aerocele, and in any other case in which an attack of meningitis has occurred after sinus injury, with recovery. The main problem is to know what to do about the other cases in which X-ray shows injury to the sinus walls. What is the risk of meningitis, either immediate or remote, in such cases, treated conservatively? This question can only be answered by a systematic long-term follow-up of an unselected series of cases. As Major Calvert's studies show there is a risk of meningitis in such cases, but whether it is small or large we do not know. Meanwhile more information can be obtained by X-ray studies and by correlating the X-ray findings with the condition of the dura as seen at operation and necropsy. It may be that certain radiographic signs, such as tilting of the crista galli, will prove to be fairly constantly accompanied by tearing of the dura: and that, with certain radiographic appearances, the presence or absence of anosmia will have a certain significance in relation to the state of the dura.

I have been struck with the low incidence of sinusitis after fracture into the sinuses. In Major Calvert's series of about 100 cases there were only three in which there was clinical evidence of frontal sinusitis in the weeks following the injury. This differs from what happens in the mastoid process: in my experience, when a fracture involves the mastoid, mastoiditis often occurs.

The infrequency of overt sinusitis after injury suggests that intracranial infection can occur—indeed, usually does occur—through sinuses that are not inflamed. If this is true, then it follows that the local operation on the injured sinuses as suggested by Teachenor is based on a false premise.

In 1937 I reported before this Section the case of a man who had suffered from meningitis and left frontal aerocele three years after a motor-cycle accident (Cairns, H. (1937), *J. Laryng. & Otol.*, 52, 389). During the meningitis, which was not severe, this man's left frontal sinus was explored and its mucosal lining was removed. In the postero-lateral bony wall of the sinus there was a gap through which the soft tissues were seen to bulge. The argument advanced at the time was that, as the mucous membrane had been removed, the left frontal sinus would not be involved when the patient contracted an upper respiratory infection, and that therefore there was no further risk of meningitis.

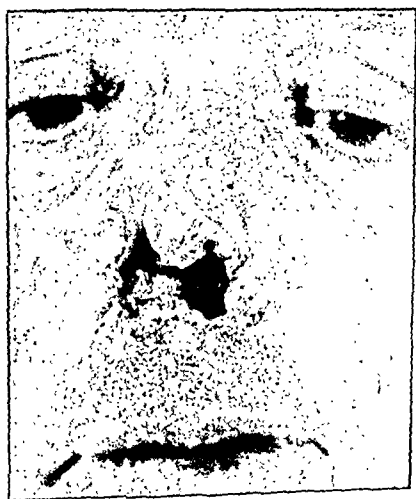
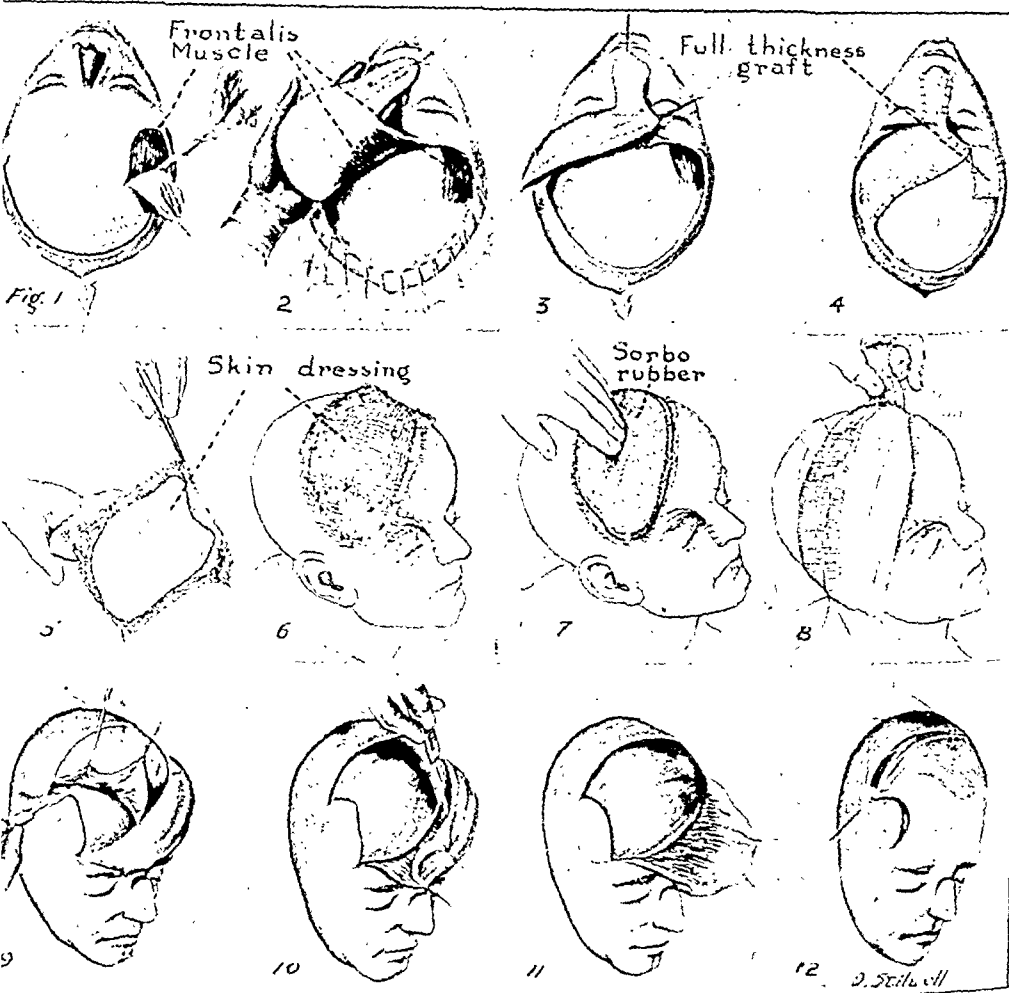
During the next eighteen months the patient had two heavy colds without any complication, but two and a quarter years after the operation (and five and a half years after the original head injury), he developed a pneumococcal meningitis and died. The meningitis supervened one month after a cold. At necropsy a small acute abscess was found in the left frontal lobe beneath the defect in the bony wall of the frontal sinus.

Local operation on the sinuses was ineffective in preventing meningitis in this case. Doubtless the mucous membrane of the sinuses regenerates quickly after removal.

Operation should provide for wide exposure of the dural surface of the accessory sinuses of both sides. I favour when possible the frontal flap and extradural approach, though the intradural approach of Taylor and Eden is doubtless equally effective in many cases. For most cases complete exenteration of the bony walls of the sinuses seems an unnecessarily mutilating procedure.

All these are operations of magnitude, and if performed in the first hours after the injury will usually need to be accompanied by blood transfusion. For this and other reasons I think it is better in most cases to defer repair of the dural defect until a week or two after injury, the patient meanwhile being kept on sulphonamides, with due care that he does not blow his nose or contract a cold. Wounds on the forehead or bridge of the nose should be closed in the first twenty-four hours after injury, and even when X-rays have shown that dural repair is necessary surgical intervention in the acute stage should usually be limited to débridement and closure of the scalp.

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Repair of nasal defect by forehead flap. Forehead defect is placed laterally and is thus less conspicuous.

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